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Date May 24, 2010

To: Gregg Townsend, NYSDEC, Region 7 (1 ltr; hc provided on October 16, 2009)
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Joseph J. Heath, Esq. (1 ltr; hc provided on October 16, 2009)

Re: Letter of Transmittal – Wastebed B/Harbor Brook Site Document Repository Addition
The below document has been approved by the New York State Department of Environmental Conservation (NYSDEC) and is enclosed for your document holdings:

- Wastebed B/Harbor Brook Human Health Risk Assessment Revised Report, dated October 2009.

Sincerely,



John P. McAuliffe, P.E.
Program Director, Syracuse

Enc.

cc: Tracy A. Smith – NYSDEC (ltr only)

New York State Department of Environmental Conservation

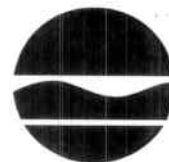
Division of Environmental Remediation

Remedial Bureau D – 12th Floor

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Alexander B. Grannis
Commissioner

May 19, 2010

Mr. John P. McAuliffe
Honeywell International, Inc.
Suite 700
5000 Brittonfield Parkway
East Syracuse, NY 13057

Re: Wastebed B/Harbor Brook HHRA

Dear Mr. McAuliffe:

The New York State Department of Environmental Conservation has reviewed the *Human Health Risk Assessment Report, Wastebed B/Harbor Brook Site* (HHRA) dated October 2009 and prepared by O'Brien & Gere on behalf of Honeywell. Based on our review, the HHRA is approved. However, based on the high vapor pressure of many of the compounds detected, a vapor intrusion evaluation will need to be conducted prior to the construction of occupied buildings at the site. Based on the vapor intrusion evaluation, preventive measures (e.g., use of a vapor barrier or installation of a venting system) may be included in the design and construction of buildings at the site to mitigate the risk of exposure to on-site soil gas. If you have any questions, please contact me at 518-402-9796.

Sincerely,

Tracy A. Smith
Project Manager

Honeywell
5000 Brittonfield Parkway
Suite 700
East Syracuse, NY 13057
315-431-4443
315-431-4777 Fax

October 16, 2009

Mr. Tracy A. Smith
Remedial Bureau D
New York State Department of Environmental Conservation
625 Broadway – 12th Floor
Albany, NY 12233-7016

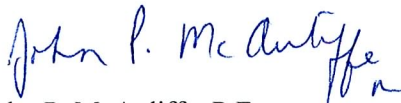
RE: Wastebed B/Harbor Brook Site, Geddes and Syracuse, Onondaga County, NY
Order on Consent: Index # D-7-0002-02-08
Revised HHRA Report and RAGS D Tables 1 through 10

Dear Mr. Smith:

Honeywell is pleased to submit the revised Human Health Risk Assessment (HHRA) report for the Wastebed B/Harbor Brook Site in Geddes and Syracuse, New York. Please note that the report included in this submittal reflects NYSDEC/USEPA comments dated May 1, 2009 to which Honeywell provided a response to comments on June 11, 2009 and comments provided during the subsequent conference call between Honeywell and the NYSDEC on August 18, 2009.

Please contact me at (315) 431-4443 or James Heckathorne of O'Brien & Gere at (315) 437-6100 with any questions.

Sincerely,



John P. McAuliffe, P.E.
Program Director, Syracuse

Attachments

cc:	Mr. Robert Nunes – USEPA (4)	Ms. Betsy Henry – Exponent (ltr only)
	Mr. Gregg Townsend – NYSDEC Reg 7 (copy, 1 CD)	Joseph J. Heath, Esq. (copy and CD)
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	Mr. Geoffrey Laccetti – NYSDOH (ltr only)	HETF/Onondaga Nation (CD)
	Margaret Sheen, Esq. – NYSDEC (ltr only)	Lindsay Speer – Onondaga Nat. (ec or ltr only)
	Argie Cirillo, Esq. – USEPA (ltr only)	Heidi Kuhl – Onondaga Nat. (ec or ltr only)
	Brian D. Israel, Esq. – Arnold & Porter (ec or CD)	Mr. Christopher Calkins – O'Brien & Gere
	Mr. Alfred J. Labuz – Honeywell (ltr only)	Mr. William A. Schew – O'Brien & Gere
	Mr. Michael Spera – AECOM	
	Mr. David Coburn – Onondaga County Dept. of Environment (1 copy, 1 CD)	

REVISED REPORT

**Human Health Risk Assessment
Wastebed B/Harbor Brook Site
Geddes and Syracuse, New York**

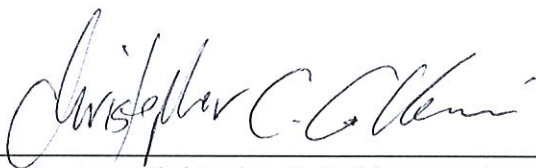
Honeywell

October 2009

REVISED REPORT

Human Health Risk Assessment
Wastebed B/Harbor Brook Site
Geddes and Syracuse, New York

Honeywell



Christopher C. Calkins
Vice President

October 2009



O'BRIEN & GERE

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- 2.25d Derivation of Total Xylene Concentrations – Dredge Spoil Area #1: Surface Soil

- 2.26a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – Dredge Spoil Area #1 (Current/Future): Subsurface Soil
- 2.26b Derivation of Toxic Equivalents for Dioxins – Dredge Spoil Area #1: Subsurface Soil
- 2.26c Derivation of PCB Equivalents for Chlorinated Chemicals – Dredge Spoil Area #1: Subsurface Soil
- 2.26d Derivation of Total Xylene Concentrations – Dredge Spoil Area #1: Subsurface Soil
- 2.27a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – Dredge Spoil Area #2 (Current/Future): Surface Soil
- 2.27b Derivation of Toxic Equivalents for Dioxins – Dredge Spoil Area #2: Surface Soil
- 2.27c Derivation of PCB Equivalents for Chlorinated Chemicals – Dredge Spoil Area #2: Surface Soil
- 2.27d Derivation of Total Chlordane Concentrations – Dredge Spoil Area #2: Surface Soil
- 2.27e Derivation of Total Xylene Concentrations – Dredge Spoil Area #2: Surface Soil
- 2.28a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – Dredge Spoil Area #2 (Current/Future): Subsurface Soil
- 2.28b Derivation of Toxic Equivalents for Dioxins – Dredge Spoil Area #2: Subsurface Soil
- 2.28c Derivation of PCB Equivalents for Chlorinated Chemicals – Dredge Spoil Area #2: Subsurface Soil
- 2.28d Derivation of Total Chlordane Concentrations – Dredge Spoil Area #2: Subsurface Soil
- 2.28e Derivation of Total Xylene Concentrations – Dredge Spoil Area #2: Subsurface Soil
- 2.29a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – Dredge Spoil Area #2 (Current/Future): Shallow Ground Water
- 2.29b Derivation of Total Xylene Concentrations – Dredge Spoil Area #2: Shallow Ground Water
- 2.30a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – Dredge Spoil Area #2 (Current/Future): Shallow Ground Water – Vapor Intrusion
- 2.30b Derivation of Total Xylene Concentrations – Dredge Spoil Area #2: Shallow Ground Water – Vapor Intrusion
- 2.31a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – AOS #1 (Current/Future): Surface Soil
- 2.31b Derivation of Toxic Equivalents for Dioxins – AOS #1: Surface Soil
- 2.31c Derivation of PCB Equivalents for Chlorinated Chemicals – AOS #1: Surface Soil
- 2.31d Derivation of Total Chlordane Concentrations – AOS #1: Surface Soil
- 2.31e Derivation of Total Xylene Concentrations – AOS #1: Surface Soil
- 2.32a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – Additional Area of Study #1 (Current/Future): Subsurface Soil
- 2.32b Derivation of Toxic Equivalents for Dioxins – Additional Area of Study #1: Subsurface Soil
- 2.32c Derivation of PCB Equivalents for Chlorinated Chemicals – Additional Area of Study #1: Subsurface Soil
- 2.32d Derivation of Total Chlordane Concentrations – AOS #1: Subsurface Soil
- 2.32e Derivation of Total Xylene Concentrations – AOS #1: Subsurface Soil
- 2.33a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – AOS #1 (Current/Future): Shallow Ground Water
- 2.33b Derivation of Total Xylene Concentrations – AOS #1: Shallow Ground Water
- 2.34a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – AOS#1 (Current/Future): Shallow Ground Water – Vapor Intrusion
- 2.34b Derivation of Total Xylene Concentrations – AOS #1: Shallow Ground Water – Vapor Intrusion
- 2.35 Occurrence, Distribution, and Selection of Chemicals of Potential Concern – AOS #2 (Current/Future): Surface Soil

- 2.36a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – AOS #2 (Current/Future): Surface Sediment
- 2.36b Derivation of Toxic Equivalents for Dioxins – AOS #2: Surface Sediment
- 2.37a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – SYW-12 (Current/Future): Surface Soil
- 2.37b Derivation of Toxic Equivalents for Dioxins – SYW-12 (Current/Future): Surface Soil
- 2.37c Derivation of PCB Equivalents for Chlorinated Chemicals – SYW-12 (Current/Future): Surface Soil
- 2.37d Derivation of Total Chlordane Concentrations – SYW-12: Surface Soil
- 2.37e Derivation of Total Xylene Concentrations – SYW-12: Surface Soil
- 2.38a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – SYW-12 (Current/Future): Subsurface Soil
- 2.38b Derivation of Toxic Equivalents for Dioxins – SYW-12 (Current/Future): Subsurface Soil
- 2.38c Derivation of PCB Equivalents for Chlorinated Chemicals – SYW-12 (Current/Future): Subsurface Soil
- 2.38d Derivation of Total Chlordane Concentrations – SYW-12: Subsurface Soil
- 2.38e Derivation of Total Xylene Concentrations – SYW-12: Subsurface Soil
- 2.39a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – SYW-12 (Current/Future): Shallow Ground Water
- 2.39b Derivation of Total Xylene Concentrations – SYW-12: Shallow Ground Water
- 2.40a Occurrence, Distribution, and Selection of Chemicals of Potential Concern – SYW-12 (Current/Future): Shallow Ground Water – Vapor Intrusion
- 2.40b Derivation of Total Xylene Concentrations – SYW-12: Shallow Ground Water – Vapor Intrusion
- 3.1a Exposure Point Concentration Summary – Exposure Unit 1 (Current/Future): Site-Wide Surface Soil Reasonable Maximum Exposure/Central Tendency
- 3.1b Exposure Point Concentration Summary – Exposure Unit 1 (Current/Future): Site-Wide Subsurface Soil Reasonable Maximum Exposure/Central Tendency
- 3.1c Exposure Point Concentration Summary – Exposure Unit 1 (Current/Future): Site-Wide Surface Sediments Reasonable Maximum Exposure/Central Tendency
- 3.1d Exposure Point Concentration Summary – Exposure Unit 1 (Current/Future): Site-Wide Upper Sediment Reasonable Maximum Exposure/Central Tendency
- 3.1e Exposure Point Concentration Summary – Exposure Unit 1 (Current/Future): Site-Wide Shallow Ground Water Reasonable Maximum Exposure/Central Tendency
- 3.1f Exposure Point Concentration Summary – Exposure Unit 1 (Current/Future): Site-Wide Surface Water Reasonable Maximum Exposure/Central Tendency
- 3.1g Exposure Point Concentration Summary – Exposure Unit 1 (Current/Future): Onondaga Lake Fish Tissue Reasonable Maximum Exposure/Central Tendency
- 3.2 Exposure Point Concentration Summary – Exposure Unit 2 (Current/Future): Surface Soil Reasonable Maximum Exposure/Central Tendency
- 3.3a Exposure Point Concentration Summary – Exposure Unit 3 (Current/Future): Surface Sediment Reasonable Maximum Exposure/Central Tendency
- 3.3b Exposure Point Concentration Summary – Exposure Unit 3 (Current/Future): Surface Water Reasonable Maximum Exposure/Central Tendency
- 3.4 Exposure Point Concentration Summary – Exposure Unit 4 (Current/Future): Surface Soil Reasonable Maximum Exposure/Central Tendency
- 3.5 Exposure Point Concentration Summary – Exposure Unit 5 (Current): Surface Soil Reasonable Maximum Exposure/Central Tendency

- 3.6a Exposure Point Concentration Summary – Exposure Unit 6 (Future): Surface Soil Reasonable Maximum Exposure/Central Tendency
- 3.6b Exposure Point Concentration Summary – Exposure Unit 6 (Future): Surface Sediment Reasonable Maximum Exposure/Central Tendency
- 3.6c Exposure Point Concentration Summary – Exposure Unit 6 (Future): Surface Water Reasonable Maximum Exposure/Central Tendency
- 3.6d Exposure Point Concentration Summary – Exposure Unit 6 (Future): Onondaga Lake Fish Tissue Reasonable Maximum Exposure/Central Tendency
- 3.7 Exposure Point Concentration Summary – Exposure Unit 7 (Future): Surface Soil Reasonable Maximum Exposure/Central Tendency
- 3.8 Exposure Point Concentration Summary – Exposure Unit 8 (Future): Site Wide All Ground Water Reasonable Maximum Exposure/Central Tendency
- 3.9a Exposure Point Concentration Summary – Exposure Unit 9 - SYW-12 (Current/Future): Surface Soil Reasonable Maximum Exposure/Central Tendency
- 3.9b Exposure Point Concentration Summary – Exposure Unit 9 - SYW-12 (Current/Future): Subsurface Soil Reasonable Maximum Exposure/Central Tendency
- 3.9c Exposure Point Concentration Summary – Exposure Unit 9 - SYW-12 (Current/Future): Shallow Ground Water Reasonable Maximum Exposure/Central Tendency
- 4.1a RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Soil
- 4.1a RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 1 (Current/Future): Surface Soil
- 4.1b RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Soil & Subsurface Soil
- 4.1c RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Future): Surface Soil & Subsurface Soil
- 4.1d RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Shallow Ground Water
- 4.1e RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Future): Shallow Ground Water
- 4.1f RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Sediment
- 4.1f RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 1 (Current/Future): Surface Sediment & Subsurface Sediment
- 4.1g RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Sediment
- 4.1h RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Future): Surface Sediment & Subsurface Sediment
- 4.1i RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Water
- 4.1i RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 1 (Current/Future): Surface Water
- 4.1j RME: Values Used for Daily Intake Calculations – Exposure Unit 1 (Future): Surface Water
- 4.1k RME: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Fish Tissue
- 4.2 RME: Values Used For Daily Intake Calculations – Exposure Unit 2 (Current/Future): Surface Soil
- 4.3a RME: Values Used For Daily Intake Calculations – Exposure Unit 3 (Current/Future): Surface Sediment

- 4.3b RME: Values Used For Daily Intake Calculations – Exposure Unit 3 (Current/Future): Surface Water
- 4.4 RME: Values Used For Daily Intake Calculations – Exposure Unit 4 (Current/Future): Surface Soil
- 4.5 RME: Values Used For Daily Intake Calculations – Exposure Unit 5 (Current): Surface Soil
- 4.6a RME: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Surface Soil
- 4.6a RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 6 (Future): Surface Soil
- 4.6b RME: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Surface Sediment
- 4.6b RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 6 (Future): Surface Sediment
- 4.6c RME: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Surface Water
- 4.6c RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 6 (Future): Surface Water
- 4.6d RME: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Fish Tissue
- 4.7 RME: Values Used For Daily Intake Calculations – Exposure Unit 7 (Future): Surface Soil
- 4.8 RME: Values Used For Daily Intake Calculations – Exposure Unit 8 (Future): Potable Ground Water
- 4.8 RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 8 (Future): Potable Ground Water
- 4.9a RME: Values Used For Daily Intake Calculations – Exposure Unit 9 (Current/Future): Surface Soil
- 4.9a RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 9 (Current/Future): Surface Soil
- 4.9b RME: Values Used For Daily Intake Calculations – Exposure Unit 9 (Future): Surface Soil
- 4.9b RME Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 9 (Future): Surface Soil
- 4.9c RME: Values Used For Daily Intake Calculations – Exposure Unit 9 (Current/Future): Surface Soil & Subsurface Soil
- 4.9d RME: Values Used For Daily Intake Calculations – Exposure Unit 9 (Future): Surface Soil & Subsurface Soil
- 4.9e RME: Values Used For Daily Intake Calculations – Exposure Unit 9 (Current/Future): Shallow Ground Water
- 4.9f RME: Values Used For Daily Intake Calculations – Exposure Unit 9 (Future): Shallow Ground Water
- 4.10 RME: Table 4 Supplement B – Age Dependent Adjustment Factor: Exposure Parameters
- 4.1a CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Soil
- 4.1a CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 1 (Current/Future): Surface Soil
- 4.1b CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Soil & Subsurface Soil
- 4.1c CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Future): Surface Soil & Subsurface Soil
- 4.1d CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Shallow Ground Water
- 4.1e CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Future): Shallow Ground Water

- 4.1f CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Sediment
- 4.1f CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 1 (Current/Future): Surface Sediment
- 4.1g CT: Values Used For Daily Intake Calculations– Exposure Unit 1 (Current/Future): Surface Sediment
- 4.1h CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Future): Surface & Subsurface Sediment
- 4.1i CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Surface Water
- 4.1i CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 1 (Current/Future): Surface Water
- 4.1j CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Future): Surface Water
- 4.1k CT: Values Used For Daily Intake Calculations – Exposure Unit 1 (Current/Future): Fish Tissue
- 4.2 CT: Values Used For Daily Intake Calculations – Exposure Unit 2 (Current/Future): Surface Soil
- 4.3a CT: Values Used For Daily Intake Calculations – Exposure Unit 3 (Current/Future): Surface Sediment
- 4.3b CT: Values Used For Daily Intake Calculations – Exposure Unit 3 (Current/Future): Surface Water
- 4.4 CT: Values Used For Daily Intake Calculations – Exposure Unit 4 (Current/Future): Surface Soil
- 4.5 CT: Values Used For Daily Intake Calculations – Exposure Unit 5 (Current): Surface Soil
- 4.6a CT: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Surface Soil
- 4.6a CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 6 (Future): Surface Soil
- 4.6b CT: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Surface Sediment
- 4.6b CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 6 (Future): Surface Sediment
- 4.6c CT: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Surface Water
- 4.6c CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 6 (Future): Surface Water
- 4.6d CT: Values Used For Daily Intake Calculations – Exposure Unit 6 (Future): Fish Tissue
- 4.6d CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 6 (Future): Fish Tissue
- 4.7 CT: Values Used For Daily Intake Calculations – Exposure Unit 7 (Future): Surface Soil
- 4.8 CT: Values Used For Daily Intake Calculations – Exposure Unit 8 (Future): Potable Ground Water
- 4.8 CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 8 (Future): Potable Ground Water
- 4.9a CT: Values Used For Daily Intake Calculations – Exposure Unit 9 (Current/Future): Surface Soil
- 4.9a CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 9 (Current/Future): Surface Soil
- 4.9b CT: Values Used For Daily Intake Calculations – Exposure Unit 9 (Future): Surface Soil
- 4.9b CT Supplement A: Values Used For Daily Intake Calculations (mutagenic mode of action) – Exposure Unit 9 (Future): Surface Soil

- 4.9c CT: Values Used For Daily Intake Calculations – Exposure Unit 9 (Current/Future): Surface Soil & Subsurface Soil
- 4.9d CT: Values Used For Daily Intake Calculations – Exposure Unit 9 (Future): Surface Soil and Subsurface Soil
- 4.9e CT: Values Used For Daily Intake Calculations – Exposure Unit 9 (Current/Future): Shallow Ground Water
- 4.9f CT: Values Used For Daily Intake Calculations – Exposure Unit 9 (Future): Shallow Ground Water
- 4.10 CT: Table 4 Supplement B – Age Dependent Adjustment Factor: Exposure Parameters
- 5.1 Non-Cancer Toxicity Data – Oral/Dermal
- 5.2 Non-Cancer Toxicity Data – Inhalation
- 6.1 Cancer Toxicity Data – Oral/Dermal
- 6.2 Cancer Toxicity Data – Inhalation
- 7.1 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Trespasser (Older Child) (Current/Future)
- 7.1 RME Supplement A: Calculation of Cancer Risks For COPC with Mutagenic Mode of Action – Trespasser (Older Child) (Current/Future)
- 7.2 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Trespasser (Adult) (Current/Future)
- 7.3 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Utility Worker (Current/Future)
- 7.3a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Utility Worker (Current/Future)
- 7.4 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Construction Worker (Future)
- 7.4a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Construction Worker (Future)
- 7.5 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Surveillance Worker (Current/Future)
- 7.6 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Ditch Worker (Current/Future)
- 7.7 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Railroad Worker (Current/Future)
- 7.7a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Railroad Worker (Current/Future)
- 7.8 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Commercial/Industrial Worker (Current/Future)
- 7.9 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Commercial/Industrial Worker (Future)
- 7.9a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Commercial/Industrial Worker (Future)
- 7.10 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Recreational Visitor (Child) (Future)
- 7.10 RME Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode Of Action – Recreational Visitor (Child) (Future)
- 7.10a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Recreational Visitor (Child) (Current/Future)
- 7.10a RME Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode of Action – SYW-12 – Recreational Visitor (Child) (Current/Future)

- 7.11 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Recreational Visitor (Adult) (Future)
- 7.11a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Recreational Visitor (Adult) (Current/Future)
- 7.12 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Resident (Child) (Future)
- 7.12 RME Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode Of Action – Resident (Child) (Current/Future)
- 7.12a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Resident (Child) (Future)
- 7.12a RME Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode of Action – SYW-12 – Resident (Child) (Current/Future)
- 7.13 RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Resident (Adult) (Future)
- 7.13a RME: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Resident (Adult) (Future)
- 7.1 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Trespasser (Older Child) (Current/Future)
- 7.1 CT Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode of Action – Trespasser (Older Child) (Current/Future)
- 7.2 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Trespasser (Adult) (Current/Future)
- 7.3 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Utility Worker (Current/Future)
- 7.3a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Utility Worker (Current/Future)
- 7.4 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Construction Worker (Future)
- 7.4a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Construction Worker (Future)
- 7.5 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Surveillance Worker (Current/Future)
- 7.6 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Ditch Worker (Current/Future)
- 7.7 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Railroad Worker (Current/Future)
- 7.7a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Railroad Worker (Current/Future)
- 7.8 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Commercial/Industrial Worker (Current/Future)
- 7.9 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Commercial/Industrial Worker (Future)
- 7.9a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Commercial/Industrial Worker (Future)
- 7.10 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Recreational Visitor (Child) (Future)
- 7.10 CT Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode of Action – Recreational Visitor (Child) (Current/Future)

- 7.10a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Recreational Visitor (Child) (Current/Future)
- 7.10a CT Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode Of Actions – SYW-12 – Recreational Visitor (Child) (Current/Future)
- 7.11 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Recreational Visitor (Adult) (Future)
- 7.11a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Recreational Visitor (Adult) (Current/Future)
- 7.12 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Resident (Child) (Future)
- 7.12 CT Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode Of Action – Resident (Child) (Current/Future)
- 7.12a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Resident (Child) (Future)
- 7.12a CT Supplement A: Calculation of Chemical Cancer Risks For COPC With Mutagenic Mode of Action – SYW-12 – Resident (Child) (Current/Future)
- 7.13 CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – Resident (Adult) (Future)
- 7.13a CT: Calculation of Chemical Cancer Risks and Non-Cancer Hazards – SYW-12 – Resident (Adult) (Future)
- 9.1 RME: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Older Child) (Current/Future)
- 9.2 RME: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Adult) (Current/Future)
- 9.3 RME: Summary of Receptor Risks and Hazards for COPCs – Utility Worker (Current/Future)
- 9.3a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Utility Worker (Current/Future)
- 9.4 RME: Summary of Receptor Risks and Hazards for COPCs – Construction Worker (Future)
- 9.4a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Construction Worker (Future)
- 9.5 RME: Summary of Receptor Risks and Hazards for COPCs – Surveillance Worker (Current/Future)
- 9.6 RME: Summary of Receptor Risks and Hazards for COPCs – Ditch Worker (Current/Future)
- 9.7 RME: Summary of Receptor Risks and Hazards for COPCs – Railroad Worker (Current/Future)
- 9.7a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Railroad Worker (Current/Future)
- 9.8 RME: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Current/Future)
- 9.9 RME: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Future)
- 9.9a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Commercial/Industrial Worker (Future)
- 9.10 RME: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Child) (Future)
- 9.10a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Child) (Current/Future)

- 9.11 RME: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Adult) (Future)
- 9.11a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Adult) (Current/Future)
- 9.12 RME: Summary of Receptor Risks and Hazards for COPCs – Resident (Child) (Future)
- 9.12a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Child) (Future)
- 9.13 RME: Summary of Receptor Risks and Hazards for COPCs – Resident (Adult) (Future)
- 9.13a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Adult) (Future)
- 9.1 CT: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Older Child) (Current/Future)
- 9.2 CT: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Adult) (Current/Future)
- 9.3 CT: Summary of Receptor Risks and Hazards for COPCs – Utility Worker (Current/Future)
- 9.3a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Utility Worker (Current/Future)
- 9.4 CT: Summary of Receptor Risks and Hazards for COPCs – Construction Worker (Future)
- 9.4a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Construction Worker (Future)
- 9.5 CT: Summary of Receptor Risks and Hazards for COPCs – Surveillance Worker (Current/Future)
- 9.6 CT: Summary of Receptor Risks and Hazards for COPCs – Ditch Worker (Current/Future)
- 9.7 CT: Summary of Receptor Risks and Hazards for COPCs – Railroad Worker (Current/Future)
- 9.7a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Railroad Worker (Current/Future)
- 9.8 CT: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Current/Future)
- 9.9 CT: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Future)
- 9.9a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Commercial/Industrial Worker (Future)
- 9.10 CT: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Child) (Future)
- 9.10a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Child) (Current/Future)
- 9.11 CT: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Adult) (Future)
- 9.11a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Adult) (Current/Future)
- 9.12 CT: Summary of Receptor Risks and Hazards for COPCs – Resident (Child) (Future)
- 9.12a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Child) (Future)
- 9.13 CT: Summary of Receptor Risks and Hazards for COPCs – Resident (Adult) (Future)
- 9.13a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Adult) (Future)
- 10.1 RME: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Older Child) (Current/Future)

- 10.2 RME: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Adult) (Current/Future)
- 10.3 RME: Summary of Receptor Risks and Hazards for COPCs – Utility Worker (Current/Future)
- 10.3a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Utility Worker (Current/Future)
- 10.4 RME: Summary of Receptor Risks and Hazards for COPCs – Construction Worker (Future)
- 10.4a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Construction Worker (Future)
- 10.5 RME: Summary of Receptor Risks and Hazards for COPCs – Surveillance Worker (Current/Future)
- 10.6 RME: Summary of Receptor Risks and Hazards for COPCs – Ditch Worker (Current/Future)
- 10.7 RME: Summary of Receptor Risks and Hazards for COPCs – Railroad Worker (Current/Future)
- 10.7a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Railroad Worker (Current/Future)
- 10.8 RME: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Current/Future)
- 10.9 RME: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Future)
- 10.9a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Commercial/Industrial Worker (Future)
- 10.10 RME: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Child) (Future)
- 10.10a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Child) (Current/Future)
- 10.11 RME: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Adult) (Future)
- 10.11a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Adult) (Current/Future)
- 10.12 RME: Summary of Receptor Risks and Hazards for COPCs – Resident (Child) (Future)
- 10.12a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Child) (Future)
- 10.13 RME: Summary of Receptor Risks and Hazards for COPCs – Resident (Adult) (Future)
- 10.13a RME: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Adult) (Future)
- 10.1 CT: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Older Child) (Current/Future)
- 10.2 CT: Summary of Receptor Risks and Hazards for COPCs – Trespasser (Adult) (Current/Future)
- 10.3 CT: Summary of Receptor Risks and Hazards for COPCs – Utility Worker (Current/Future)
- 10.3a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Utility Worker (Current/Future)
- 10.4 CT: Summary of Receptor Risks and Hazards for COPCs – Construction Worker (Future)
- 10.4a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Construction Worker (Future)
- 10.5 CT: Summary of Receptor Risks and Hazards for COPCs – Surveillance Worker (Current/Future)
- 10.6 CT: Summary of Receptor Risks and Hazards for COPCs – Ditch Worker (Current/Future)

- 10.7 CT: Summary of Receptor Risks and Hazards for COPCs – Railroad Worker (Current/Future)
- 10.7a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Railroad Worker (Current/Future)
- 10.8 CT: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Current/Future)
- 10.9 CT: Summary of Receptor Risks and Hazards for COPCs – Commercial/Industrial Worker (Future)
- 10.9a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Commercial/Industrial Worker (Future)
- 10.10 CT: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Child) (Future)
- 10.10a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Child) (Current/Future)
- 10.11 CT: Summary of Receptor Risks and Hazards for COPCs – Recreational Visitor (Adult) (Future)
- 10.11a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Recreational Visitor (Adult) (Current/Future)
- 10.12 CT: Summary of Receptor Risks and Hazards for COPCs – Resident (Child) (Future)
- 10.12a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Child) (Future)
- 10.13 CT: Summary of Receptor Risks and Hazards for COPCs – Resident (Adult) (Future)
- 10.13a CT: Summary of Receptor Risks and Hazards for COPCs – SYW-12 – Resident (Adult) (Future)

List of Acronyms

1,1,1-TCA - 1,1,1-Trichloroethane
1,1-DCE - 1,1-Dichloroethene
2,3,7,8-TCDD - 2,3,7,8-Tetrachloro-dibenzo-p-dioxin
ABS - Dermal Absorption Factor
ABS_{GI} - Gastrointestinal Absorption Efficiency
ADAF - Age Dependent Adjustment Factor
AF - Soil/Sediment-to-Skin Adherence Factors
AOS - Additional Area of Study
ARAR - Applicable or Relevant and Appropriate Requirements
AT - Averaging Time
AT-C - Averaging Time for Exposure to Potentially Carcinogenic Compounds
AT-NC - Averaging Time for Exposure to Non-Carcinogens
ATSDR - Agency for Toxic Substances and Disease Registry
B - Beta Constant
B&B - Blasland & Bouck
BMD - Benchmark Dose
BW - Body Weight
CalEPA - California Environmental Protection Agency
CAS - Chemical Abstract Service
CDI - Chronic Daily Intake
CERCLA - Comprehensive Environmental Response, Compensation, and Liability Act
cis-1,2-DCE - cis-1,2-Dichloroethene
COPC - Constituents of Potential Concern
CSF - Cancer Slope Factor
CT - Central Tendency
delta-BHC - delta-Benzenhexachloride
DL - Detection Limit
DSA - Dredge Spoil Area
ED - Exposure Duration
EF - Exposure Frequency
EMPC - Estimated Maximum Possible Concentration
EPAR - Exposure Pathway Analysis Report
EPC - Exposure Point Concentration
ET - Exposure Time
EU - Exposure Unit
EV - Event Frequency
FA - Fraction Absorbed
FS - Feasibility Study
HEAST - USEPA Health Effects Assessment Summary Table
HHRA - Human Health Risk Assessment
HSDB - Hazardous Substances Data Bank
InR - Inhalation Rate
IR - Ingestion Rate
IRIS - Integrated Risk Information System
IRM - Interim Remedial Measure
IRsoil - Incidental Ingestion Rate for soil

IRwater (potable) - Ingestion Rate for Drinking Water
IUR - Inhalation Unit Risk
 K_p - Permeability Coefficient
LEF - Lower East Flume
LOAEL - Lowest Observed Adverse Effect Level
MADEP - Massachusetts Department of Environmental Protection
MMOA - Mutagenic Mode of Action
MRL - Minimal Risk Levels
NAPL - Non-aqueous Phase Liquids
NCEA - USEPA National Center for Environmental Assessment
NCP - National Oil and Hazardous Substances Pollution Contingency Plan
NOAEL - No Observed Adverse Effect Level
NYSDEC - New York State Department of Environmental Conservation
NYSDOH - New York State Department of Health
NYSDOT - New York State Department of Transportation
PAH - Polycyclic Aromatic Hydrocarbon
PCB - Polychlorinated Biphenyls
PCDD - Polychlorinated Dibenzo-p-dioxin
PCDF - Polychlorinated Dibenzofuran
PCE - Perchloroethylene
PEF - Particulate Emission Factor
PPRTV - Provisional Peer-Reviewed Toxicity Values
PRG - Preliminary Remediation Goal
PSA - Preliminary Site Assessment
RAGS - Risk Assessment Guidance for Superfund
RBC - Risk Based Concentration
RCP - Reinforced Concrete Pipe
RfD - Reference Dose
RI - Remedial Investigation
RME - Reasonable Maximum Exposure
ROS - Regression on Order Statistics
SA - Skin Surface Area
STSC - USEPA Superfund Technical Support Center
SVOC - Semivolatile Compound
SYW-12 - SYW-12 State Wetland
 t^* - Time to Reach Steady State
TCE - Trichloroethene
TCLP - Toxicity Characteristic Leaching Procedure
TEF - Toxic Equivalency Factors
TEQ - Toxic Equivalent Concentration
 t_{event} - Event Duration
 τ_{event} - Lag Time Per Event
UCL - Upper Confidence Level
UEF - Upper East Flume
USEPA - United States Environmental Protection Agency
USEPA OSWER - USEPA Office of Solid Waste and Emergency Response
ve - Viable Epidermis
VF - Volatilization Factor
VOC - Volatile Organic Compound

Executive Summary

This human health risk assessment (HHRA) was performed to provide the necessary risk information needed to assist in the decision making process at the Wastebed B/Harbor Brook site (“Site”), in Geddes, and Syracuse, New York. The objective of the HHRA was to assess potential risks to human health associated with Site-related chemical substances under current and reasonably foreseeable future land uses and to facilitate the consideration and evaluation of possible future remedial actions. Health risks were evaluated for potential trespassers, workers (utility, construction, surveillance, drainage ditch, railroad, and commercial/industrial) recreational visitors, and hypothetical residents under current and future exposure scenarios.

Wastebed B/Harbor Brook Site originally consisted of four areas: 1) Harbor Brook, 2) the Lakeshore Area (including Wastebed B, the East Flume, Dredge Spoils Areas (DSA) #1 and #2, wetlands along the lakeshore, and the Route I-690 drainage ditch), 3) the Penn-Can Property and 4) the Railroad Area. Three additional areas were added to the HHRA at the request of NYSDEC and include Area of Study (AOS) #1, AOS #2, and SYW-12. AOS #1 is located east of Harbor Brook and to the north I-690 and AOS #2 is located to the south of I-690. The third area, SYW-12, is a wetland area situated along the northeastern shoreline of Onondaga Lake adjacent to the mouth of Ley Creek.

These areas have been used for a variety of purposes over the years. Harbor Brook, which originates southeast of Syracuse, New York drains a watershed of approximately 13.2 square miles and flows through the western side of Syracuse passing Wastebeds D and E, and discharges to the southwest corner of Onondaga Lake adjacent to the eastern end of Wastebed B. Historically, Wastebed B was used for the deposition of Solvay waste, a non-hazardous waste consisting primarily of calcium carbonate, calcium silicate and magnesium hydroxide with lesser amounts of sulfates, salts, and metal oxides. The East Flume, which was originally an excavated drainage ditch, primarily received process cooling waters from the former Main and Willis Avenue Plants. In addition to cooling waters, the East Flume in early history carried a combined (Solvay, sanitary, mercury, and organic) waste stream from the Main and Willis Avenue Plants to Onondaga Lake. The Dredge Spoils Areas (DSAs #1 and #2) received dredge spoils from the Upper East Flume and from Onondaga Lake, respectively. The I-690 Drainage Ditch appears to have been designed as a storm water drainage feature for the interstate and is currently maintained by the New York State Department of Transportation (NYSDOT). The Penn-Can Property, located to the south of Wastebed B, has historically been used for the production and storage of asphalt products. Currently, the Penn-Can Property is used by the Spano Container Corporation for the storage of equipment. The historical uses of the Railroad Area are not known, however, it does not appear to have been used for production or disposal purposes in the past. AOS #1 is a floodplain created by deposition of Onondaga Lake and Harbor Brook sediments during the 1950's and 1960's. There is also evidence that fill (non-Solvay waste) was likely placed during this time. AOS #2 includes parts of Wastebeds D and E. Lastly, SYW-12, which is currently an unoccupied wetland area, was historically part of the Iron Pier, a barge terminal for activities on Onondaga Lake.

Based on current conditions at the main portion of the Site (excluding SYW-12) and the nature of the surrounding area, the following current receptor populations were identified:

- Older child trespasser (Exposure Unit 1 – Site-Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)

- Adult trespasser (Exposure Unit 1 – Site-Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)
- Utility worker (Exposure Unit 1 – Site-Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)
- Surveillance worker (Exposure Unit 2 – Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2)
- Drainage Ditch worker (Exposure Unit 3 – I-690 Storm Sewer and Drainage Ditch)
- Railroad worker (Exposure Unit 4 – Railroad Area)
- Commercial/Industrial Worker (Exposure Unit 5 – Penn-Can Property)

Based on current conditions at the State Wetland SYW-12, the following current receptor pathways were identified:

- Child recreational visitor
- Adult recreational visitor
- Railroad worker
- Utility worker

Future land use at Wastebed B/Harbor Brook Site is likely to include all of the activities outlined above. It is also possible that additional industrial or commercial properties will be present on the Site, and the exposure areas located north of I-690 and near Onondaga Lake may be used for recreation. It is also possible, though extremely unlikely, that future residents and commercial/industrial workers could use Site ground water as potable water. Based on these considerations, the following receptors were identified under reasonably foreseeable future conditions:

- Older child trespasser (Exposure Unit 1 – Site-Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)
- Adult trespasser (Exposure Unit 1 – Site-Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)
- Utility worker (Exposure Unit 1 – Site-Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)
- Surveillance worker (Exposure Unit 2 – Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2)
- Ditch worker (Exposure Unit 3 – I-690 Storm Sewer and Drainage Ditch)
- Railroad worker (Exposure Unit 4 – Railroad Area)

- Commercial/Industrial worker (Exposure Units 5, 7 & 8 – Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area, SYW-12)
- Construction worker (Exposure Unit 1 – Site-Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)
- Child (< 6 years) resident (Exposure Units 6 & 8 – Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area, SYW-12)
- Adult resident (Exposure Units 6 & 8 – Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area, SYW-12)
- Child recreational visitor (Exposure Unit 6 – Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1)
- Adult recreational visitor (Exposure Unit 6)

Based on potential future conditions at the State Wetland SYW-12, the following receptor pathways were identified:

- Child recreational visitor
- Adult recreational visitor
- Railroad worker
- Utility worker
- Construction worker
- Commercial/Industrial worker
- Child resident
- Adult resident

Exposure media considered in both current and future scenarios include soil, sediment, surface water, ground water, fish tissue, and ambient air. Receptors that may be exposed to surface soils (0-2 ft bgs) include trespassers, utility workers, surveillance workers, railroad workers, commercial/industrial workers, residents, and recreational visitors. Construction workers, utility workers, commercial/industrial workers and residents may contact upper soils (0-10 ft bgs). Trespassers, utility workers, construction workers, ditch workers, and recreational visitors may be exposed to surface sediment (0-1 ft bgs). Utility workers and construction workers may be exposed to upper sediment (0-10 ft bgs). Trespassers, utility workers, construction workers, ditch workers, and recreational workers may be exposed to surface water.

The use of ground water at the Site for potable applications is considered hypothetical, and, as mentioned previously, extremely unlikely. The Site is unlikely to be developed as a residential area. Secondly, Site ground water is highly unlikely to be used as a drinking or industrial supply in the future, since the area is supplied by municipal water from Onondaga County Water Authority (OCWA). Lastly, the yield of the overburden ground water unit is inadequate for water supply wells and the high salinity of the deep aquifer (3,000 mg/l chloride) precludes its use as drinking water. However, because the use designation for this aquifer is classified as a potable water supply and the

National Contingency Plan states the ground water must be returned to its most beneficial use, this pathway was evaluated.

Risk and Hazard Summary

Hazard indices (HI) and cancer risks (CR) were derived based on the reasonable maximum exposure (RME) and central tendency (CT) concentrations for the identified receptor scenarios. In general, for exposure to media other than ground water, estimated current and future non-cancer hazards are within the acceptable regulatory limit ($HI < 1$) for most workers (surveillance, drainage ditch, railroad, commercial/industrial). For other receptors, estimated current and future non-cancer hazards exceed the acceptable regulatory threshold.

Estimated current and future cancer risks are within the acceptable regulatory range ($CR = 10^{-4}$ to 10^{-6}) for the surveillance, drainage ditch, and railroad workers. For other receptors, the estimated current and future cancer risks exceeded the acceptable regulatory threshold.

Although future use of ground water for potable water is unlikely, potential future exposure to ground water as potable water by residents and commercial/industrial workers was evaluated and found to pose unacceptable cancer risks and non-cancer hazards.

The calculated cancer risks and non-cancer hazards are summarized in the table below.

Summary of Current/Future Exposure Scenario Cancer Risks and Non-Cancer Hazards.

Timeframe	Receptor	Exposure Medium	Cancer Risk		Non-Cancer Hazards	
			RME	CT	RME	CT
Current/ Future	Older Child Trespasser	Fish Tissue	1×10^{-4}	3×10^{-5}	2×10^1	5×10^0
		Surface Sediment	7×10^{-4}	4×10^{-5}	7×10^{-1}	6×10^{-1}
		Surface Soil	2×10^{-4}	9×10^{-6}	1×10^0	1×10^{-1}
		Outdoor Air	9×10^{-8}	3×10^{-8}	7×10^{-4}	3×10^{-4}
		Surface Water	3×10^{-4}	2×10^{-4}	2×10^{-1}	8×10^{-2}
		All Media	1×10^{-3}	2×10^{-4}	2×10^1	6×10^0
Current/ Future	Adult Trespasser	Fish Tissue	8×10^{-4}	6×10^{-5}	3×10^1	6×10^0
		Surface Sediment	2×10^{-4}	2×10^{-5}	1×10^{-1}	5×10^{-2}
		Surface Soil	4×10^{-5}	4×10^{-6}	1×10^{-1}	6×10^{-2}
		Outdoor Air	5×10^{-7}	3×10^{-8}	8×10^{-4}	2×10^{-4}
		Surface Water	5×10^{-4}	8×10^{-5}	1×10^{-1}	7×10^{-2}
		All Media	2×10^{-3}	2×10^{-4}	3×10^1	6×10^0
Current/ Future	Utility Worker	Surf./Subsurface Sediment	1×10^{-4}	3×10^{-6}	7×10^0	$1 \times 10^{+3}$
		Surface/Subsurface Soil	1×10^{-4}	4×10^{-6}	4×10^{-1}	3×10^{-2}
		Outdoor Air	2×10^{-5}	2×10^{-6}	1×10^{-0}	1×10^{-1}
		Surface Water	2×10^{-4}	1×10^{-5}	7×10^{-2}	2×10^{-2}
		Shallow Ground Water	4×10^{-6}	3×10^{-7}	3×10^{-2}	7×10^{-3}
		All Media	4×10^{-4}	2×10^{-5}	8×10^0	$1 \times 10^{+3}$
Current/ Future	Utility Worker SYW-12	Surface/Subsurface Soil	1×10^{-5}	4×10^{-7}	6×10^{-2}	5×10^{-3}

Timeframe	Receptor	Exposure Medium	Cancer Risk		Non-Cancer Hazards	
			RME	CT	RME	CT
Current/ Future	Utility Worker SYW-12	Outdoor Air	9×10^{-9}	8×10^{-10}	1×10^{-4}	3×10^{-5}
		Shallow Ground Water	4×10^{-4}	4×10^{-5}	1×10^{-1}	3×10^{-2}
		All Media	4×10^{-4}	4×10^{-5}	2×10^{-1}	4×10^{-2}
Current/ Future	Surveillance Worker	Surface Soil	6×10^{-6}	2×10^{-6}	1×10^{-1}	1×10^{-1}
		Outdoor Air	7×10^{-7}	3×10^{-8}	1×10^{-3}	2×10^{-4}
		All Media	7×10^{-6}	2×10^{-6}	1×10^{-1}	1×10^{-1}
Current/ Future	Drainage Ditch Worker	Surface Sediment	2×10^{-6}	2×10^{-7}	2×10^{-2}	8×10^{-3}
		Outdoor Air	1×10^{-8}	2×10^{-9}	1×10^{-4}	6×10^{-5}
		Surface Water	2×10^{-7}	4×10^{-8}	3×10^{-2}	1×10^{-2}
		All Media	2×10^{-6}	2×10^{-7}	4×10^{-2}	2×10^{-2}
Current/ Future	Railroad Worker	Surface Soil	9×10^{-6}	2×10^{-6}	8×10^{-2}	7×10^{-2}
		Outdoor Air	2×10^{-8}	3×10^{-9}	2×10^{-3}	9×10^{-4}
		All Media	9×10^{-6}	2×10^{-6}	8×10^{-2}	7×10^{-2}
Current/ Future	Railroad Worker SYW-12	Surface Soil	4×10^{-5}	9×10^{-6}	2×10^{-1}	1×10^{-1}
		Outdoor Air	3×10^{-8}	6×10^{-9}	5×10^{-4}	3×10^{-4}
		All Media	4×10^{-5}	9×10^{-6}	2×10^{-1}	1×10^{-1}
Current/ Future	Commercial/Industrial Worker	Surface Soil	3×10^{-4}	4×10^{-5}	9×10^{-1}	3×10^{-1}
		Outdoor Air	1×10^{-7}	4×10^{-8}	6×10^{-3}	5×10^{-3}
		All Media	3×10^{-4}	4×10^{-5}	9×10^{-1}	3×10^{-1}
Current/Future	Child Recreator SYW-12	Surface Soil	4×10^{-4}	3×10^{-5}	9×10^{-1}	9×10^{-2}
		Outdoor Air	7×10^{-9}	2×10^{-9}	5×10^{-4}	2×10^{-4}
		All Media	4×10^{-4}	3×10^{-5}	9×10^{-1}	9×10^{-2}
Current/Future	Adult Recreator SYW-12	Surface Soil	1×10^{-5}	2×10^{-6}	4×10^{-2}	2×10^{-2}
		Outdoor Air	1×10^{-8}	8×10^{-10}	1×10^{-4}	3×10^{-5}
		All Media	1×10^{-5}	2×10^{-6}	4×10^{-2}	2×10^{-2}
		Outdoor Air	1×10^{-5}	3×10^{-6}	1×10^{-2}	1×10^{-2}
		Potable Water	6×10^{-2}	1×10^{-2}	9×10^1	9×10^1

Summary of Future Exposure Scenario Cancer Risks and Non-Cancer Hazards.

Timeframe	Receptor	Exposure Medium	Cancer Risk		Non-Cancer Hazards	
			RME	CT	RME	CT
Future	Child Recreator	Fish Tissue	3×10^{-4}	6×10^{-5}	4×10^1	1×10^1
		Surface Sediment	7×10^{-3}	1×10^{-3}	4×10^0	6×10^{-1}
		Surface Soil	1×10^{-4}	4×10^{-5}	3×10^0	3×10^{-1}
		Outdoor Air	5×10^{-7}	2×10^{-7}	3×10^{-3}	9×10^{-4}
		Surface Water	1×10^{-3}	8×10^{-4}	5×10^{-1}	2×10^{-1}
		All Media	9×10^{-3}	2×10^{-3}	5×10^1	1×10^1
Future	Adult Recreator	Fish Tissue	8×10^{-4}	6×10^{-5}	3×10^1	6×10^0
		Surface Sediment	2×10^{-4}	3×10^{-5}	2×10^{-1}	8×10^{-2}
		Surface Soil	3×10^{-5}	3×10^{-6}	2×10^{-1}	6×10^{-2}
		Outdoor Air	7×10^{-7}	5×10^{-8}	8×10^{-4}	2×10^{-4}
		Surface Water	5×10^{-4}	8×10^{-5}	2×10^{-1}	1×10^{-1}

Timeframe	Receptor	Exposure Medium	Cancer Risk		Non-Cancer Hazards	
			RME	CT	RME	CT
Future	Adult Recreator	All Media	2×10^{-3}	2×10^{-4}	3×10^1	6×10^0
Future	Construction Worker	Surf./Subsurface Sediment	6×10^{-5}	4×10^{-5}	1×10^1	9×10^0
		Surface & Subsurface Soil	5×10^{-5}	2×10^{-5}	4×10^0	2×10^0
		Outdoor Air	2×10^{-5}	5×10^{-6}	1×10^1	3×10^0
		Surface Water	8×10^{-5}	4×10^{-5}	9×10^{-1}	4×10^{-1}
		Shallow Ground Water	2×10^{-6}	9×10^{-7}	3×10^{-1}	2×10^{-1}
		All Media	2×10^{-4}	1×10^{-4}	3×10^1	1×10^1
Future	Construction Worker SYW-12	Surface & Subsurface Soil	5×10^{-6}	2×10^{-6}	7×10^{-1}	3×10^{-1}
		Outdoor Air	1×10^{-8}	3×10^{-9}	4×10^{-3}	9×10^{-4}
		Shallow Ground Water	2×10^{-4}	1×10^{-4}	2×10^0	8×10^{-1}
		All Media	2×10^{-4}	1×10^{-4}	2×10^0	1×10^0
Future	Commercial/Industrial Worker	Surface Soil	2×10^{-4}	3×10^{-5}	1×10^0	4×10^{-1}
		Outdoor Air	5×10^{-6}	2×10^{-6}	9×10^{-3}	8×10^{-3}
		Potable Water	4×10^{-3}	1×10^{-3}	6×10^1	5×10^1
		All Media	4×10^{-3}	1×10^{-3}	6×10^1	5×10^1
Future	Commercial/Industrial Worker SYW-12	Surface Soil	6×10^{-5}	8×10^{-6}	3×10^{-1}	1×10^{-1}
		Outdoor Air	1×10^{-7}	3×10^{-8}	2×10^{-3}	2×10^{-3}
		All Media	6×10^{-5}	8×10^{-6}	3×10^{-1}	1×10^{-1}
Future	Child Resident	Surface Soil	1×10^{-3}	4×10^{-4}	3×10^1	5×10^0
		Outdoor Air	8×10^{-6}	8×10^{-6}	5×10^{-2}	5×10^{-2}
		Potable Water	7×10^{-1}	1×10^{-1}	2×10^2	2×10^2
		Shower Vapor	9×10^{-3}	3×10^{-3}	5×10^2	2×10^2
		All Media	7×10^{-1}	1×10^{-1}	8×10^2	4×10^2
Future	Adult Resident	Surface Soil	9×10^{-5}	2×10^{-5}	7×10^{-1}	6×10^{-1}
		Outdoor Air	1×10^{-5}	3×10^{-6}	1×10^{-2}	1×10^{-2}
		Potable Water	6×10^{-2}	1×10^{-2}	9×10^1	9×10^1
		Shower Vapor	6×10^{-3}	2×10^{-3}	7×10^1	3×10^1
		All Media	7×10^{-2}	2×10^{-2}	2×10^2	1×10^2
Future	Child Resident SYW-12	Surface Soil	7×10^{-4}	3×10^{-4}	7×10^0	1×10^0
		Outdoor Air	1×10^{-7}	1×10^{-7}	9×10^{-3}	9×10^{-3}
		All Media	7×10^{-4}	3×10^{-4}	7×10^0	1×10^0
Future	Adult Resident SYW-12	Surface Soil	5×10^{-5}	8×10^{-6}	2×10^{-1}	1×10^{-1}
		Outdoor Air	2×10^{-7}	5×10^{-8}	2×10^{-3}	2×10^{-3}
		All Media	5×10^{-5}	8×10^{-6}	2×10^{-1}	1×10^{-1}

Conclusions

For a number of exposure scenarios and exposure pathways, the estimated current and future non-cancer hazards are within the acceptable regulatory limits (*i.e.*, cancer risk of 10^{-4} to 10^{-6} , hazard index of <1). For those scenarios that exceed these thresholds, RAGS Table 10 series provides a description of those constituents that are considered risk drivers.

The greatest cancer risk posed to current receptors is 2×10^{-3} for the adult trespasser and the greatest non-cancer hazard is 30 for the same receptor. The greatest cancer risk and non-cancer hazard posed to a potential future receptor is for the future child resident. The cancer risk of 7×10^{-1} is driven primarily by exposure to ground water as a drinking water source and to surface soil. The non-cancer

hazard of 8×10^2 is also driven primarily by exposure to ground water as a drinking water source and to surface soil. As noted previously, the use of ground water at the Site for potable applications is considered hypothetical and is extremely unlikely for several reasons: 1) the area is supplied by municipal water from OCWA; 2) the yield of the overburden ground water unit is inadequate for water supply wells; and 3) the high salinity of the deep aquifer (3,000 mg/l chloride) precludes its use as drinking water.

Because of the uncertainties inherent in the risk assessment process, none of the exposure and risk calculations presented in this report should be interpreted as precise measures of the true risk. Rather, all risks and hazards should be interpreted as uncertain estimates. Because many (but not all) of the approaches for dealing with uncertainty are intended to be conservative (*i.e.*, are more likely to overestimate than underestimate), the risk and hazard values above should generally be thought of as high-end estimates of the true risks and hazards, and actual values are probably somewhat lower than the calculated values.

1. Introduction

This is the Human Health Risk Assessment (HHRA) Report for the Wastebed B/Harbor Brook Site (Site) in Geddes and Syracuse, New York. A Site location plan is included as **Figure 1**. This HHRA was performed pursuant to the Administrative Consent Order (D-7-0001-00-02) between the New York State Department of Environmental Conservation (NYSDEC) and Honeywell International, Inc. (Honeywell) dated April 10, 2000 (NYSDEC 2000).

On February 19, 2004, an Exposure Pathway Analysis Report (EPAR) (O'Brien & Gere 2004a) was submitted to the NYSDEC for review. This deliverable consisted of USEPA's Risk Assessment Guidance for Superfund (RAGS) Part D, Tables 1 and 2. Subsequent to this submittal the NYSDEC requested that the RAGS Table 4 series be submitted. On March 26, 2004, Honeywell submitted a supplemental exposure pathway analysis report (EPAR) deliverable consisting of RAGS Part D Table 4 series. On January 21, 2005, the NYSDEC provided comments on the EPAR report. These comments and a proposed path forward for the completion of the remainder of the HHRA were submitted to the NYSDEC on February 21, 2005. A conference call was held with NYSDEC, USEPA, and Honeywell to discuss the February 21, 2005 response to comments on May 17, 2006.

A Supplemental Remedial Investigation (RI) Work Plan was submitted to the NYSDEC on July 6, 2006 based on an April 21, 2006 conference call between the NYSDEC and Honeywell. The Supplemental RI Work Plan was revised based on August 16, 2006 NYSDEC comments and resubmitted to the NYSDEC on September 15, 2006. This Work Plan was accepted by the NYSDEC on September 19, 2006. The HHRA was not advanced at that time due to the additional samples being collected as part of the Supplemental RI.

On June 4, 2007, Honeywell provided a letter to the NYSDEC regarding PAH surrogates at the Site. On September 12, 2007, Honeywell submitted the May 17, 2006 meeting notes and an interim schedule for completion of the HHRA.

On February 25, 2008, Honeywell submitted an interim HHRA deliverable, RAGS Part D Series Tables 1-6, which reflected Honeywell's February 21, 2005 response to comments letter and the subsequent May 17, 2006 conference call. On March 12, 2008, NYSDEC provided comments on the RAGS Part D Series Tables 1-6, and on March 14, 2008, Honeywell, O'Brien & Gere, NYSDEC, and USEPA met to discuss a subset of these comments. Following the meeting, Honeywell provided a response to comments letter dated April 14, 2008 reflecting the discussions at the meeting. The NYSDEC responded to this response to comments letter with some clarifying comments in a letter dated May 9, 2008. On June 6, 2008, Honeywell then submitted a final interim HHRA deliverable, RAGS Part D Series Tables 1-10, which reflected Honeywell's April 14, 2008 response to comments letter and NYSDEC's June 6, 2008 clarifying comments letter. On July 25, 2008, NYSDEC provided comments on the RAGS Part D Series Tables 1-10, to which Honeywell provided a response to comments on August 28, 2008. Honeywell then prepared a draft HHRA report reflecting Honeywell's August 28, 2008 response to comments letter.

On May 1, 2009, NYSDEC provided comments on the draft HHRA report to which Honeywell provided responses on June 11, 2009. On August 18, 2009, Honeywell, O'Brien & Gere, NYSDEC, and USEPA met to discuss a subset of these comments. On October 2, 2009, USEPA additionally provided specific comments on RAGS Part D Series Tables 2 related to vapor intrusion.

It should be noted that two interim remedial measures (IRMs) are ongoing in addition to the current Remedial Investigation/Feasibility Study (RI/FS) being conducted: the East Flume IRM, and the Wastebed B/Harbor Brook IRM.

1.1. Site Description and Background

The Wastebed B/Harbor Brook Site originally consisted of four areas: 1) Harbor Brook, 2) the Lakeshore Area (including Wastebed B, the East Flume, Dredge Spoils Areas (DSA) #1 and #2, wetlands along the lakeshore, and the Route I-690 drainage ditch), 3) the Penn-Can Property and 4) the Railroad Area. Two additional areas of study (AOS) were added at the request of NYSDEC and include areas east of Harbor Brook, to the north (AOS #1) and south (AOS #2) of I-690. A description of these areas, as well as the AOS east of Harbor Brook, is presented in Section 1.1, below. A Site Plan is included as **Figure 2**. The SYW-12 area was added to the Site during the Supplemental RI. The SYW-12 Site Plan is included as **Figure 3**. A brief description of the various areas comprising the Site is presented below.

1.1.1. Lakeshore Area

The Lakeshore Area is comprised of Wastebed B and areas along the shore of Onondaga Lake. The East Flume, Dredge Spoils Areas #1 and #2, and the I-690 Ditch are treated separately and not included in the Lakeshore Area. The Lakeshore area is approximately 3,200 feet wide (east to west) and 800 ft deep (north to south) and is situated along the southern shore of Onondaga Lake, near the southwest corner of the lake. The northern boundary of the Lakeshore Area is Onondaga Lake which limits access from the north, however, recreational boaters could potentially access the site from the water side. The Upper East Flume (UEF) (described below) defines the western extent of this area and the eastern extent is defined by Harbor Brook near its confluence with Onondaga Lake. The southern extent of the Lakeshore Area is defined by Route I-690. A fence to the south on the I-690 side and to the west requires entrance through a locked gate. The west is more open. It is possible for people on foot to cross the railroad tracks and access the Lakeshore Area. The ecological communities in the Lakeshore Area are representative of *successional old field*, *successional northern hardwoods*, *ditch/artificial intermittent stream*, and *freshwater wetland* habitats. Topography of the Lakeshore Area is generally flat with a relatively significant slope to the north in the north-central portion of the area due to the presence of a constructed berm (described below). A topographic map is included as **Figure 4**. A discussion of Wastebed B is provided below in this section.

1.1.2. Wastebed B

Historical use of Wastebed B was for the deposition of Solvay waste, a non-hazardous waste consisting primarily of calcium carbonate, calcium silicate and magnesium hydroxide with lesser amounts of sulfates, salts and metal oxides. Wastebed B received Solvay waste from approximately 1898 to 1926 (Blasland & Bouck (B&B) 1989). Wastebed B was engineered to receive waste by construction of a bulkhead into Onondaga Lake. The bed covers approximately 28 acres, including the relatively flat area between the lake water's edge and the raised, bermed portion of the wastebed (B&B, 1989). Between approximately 1898 and 1908, the filling of Wastebed B was initiated by construction of wooden bulkheads in the lake and placement of Solvay waste out to the bulkhead line. Coke plant waste from the former Main Plant Site may have been disposed of concurrent with the Solvay waste. Additionally, sewage sludge disposal occurred on the southeast portion of the bed in the late 1950's and early 1960's (B&B 1989). Modification of the shoreline has occurred due to erosional and depositional forces, as well as historical discharges from the East Flume.

Wastebed B is currently subject to an IRM directed towards mitigating discharges of contaminated ground water and NAPL into Harbor Brook and Onondaga Lake. This IRM consists of a vertical barrier and ground water collection system. It is currently proposed that the vertical barrier be installed along the Onondaga Lake shoreline perimeter of Wastebed B and upstream along the western bank of Harbor Brook for an estimated total length of 6,000 ft. The vertical barrier will be keyed into the silt and clay layer at approximate depths between 25 ft and 40 ft below ground surface. The actual depth and alignment of vertical barrier, and the depth and configuration of the collection system, will be selected during IRM design. The Final IRM Work Plan was submitted to the NYSDEC in July 2004 (O'Brien & Gere 2004b) and was approved in August 2004.

1.1.3. Penn-Can Property

The Penn-Can Property is situated to the south of the Lakeshore Area and south of I-690 (**Figures 2 and 5**). The property is surrounded by a fence on the North, East, and West sides and is accessible by a gate that is locked at night when current business is shut down. Access from the south is relatively inaccessible since the banks of the railroad tracks are extremely steep (nearly vertical in some instances). This property has historically been used, and is currently being utilized, for the production and storage of asphalt products. In 1919, the Barrett Division of the Semet Solvay Company of Allied Chemical Corporation began operations. Barrett produced various asphalt emulsions and some coal tar based products used in road construction. The primary constituents of these materials were asphalt, coal tar, caustic soda (sodium hydroxide) and muriatic acid (hydrochloric acid). Until 1975, the operation included a barge loading facility, which transferred emulsions to vessels on Onondaga Lake via above ground pipelines. These pipelines were removed, as well as the above ground storage tanks, during the 1978 decommissioning of the Barrett facility. In 1978, approximately 750 to 1,000 cubic yards of asphalt tank bottoms were buried on-site in a pit with dimensions of 40 ft wide, 165 ft long, and 7 ft deep. The tank bottoms were covered with 2 ft of low permeability fill, a geotextile, and 2 ft of fill. The pit was subsequently covered with a layer of crushed stone. The locations of historical tanks, and structures, and the approximate location of the pit, are shown on **Figure 5**. In 1983, the property was purchased by Penn-Can Road Materials, Inc. Currently the property is being used by Spano Container Corporation for the storage of equipment. The area is approximately 1,600 ft wide (east to west) and 450 ft deep (north to south) and consists of buildings, above ground storage tanks and a gravel parking lot, with limited vegetation around the periphery of the area. A shallow drainage swale runs along the southern and east perimeter of the property. The coetype in this area is classified as *urban structure interior*.

1.1.4. Railroad Area

The Railroad Area, owned by CSX, is situated to the south of the Penn-Can Property and is bounded to the north, south and east by rail tracks. There are no fences and anyone walking the railroad tracks can access the Railroad Area. The area is approximately 1,400 ft wide (east to west) and 400 ft deep (north to south). The coetype in this area is classified as *successional shrubland* in the southern portion and *urban structure interior* in the northern portion. Historical uses of this area are not known. Based on review of historical aerial photographs, the area appears to have been a vacant lot and has not been used for production or disposal purposes in the past.

1.1.5. Harbor Brook

The portion of Harbor Brook subject to this RI/FS is classified as a Class C stream by the NYSDEC. Harbor Brook originates southeast of Syracuse, New York in the Town of Onondaga, flows through the western side of Syracuse passing Wastebeds D and E, and discharges to the southwest corner of Onondaga Lake adjacent to the eastern end of Wastebed B. Harbor Brook drains a watershed of

approximately 13.2 square miles and has an average flow rate of 14.3 cubic feet per second (B&B 1989).

1.1.6. East Flume

The East Flume was originally an excavated drainage ditch that primarily received process cooling waters from the former Main and Willis Avenue Plants. In addition to cooling waters, in early history the East Flume also carried a combined (Solvay, sanitary, mercury, and organic) waste stream from the Main and Willis Avenue Plants to Onondaga Lake. The East Flume currently receives storm water from Solvay Paperboard, General Chemical Corporation, Berry Plastics (formerly) Landis Plastics and the Village of Solvay. It also receives process waters from the Trigen Syracuse Energy Corporation. Water depths within the flume typically range between 2 ft and 6 ft and channel width varies approximately from a minimum of 20 ft to a maximum of 150 ft. Accessibility and/or limitations to the area are described above in the Lakeshore Area description. The banks of the flume are vegetated primarily with *Phragmites australis*.

In 1977, the upper portion of the East Flume was re-constructed to serve as a holding pond for the process cooling waters prior to their entry into a thermal diffuser and subsequent discharge to the lake. The upper portion was widened to a maximum width of approximately 150 ft and deepened to a maximum depth of approximately 6 ft. The bottom (substrate) of the UEF is constructed of crushed stone underlain by a geotextile. At the eastern end of the UEF is the ground water pumping station (former thermal diffuser building) and a high level overflow dam constructed originally to allow cooling water to flow when the former diffuser pumps were turned off. The dam and a berm to the north separate the UEF from the Lower East Flume (LEF) (described below) and Onondaga Lake, respectively. Honeywell is required under the terms of its SPDES discharge permit (No. 0002275) to collect monthly and quarterly samples of surface water from downstream of the dam.

The LEF is a narrower channel that is approximately 25 ft wide with water depths of 3 to 4 ft. The LEF meanders to the south and east and discharges to Onondaga Lake. The LEF is not specifically classified by NYSDEC, therefore it receives the classification of the surface water to which it discharges (Onondaga Lake, Class C). The source of water in the LEF is primarily water from the UEF and, to a lesser degree, ground water. The LEF discharges to Onondaga Lake near the north-central portion of the Lakeshore Area. The LEF was not modified during the 1977 re-construction and maintains the original channelized drainage ditch configuration. The substrate of the LEF is primarily unvegetated sediment. Organic sediments, approximately 2 to 10 ft in depth, are underlain by solidified inorganic sediments.

The East Flume is currently subject to an on-going interim remedial measure (IRM). The IRM focuses on the elimination (to the extent practicable within the IRM scope) of potential impacts to wildlife resources, transport of contaminants to Onondaga Lake via East Flume sediment, and exposure to trespassers via dermal contact with UEF and LEF sediments. In the future the 42-inch diameter PA Sewer will be plugged and the 60-inch diameter RCP main sewer will be redirected into the 72-inch RCP outfall sewer. A 48-inch diameter metal sewer will extend the 72-inch diameter outfall sewer through the Willis Avenue barrier wall. Additional work includes transfer of surface water from the UEF and LEF into Onondaga Lake, excavation of sediment from the UEF and LEF, installation of a low permeability membrane and clean backfill, and restoration activities.

1.1.7. I-690 Drainage Ditch

The I-690 Drainage Ditch appears to have been designed as a storm water drainage feature for the interstate and is maintained by the New York State Department of Transportation (NYSDOT) (O'Brien & Gere 2001a). The drainage ditch lies between the fence for the Lakeshore Area to the North and the I-690 guardrail to the South. The ditch flows west to east, and discharges to Harbor Brook. Near the midpoint of the ditch, an outfall from the storm drainage system beneath I-690 discharges to the ditch. I-690 catch basins are covered with metal grates and would require a crowbar to remove. Access would require the person to cross the busy I-690 highway. The bank from I-690 to the ditch is relatively steep and contains thick vegetation. There is also a fence to the East which does not have a gate. Harbor Brook impedes people on foot from entering this area on foot. Portions of the drainage ditch are vegetated with *Phragmites australis*, goldenrod (*Solidago* sp.) and grasses (*Graminae*). The substrate of the drainage ditch primarily consists of weathered Solvay waste. Based on the USGS map for the area, historical aerial photographs of the area, and a 2000 Site reconnaissance, the ditch appears to have been constructed on portions of the wastebed. At the time of the Site reconnaissance, the NYSDOT had recently removed accumulated sediments from the drainage ditch to allow for less restricted flow of intermittent surface water. Sediment samples were collected subsequent to the sediment removal by NYSDOT. Samples were collected from the I-690 ditch in May 2001 as part of the PSA and in June 2003 as part of the RI. The substrate at the time of sampling was a mixture of Solvay waste with some sediment.

1.1.8. Dredge Spoils Areas

Dredge Spoils Areas (DSAs) #1 and #2 are located in the northwestern portion of the Lakeshore Area. Accessibility and/or limitations to the area are described above in the Lakeshore Area description. The areas received dredge spoils from the UEF and from Onondaga Lake, respectively. DSA #1 is situated to the south of the UEF and is approximately 300 ft by 300 ft at its widest points (**Figure 2**). This area was created in 1979 to hold sediments removed from the UEF that had been deposited within the UEF subsequent to the 1977 construction. A berm was created around the perimeter of the area and sediments were pumped into the bermed area. The average depth of these sediments is 2 ft. Beneath the spoil materials, a layer approximately 1 to 2 ft thick of ash and cinders has been observed (O'Brien & Gere, 1999). DSA #2 is located to the east of the UEF and south of the LEF. The area is approximately 350 ft by 350 ft and bermed to the north and east. This area received sediments from the lake, which were removed during installation of the thermal diffuser pipe in 1977. The spoils in this area are approximately 3 to 5 ft thick and are underlain by Solvay waste.

1.1.9. Additional Areas of Study (AOS) #1 and #2

AOS #1 is a wetland area situated east of Harbor Brook and adjacent to the Lakeshore Area (**Figure 2**) and is accessible for people on foot to cross the railroad tracks or by boat from Onondaga Lake. AOS#1 is often inundated with water in the winter and spring. This area is part of NYS wetland SYW-19 (NYSDOT 1973). Based on review of historical aerial photographs, this area is a floodplain created by deposition of Onondaga Lake and Harbor Brook sediments during the 1950's and 1960's. There is also evidence that fill (non-Solvay waste) was likely placed during this time.

AOS #2 is situated east of Harbor Brook and south of I-690 between Harbor Brook and the top of the Wastebeds D and E berm (**Figure 2**). This area can be accessed from the West by an old bridge that goes over Harbor Brook or from the east via Hiawatha Blvd. The Hiawatha Blvd. entrance would require a receptor to enter through one of the two car dealerships. Steep slopes associated with I-690 limit access from the North and South. Wastebeds D and E have a combined surface area of approximately 44 acres. Aerial photos indicate that these beds were inactive by 1926 (B&B 1989).

1.1.10. State Wetland SYW-12

SYW-12 is a 40.7 acre wetland area situated along the northeastern shoreline of Onondaga Lake and to the south of Ley Creek. This area was delineated as part of the Revised Onondaga Lake Wetland / Floodplain Assessment Draft Report (Parsons and O'Brien & Gere 2009). The area is bounded by railroad tracks to the east and the Lake to the west. Onondaga Lake provides access to the location from recreational boating. In addition to the CSX railroad tracks there are access roads adjacent to the tracks. These roads are unpaved and are used by CSX rail workers to access tracks for maintenance. These roads are also used by utility workers to access the Site as needed to maintain the on-Site utilities. A fence is located to the East between the railroad tracks and Carousel Mall. I-81 and the Park Street off-ramp lie to the North. Vehicles or pedestrians would be required to access this location from a gate under the elevated off-ramp. This gate tends to be locked. The primary vegetation in this area includes a monoculture of common reed and a forested floodplain consisting of cottonwoods.

1.1.11. Hypothetical Potable Water Source Area (Site-Wide)

The use of ground water at the Site for potable applications is considered hypothetical. The Site is unlikely to be developed as a residential area. However, this pathway has been evaluated because the use designation for this aquifer is classified as a potable water supply, and the National Contingency Plan states the ground water must be returned to its most beneficial use. Therefore, this source area consists of all ground water data collected at the Site from any depth. It should be noted that "Site-Wide" for this exposure medium refers to all exposure areas, including SYW-12, which for other media is evaluated distinctly from the rest of the Site.

1.2. Data Sources

Field investigation activities executed in support of the Site investigation and risk assessments involved the collection and analysis of a large number of samples of various media at the Site (surface soil, subsurface soil, surface sediment, seep sediment, wetland sediment, ground water, surface water, seep water, and indoor air). Samples have been analyzed for a range of analytes, including volatile and semivolatile organics, metals, dioxins/furans, polychlorinated biphenyls, pesticides, wet chemistry parameters, as well as other compounds. The analytes identified detectable levels of targeted compounds in each of the sampled media.

Figures 6, 6A-I, and 7 present site sampling locations. A copy of the HHRA database (data set) is provided as **Appendix A**. A comprehensive list of samples used in this assessment, sorted by exposure area is provided as **Appendix B**. This appendix presents information such as start and end depths, geographic coordinates, sample dates, and matrix type for each exposure area and medium. Since not all chemicals are present in each sample, the number of data points shown in the RAGS D Table 2 series may be smaller than the number of data points listed in Appendix B. Because of the size of these appendices, both of these appendices are provided only as electronic files in this submittal; no print copies have been supplied. **Attachment A** of this report includes the RAGS Part D Tables. Site sample locations are depicted in **Figures 6, 6A-I, and 7**.

The table column headings used in **Appendix A** are defined below.

Exposure Area: Refers to a specific area of the Wastebed B/Harbor Brook Site. These include Harbor Brook, East Flume, Lakeshore Area, Penn-Can Property, Railroad Area, Interstate-690 Storm Sewer and Drainage Ditch, Dredge Spoil Area #1 (DSA #1), Dredge Spoil Area #2 (DSA #2), Additional

Area of Study #1 (AOS #1), Additional Area of Study #2 (AOS #2), and SYW-12. For the bulk of this assessment (RAGS Table 3 Series and beyond), these exposure areas were grouped into Exposure Units (See Section 2).

Sample Location: This column presents the specific field sample number that correlates to the sample locations on **Figures 6, 6A-I, and 7**.

Start Depth: The depth interval from which the sample collection began (measured from the ground surface or the top of the sediment/water interface). For ground water samples, this value represents the top of the well screen. The vapor samples were collected from a discrete depth (the start depth and end depth are the same).

End Depth: The depth interval from which the sample collection ended (measured from the ground surface or the top of the sediment/water interface to the deepest part of the sample). For ground water samples, this value represents the bottom of the well screen. The vapor samples were collected from a discrete depth (the start depth and end depth are the same).

System Type Code: The following is a clarification of the sample type codes in the **Appendix A** data set:

- EFSED – East Flume sediment
- GP – Geoprobe (soil sample)
- GWS – Ground water screening sample collected at the water table from a temporary well installed in the soil boring during advancement of the boring
- HP – Hydroprobe (ground water sample)
- MW – Monitoring well (ground water sample)
- OUT – Outfall 015 (East Flume surface water)
- QC – Quality control sample
- SB – Soil boring
- SC – Soil vapor
- SED – Sediment sample
- SP – Seep sample
- SS – Surface soil sample
- STW – Storm water sample
- SW – Surface water sample
- TCLP – Soil or sediment sample on which leachate was analyzed
- TP – Soil sample collected from a test pit
- WSD – Wetland sediment (SYW-12 exposure area soil)

The “EFSED” samples were samples collected by O’Brien & Gere and the NYSDEC during the Willis Avenue RI in 1997 and 1999. The “SED” samples in the lower East Flume are samples that were collected in 1993 as part of the Onondaga Lake RI. The QC samples and the TCLP samples were not used in the quantitative analysis. In addition, tar samples were excluded because Site drums containing tar were removed by NYSDOT as part of the I-690 bridge project. Because these samples are not usable, they do not appear in the **Appendix A** Site data set, but are noted in **Appendix B** as not utilized in the HHRA.

Sample Matrix: The sample matrix code is “Soil” for soil and sediment and “Water” for surface water and ground water. While there is no “Oil” matrix, samples HB-T-3-OIL and HB-T-5-OIL were collected during the Harbor Brook Sediment IRM (2001) by Blasland, Bouck, and Lee. These samples consisted of NAPL collected from the sediment cores advanced at these transects.

Sample Date: Date that the sample was collected.

CAS Number: Chemical Abstract Service (CAS) registry numbers are unique numerical identifiers for chemical compounds.

Chemical: Name of analyte.

Concentration: Value that represents the amount of a given substance in a given volume. In **Appendix A**, the polychlorinated biphenyl (PCB) concentrations are represented as individual Aroclors (instead of the “Less chlorinated”, “Highly chlorinated”, and “Total PCBs” groupings that appear in the RAGS Tables [see discussion below in Section 3.1]). Likewise, this database presents the dioxin/furan congeners, not just the 2,3,7,8-tetrachloro-dibenzo-p-dioxin (2,3,7,8-TCDD) toxicity equivalent values. The RAGS 2 Table Series presents the results of these conversions.

Unit: Unit of chemical concentration. All non-aqueous data are reported in mg/kg, µg/kg, or ng/kg on a dry-weight basis. Surface water and ground water data are reported in mg/L, µg/L, or ng/L. Soil vapor data are reported in µg/m³.

Detection Flag: This column indicates whether the result in the concentration column was identified as a detected concentration or not. If it was not detected, the concentration represents the reporting limit.

Interpreted Qualifier: Data with the following qualifiers were included in the quantitative analysis: No qualifier, J, UJ, U, and EMPC (J for dioxin/furan). Only “B” NYSDEC data which were validated were included.

1.2.1. Development of the Data Set

Data utilized for this evaluation are the result of the data collection efforts targeted to support the characterization of the Site through the RI/FS process and investigations performed prior to the onset of Site PSA/RI/FS. As a result, analytical data has been collected over significant spatial and temporal scales by multiple investigators. It should also be noted that fish tissue data used within this risk assessment was collected as part of the Onondaga Lake RI (NYSDEC 2002).

Table 1 presents a summary of the data collected during the previous investigations, preliminary site assessment (PSA), remedial investigation (RI), and supplemental RI that are being utilized in this risk assessment. These data collection events are described here. In general, data collected over multiple collection events for the same location have been given equal weight in the HHRA.

1.2.1.1. Previous Investigations

Previous studies, performed prior to the PSA, RI and Supplemental RI, are described below:

Lower East Flume Sediment Sampling Performed by PTI as part of the Onondaga Lake RI

Surface water data were collected from the LEF by PTI (now Exponent) as part of the Onondaga Lake RI. Samples were collected monthly from April to December 1992, during both low flow and high flow conditions. Samples were analyzed for target compound list/target analyte list (TCL/TAL) compounds.

As part of the Onondaga Lake RI, PTI also collected fifteen sediment samples from the 0-2 cm (0 to 0.07 ft) interval at five locations within the LEF in 1993. One sample from each location was analyzed for TCL/TAL compounds, grain size, total organic carbon (TOC), chloride and calcium carbonate.

Willis Avenue RI East Flume Sediment Sampling

Sediment sampling in the East Flume was conducted during Phases 2 and 3 of the Willis Avenue RI. The number of samples collected and analyses performed for each phase of the Willis Avenue RI is summarized below and discussed in the succeeding paragraphs.

Phase 2

Eight core samples were collected from the UEF during Phase 2 to characterize sediments within the flume. The UEF is the area located between the P.A. Sewer/Main Sewer outfall and the spillway adjacent to Onondaga Lake on the northwest portion of the Lakeshore Area. Sample designations and core lengths were: EF#1 (0 to 1.5 ft), EF#2 (0 to 2.2 ft), EF#3 (0 to 3 ft), EF#4 (0 to 1.5 ft), EF#5A (0 to 2 ft), EF#5B (3 to 4 ft), EF#6 (0 to 3.25 ft), and EF#7 (0 to 2.25 ft). In addition, one sediment core ((EF#8 (0 to 1 ft)) was collected down gradient of the East Flume spillway. Analyses included eight samples from seven locations for volatile organic compounds (VOCs), PCBs/pesticides, and mercury and one sample from one location for PCDD/PCDFs and TOC.

Phase 3

A total of 19 sediment samples were collected from the East Flume during Phase 3. Seven shallow sediment samples (0 to 0.5 ft) and one deep sediment sample (UEF-6 (0.5 to 2.6 ft)) were collected from the UEF. Analyses included seven samples from seven locations for TCL/TAL parameters and PCDD/PCDFs and TOC and 12 samples from 12 locations for PCDD/PCDFs and TOC.

Shallow sediments were analyzed for TCL/TAL parameters, PCDD/PCDFs, and TOC, and deep sediments were analyzed for PCDD/PCDFs and TOC. In addition, the NYSDEC collected two samples from the UEF: UEF-6 (0.5 to 1.5 ft) and UEF-6 (1.5 to 2.5 ft). NYSDEC samples were analyzed for PCDD/PCDFs and TOC.

Five shallow (0 to 0.5 ft) and six deep (0.5 ft to refusal) sediment samples were collected from the lower East Flume (LEF). Shallow and deep samples were analyzed for PCDD/PCDFs and TOC. In addition, the NYSDEC collected five samples from the LEF: LEF-1 (0.5 to 1.5 ft), LEF-1 (1.5 to 2.3 ft), LEF-2 (0.5 to 1.5 ft), LEF-2 (1.5 to 2.3 ft), and LEF-3 (0 to 0.5 ft). The NYSDEC samples were analyzed for PCDD/PCDFs and TOC. The NYSDEC sample LEF-2 (1.5 to 2.3 ft) was also analyzed for VOCs, semivolatile organic compounds (SVOCs), and PCBs/pesticides. The NYSDEC also collected a sample at EF#8 (0.5 to 1.2 ft) during Phase 3. This sample was analyzed for PCDD/PCDFs and TOC.

Surface water in the East Flume has been sampled under SPDES permit since 1980. An NPDES permit was in place from 1973 to 1980. The most recent quarterly and monthly data are utilized

herein. Water samples are collected twice a month and analyzed for chlorinated phenols. Monthly samples are collected and analyzed for ammonia nitrogen, total phosphorous, chloride, total dichlorobenzenes, mercury, and antimony. Quarterly samples are analyzed for oil & grease, total dissolved solids, total suspended solids, fecal coliform, total coliform, aluminum, arsenic, zinc, cadmium, copper, lead, iron, manganese, nickel, and chromium.

Harbor Brook Sediment Sampling by O'Brien & Gere in November 1996

In November 1996, and concurrent with NYSDEC sampling discussed above, O'Brien & Gere sampled sediment from Harbor Brook at Honeywell's request. Twelve sediment samples were collected from eight locations within Harbor Brook, including one sample upstream of the Site. The samples were collected from the 0 to 12-inch depth interval and from 12 inches to refusal (maximum of 26.5 inches). Samples were analyzed for VOCs, SVOCs, PCBs, pesticides, and inorganics. Two surface water samples were also collected and analyzed for inorganics as part of this sampling effort.

Harbor Brook Surface Water and Sediment Sampling Performed by NYSDEC in November 1996 and October 1997

Surface water and sediment within Harbor Brook were sampled by NYSDEC in November 1996 and October 1997. NYSDEC collected 20 sediment samples during this effort. The samples were analyzed for VOCs, SVOCs, PCBs and metals. These data were not used in the risk assessment. These data as provided did not have sample depths included with some samples and many of the samples were co-located with more recently collected sediment samples.

Harbor Brook Seep Sample

In April 1999, a ground water seep was discovered along the bank of Harbor Brook downstream of the bridge that traverses the brook north of I-690. Both Honeywell and NYSDEC sampled the seep. Honeywell analyzed the sample for VOCs, SVOCs, PCBs, pesticides and metals. The data from the Honeywell sample was utilized in the risk assessment and the NYSDEC data was not used because it was considered a duplicate.

Harbor Brook Sediment IRM Investigation

In July 2001, the Harbor Brook Sediment IRM Investigation Report (BBL 2001) was issued. The investigation included three tasks: 1) sediment probing, 2) Harbor Brook sediment sampling, and 3) wetlands soil borings. Sediment probing was conducted in March 2000 along 55 transects, 50 to 70 feet apart, extending from the mouth of Harbor Brook to Hiawatha Boulevard. In January and February 2001, sediment samples were collected from 18 cores. Eighty-one sediment samples and two dense non-aqueous phase liquid (DNAPL) samples were collected and submitted for laboratory analyses. Seventy discrete samples were analyzed for VOCs, SVOCs, pesticides, PCBs, metals, total mercury, cyanide and TOC and 42 samples (10 composites, 32 discrete) were analyzed for PCDD/PCDFs. Also, two full length cores were analyzed for VOCs, SVOCs, pesticides and metals using TCLP extraction methods. The two DNAPL samples were collected from locations T-3-2 (6 to 10 ft) and T-5-1 (6 to 18 ft) and analyzed for VOCs, SVOCs, pesticides, PCBs, metals, total mercury, cyanide, TOC and PCDD/PCDFs.

Onondaga Lake RI/FS Phase 2A

The Onondaga Lake RI/FS Phase 2A sampling was conducted during August 2000 (TAMS 2002b). As part of this effort, sediment samples were collected from four locations (S383, S384, S385, and S386) within wetlands on the Wastebed B/Harbor Brook Site. Samples were collected at depths of 0 to 0.5 ft and 0.5 to 1 ft using a piston corer. The wetland samples were analyzed for VOCs, SVOCs,

pesticides, PCBs (including Aroclor 1268), metals (including cyanide), PCDD/PCDFs, TOC and total solids.

During the summer of 2000 wetland sediment samples were collected at SYW-12. Four locations (S387, S388, S389, and S390) had samples collected from 0 to 15 cm and 15 to 30 cm. Samples were analyzed for VOCs, SVOCs, pesticides, PCBs, inorganics, TOC, and percent solids.

These data were not included in the risk assessment database. These samples were not considered necessarily representative of Site conditions thereby increasing uncertainty in the database.

Onondaga Lake Wetlands Subsurface Investigation Report (SYW-12)

The Wetland Subsurface Investigation was performed in May 2000 (C&S Companies, 2001). Thirteen subsurface borings were advanced as part of this investigation. Each of the shallow borings was advanced utilizing tripod mounted split spoon sampling apparatus. Borings were advanced to characterize subsurface soils and identify the potential existence of contamination.

Soil samples were collected from the following three intervals:

- Interval 1: existing grade to approximately 6 to 12 inches below ground surface (bgs)
- Interval 2: from 6 or 12 inches below grade to a depth of the proposed finished wetland elevation
- Interval 3: from 6 inches immediately above the proposed wetland finished grade elevation to a depth of 18 to 20 inches below the proposed wetland finished grade elevation.

Soil samples were collected and submitted to Friend Laboratories, Inc. for the following analyses:

- Samples collected from each of the three sampling intervals for Target Analyte List (TAL) metals, pH, and total organic carbon (TOC).
- Samples collected from Interval 3 were analyzed for Target Compound List (TCL) VOCs, SVOCs, PCBs, and pesticides.
- One half of the samples collected from Intervals 1 and 2 were analyzed for TCL VOCs, SVOCs, PCBs, and pesticides.

Three shallow ground water monitoring wells (B-4W, B-8W, and B10W) were installed in boreholes B-4, B-8, and B-10. Wells were constructed of 2-inch PVC screen and risers. The screen consisted of 0.01-inch slots. The wells were sampled for TCL/TAL parameters and pH.

SYW-12 Wetlands Mitigation Sampling

Four hand augered holes (M1A, M1B, M2A, and M2B) were advanced as part of this investigation. Three soil samples from these holes were submitted for RCRA TAL metals analysis for arsenic, cadmium, chromium, lead, and mercury. These samples were not included in the human health risk assessment database. These samples were not validated and the sampling conducted during the supplemental RI is considered sufficient to quantify risk at SYW-12.

Willis Avenue RI Dredge Spoils Area Soil Borings. During Phase 3 of the Willis Avenue RI, two soil borings were advanced using direct push drilling techniques (Geoprobe Macrocore) in Dredge Spoils Area #2 (HB-DSA#2-B1 and HB-DSA#2-B2). The borings were advanced to evaluate the hazardous characteristics of the black organic material observed at approximately 5 ft below ground surface (bgs) during excavation of test pits during Phase 2 of the Willis Avenue RI.

Willis Avenue Dredge Spoils Area Test Pits

Test pits were excavated in DSA #1 and DSA #2 during Phases 2 and 3 of the Willis Avenue RI.

Dredge Spoils Area #1

The following test pits were excavated and soil samples were collected in the DSA#1 during Phase 2 of the Willis Avenue RI:

- HB-DSA#1 NETP: 100 ft x 3 ft x 6 ft (3 samples; 1 each at 0 to 2 ft, 2 to 5 ft, and 6 ft)
- HB-DSA#1 NWTP: 180 ft x 3 ft x 4 ft (3 samples; 1 each at 0 to 2 ft, 3 to 4 ft, and 5 ft)
- HB-DSA#1 SETP: 125 ft x 3 ft x 6 ft (1 sample at 0 to 2 ft)
- HB-DSA#1 SWTP: 125 ft x 3 ft x 6 ft (no samples collected)
- HB-DSA#1 CENTER TP 40 ft x 3 ft x 6 ft (no samples collected)

The following test pits were excavated and soil samples were collected from DSA#1 during Phase 3:

- HB-DSA#1 NETP: 5 ft x 3 ft x 6 ft (1 composite sample at 6 ft)
- HB-DSA#1 SETP: 5 ft x 3 ft x 6 ft (1 composite sample at 5 ft)

Dredge Spoils Area #2

During Phase 2 of the Willis Avenue RI, the following test pits were excavated and samples collected in the DSA#2:

- HB-DSA#2 TP1: 150 ft x 3 ft x 6 ft (1 sample at 5 ft)
- HB-DSA#2 TP2: 75 ft x 3 ft x 6 ft (1 sample at 5 ft)
- HB-DSA#2 TP2A: 10 ft x 3 ft x 6 ft (no samples collected)
- HB-DSA#2 TP3: 10 ft x 3 ft x 10 ft (no samples collected)

1.2.1.2. Preliminary Site Assessment

The Harbor Brook PSA field activities were performed during the summer of 2000 and the winter of 2001. The PSA included sampling of soils via test pits, geoprobe borings and soil borings, ground water via hydropunch samples and monitoring wells, and surface water and sediment. The number of samples collected and analyses performed for the PSA is summarized below.

PSA Surface Soil Sampling

During the completion of the PSA, a total of 54 surface soil samples were collected from a depth of 0 to 2 inches. The surface soils were collected in conjunction with geoprobe boring, soil boring, and wetland boring locations. The number of samples collected at each sub-area is listed below:

- thirty-four surface soil samples collected at the Lakeshore Area (which includes six surface soil samples collected with the wetland soil borings)
- eleven surface soil samples collected at the Penn-Can Property
- nine surface soil samples collected at the Railroad Area.

PSA Soil Borings

During the completion of the PSA soil borings were advanced at seventeen locations at the Site to characterize subsurface soils and facilitate monitoring well installation. The number of soil borings and the number of samples collected at each sub-area is listed below:

- eight soil borings were advanced and nine subsurface samples collected at the Lakeshore Area
- four soil borings were advanced and six subsurface samples collected at the Penn-Can Property
- three soil borings were advanced and three subsurface samples collected at the Railroad Area.

The PSA soil borings were completed in two phases. During Phase 1, soil borings were advanced at the Lakeshore Area between July 19 and August 2, 2000. Phase 2 soil borings were advanced at the Penn-Can Property and Railroad Area between February 26 and March 27, 2001, subsequent to the execution of access agreements between Honeywell and the owners of the properties.

PSA Subsurface Sampling – Geoprobe Borings

During the completion of the PSA, geoprobe borings were advanced at thirty-four locations. The geoprobes were advanced adjacent to test pit excavation locations. The number of samples collected at each sub-area is listed below:

- twenty geoprobe borings were advanced and twenty-one subsurface samples collected at the Lakeshore Area
- eight geoprobe borings were advanced and eight subsurface samples collected at the Penn-Can Property
- six geoprobe borings were advanced and six subsurface samples collected at the Railroad Area.

The geoprobe borings at the Lakeshore Area were advanced between July 10 and July 18, 2000. The geoprobes at the Penn-Can Property and Railroad Area were advanced between February 26 and March 27, 2001. The geoprobe borings were advanced two feet into native materials (*i.e.*, marl or silt/fine sand) using direct push drilling techniques.

PSA Subsurface Sampling - Wetland Soil Borings

During the PSA, six borings (HB-HBW-01 through HB-HBW-06) were advanced in wetland areas WL2, WL4, and WL5 situated in the Lakeshore Area. Six subsurface samples were collected from these borings.

These borings were advanced using the same methods as the geoprobe borings described above in this report. The wetland soil borings were advanced from August 4 to August 8, 2000 to further evaluate the subsurface conditions at the Site.

PSA Subsurface Sampling – Test Pits

Test pits were advanced in two phases. The first phase of test pits was performed between July 5, 2000 and July 19, 2000 at the Lakeshore Area. The second phase of test pits was completed from February 26, 2001 to March 8, 2001, subsequent to access agreements being obtained for the Penn-Can Property and the Railroad Area.

A total of 48 test pits were advanced during the PSA using a tracked excavator to evaluate the physical and chemical characteristics of shallow subsurface soils (0 to 10 ft) at the Site. Test pits were excavated to be approximately 50 ft in length, 3 ft wide, and 10 ft deep.

The following TCL/TAL samples were collected from the test pits during the PSA:

- Eighteen analytic samples were collected for TCL/TAL analyses from the 32 test pits at the Lakeshore Area

- Eight analytic samples were collected from eight test pits at the Railroad Area
- Eight analytic samples were collected from eight test pits at the Penn-Can Property

PSA Surface Water Sampling

One round of surface water samples was collected during the PSA. Samples were collected from 11 locations on May 7 and 8, 2001. The number of locations sampled at each of the sub-areas of the Site during the PSA is presented below.

- five samples collected at five locations within Harbor Brook
- three samples collected at three locations at Penn-Can Property
- one sample collected at two locations at Railroad Area (one location was dry; therefore, a sample was not collected)
- two samples collected at three locations within I-690 drainage ditch on Lakeshore Area.

No surface water samples were collected at locations HBSW-4 and HBSW-11 due to the absence of surface water.

PSA Sediment Sampling

Sediment samples were collected from eight locations from 0 to 0.5 ft during the PSA. The samples were collected between May 7 and 8, 2001. The number of locations sampled at the various sub-areas of the Site is presented below.

- 3 samples collected at 3 locations at Penn-Can Property
- 2 samples collected at 2 locations at Railroad Area
- 3 samples collected at 3 locations within I-690 drainage ditch on Lakeshore Area.

PSA Ground Water Sampling

Two rounds of ground water samples were collected during the PSA from newly installed wells in the Lakeshore Area, and one round of ground water samples was collected from new and existing wells at the Penn-Can Property and Railroad Area. Samples were collected from September 26 to September 28, 2000 and May 10 to May 22, 2001. The May 2001 round encompassed all three sub-areas and the sampling was performed during a time of high ground water elevations.

PSA Ground Water Screening

During the PSA, ground water screening samples (HB-HP-01 through HB-HP-08) were collected along the northern boundary of the Lakeshore Area from eight locations to aid in the selection of soil boring and monitoring well locations. The samples were submitted to O'Brien & Gere Laboratories for TCL/TAL analyses by USEPA SW846 methods.

1.2.1.3. CSX Supplemental Sediment Sampling

Subsequent to the PSA sampling and prior to the initiation of the RI sampling, four sediment samples were collected from Harbor Brook at the request of the NYSDEC. Two samples (CSXSED-1 and CSXSED-2) were collected from underneath the CSX rail bridge, and two samples (HBSSED-14 and HBSSED-15) were collected immediately downstream of the bridge on November 14, 2002.

1.2.1.4. Remedial Investigation

The Harbor Brook RI field activities were performed between November 2002 and May 2004. The RI included sampling of soils via test pits, geoprobe borings and soil borings, ground water via

hydropunch samples and monitoring wells, and surface water and sediment. The number of samples collected and analyses performed for the RI is summarized below.

RI Surface Soil and Wetland Sampling

During the completion of the RI, surface soil samples were collected from depths of 0 to 6 inches and 6 to 12 inches. The surface soils were collected in conjunction with soil boring locations and selected surface soil sampling locations. The number of samples collected at each sub-area is listed below:

- 27 surface soil samples (14 from 0 to 0.5 ft and 13 from 0.5 to 1 ft) were collected at the Lakeshore Area
- 8 surface soil samples (4 from 0 to 0.5 ft and 4 from 0.5 to 1 ft) were collected at the Penn-Can Property
- 4 surface soil samples (2 from 0 to 0.5 ft and 2 from 0.5 to 1 ft) were collected at the Penn-Can Property as part of the Penn-Can Drum Survey
- 9 surface soil samples (5 from 0 to 0.5 ft and 4 from 0.5 to 1 ft) were collected at the Railroad Area
- 20 surface soil samples (10 from 0 to 0.5 ft and 10 from 0.5 to 1 ft) were collected at AOS #1
- 2 surface soil samples (1 from 0 to 0.5 ft and 1 from 0.5 to 1 ft) were collected at AOS #2.

Wetland area substrates were characterized by surface soil sampling within identified wetland areas at the Lakeshore Area during the RI. The following wetland soil samples were collected from eight locations, including:

- 4 locations within wetland area WL2 (HB-SS-08, HB-SS-09, HB-SS-10, HB-SS-11)
- 2 locations within wetland area WL3 (HB-RISB-01 and HB-RISB-02)
- 1 location within wetland area WL4 (HB-SS-04)
- 1 location within wetland area WL5 (HB-SS-01).

Samples collected by Honeywell were shipped to Columbia Analytical Services for analyses by USEPA SW846 methods. TCL/TAL analyses were performed using Methods 8260 [plus 10 tentatively identified compounds (TICs)], 8270C (plus 20 TICs), 8081A, 8082, 6010B, 7471, and 9010B/9014 for VOCs, SVOCs, pesticides, PCBs (including Aroclor 1268), metals, mercury, and cyanide, respectively. Five wetland soil sample locations, one from each identified wetland area at the Site, were sampled from 0 to 0.5 ft and 0.5 to 1 ft for polychlorinated dioxins and furans (PCDD/PCDFs) and methyl mercury using USEPA SW846 Method 8290 and modified EPA Method 1630, respectively. The methyl mercury samples were analyzed by Frontier Geosciences.

RI Soil Borings

During the completion of the RI, soil borings were advanced at 21 locations at the Site to characterize subsurface soils and facilitate monitoring well installation. The number of samples collected at each sub-area is listed below:

- 5 soil borings were advanced and 5 subsurface samples collected at the Lakeshore Area
- 1 soil boring was advanced and 1 subsurface samples collected at the Penn-Can Property
- 4 soil borings were advanced and 3 subsurface samples collected at the Railroad Area
- 10 soil borings were advanced and 11 subsurface samples collected at the AOS #1
- 3 soil borings were advanced and 1 subsurface sample collected at the AOS #2.

The soil borings were advanced between December 13, 2002 and March 10, 2002. Two additional soil borings were advanced within AOS #1 on May 24 and 25, 2004. The additional soil borings were advanced using direct push drilling techniques.

RI Surface Water Sampling

Two rounds of surface water samples were collected during the RI field program. The first round of surface water samples was collected from June 2 through June 4, 2003, and the second round of samples was collected on September 9, 2003. The number of locations sampled at each of the sub-areas of the Site during each round is presented below.

Round 1

- 5 samples collected at 5 locations within Harbor Brook
- 3 samples collected at 3 locations at Penn-Can Property
- 2 samples collected at 2 locations at Railroad Area
- 3 samples collected at 3 locations within I-690 drainage ditch on Lakeshore Area

Round 2

- 5 samples collected at 5 locations within Harbor Brook
- 1 sample collected at 1 location at Railroad Area.

During Round 2 surface water sampling, many of the proposed locations were dry and could not be sampled.

RI Sediment Sampling

One round of sediment samples was collected during the RI field program from Harbor Brook and on-site drainage ditches. Sediment samples were collected from June 2 through June 4, 2003. Sediment samples were collected from 0 to 0.5 ft and 0.5 to 1 ft intervals. The number of locations sampled at the various sub-areas of the Site is presented below.

Round 1

- Four samples collected at three locations within Harbor Brook
- Four samples collected at three locations at Penn-Can Property
- Four samples collected at two locations at Railroad Area
- Six samples collected at three locations within I-690 drainage ditch on Lakeshore Area
- Two samples collected at one location within the drainage ditch associated with AOS#2

RI I-690 Catch Basin Sampling

As part of the RI, three catch basins (HB-DR-69, HB-DR-70, and HB-DR-72) associated with the I-690 storm drain system were sampled to evaluate whether the storm drain system is acting as a conduit for migration of Site-related constituents. Storm sewer sediment and water were collected from each of the catch basins on June 5, 2003, and sediment was collected on September 11, 2003. No storm water was collected on September 11, 2003, because the catch basins were dry.

RI Ground Water Sampling

Two rounds of ground water samples were collected from newly installed and existing wells. The first round was collected between May 7, 2003 and May 22, 2003, during a time of high ground water elevations. The second round was collected between August 13, 2003 and August 27, 2003, during a time of low ground water elevation.

RI Seeps Reconnaissance and Sampling

The reconnaissance was performed on four separate occasions, and was focused on the shore of Onondaga Lake and the banks of Harbor Brook. Seep locations were staked and the locations were marked using a hand held GPS unit. Identified seeps were then sampled. At one seep location, it was not possible to sample the seep water, so sediment in the area of the seep was collected and sent to the laboratory for analyses. Seep water samples were collected on two occasions.

1.2.1.5. Supplemental RI

The Harbor Brook Supplemental RI field activities were conducted between October 2006 and June 2007. The Supplemental RI included sampling of soils via test pits, geoprobe borings and soil borings, ground water via hydropunch samples and monitoring wells, and surface water and sediment. The number of samples collected and analyses performed for the Supplemental RI is summarized below.

Supplemental RI Surface Soil Sampling

During the completion of the Supplemental RI, one surface soil sample was collected from a depth of 0 to 2 ft. The surface soil was collected in conjunction with soil boring location HB-SB-65 based on visual characteristics of the material.

Supplemental RI Soil Borings (Wastebed B/Harbor Brook)

During the completion of the Supplemental RI, soil borings were advanced at 27 locations at the Site to characterize subsurface soils. The soil borings were advanced between October 2006 and November 2006. The soil borings were advanced using direct push drilling techniques.

Supplemental RI Test Pits

Test pits were advanced during the Supplemental RI in November 2006. A total of 17 test pits were advanced using an excavator. These test pits were advanced to further evaluate the physical and chemical characteristics of shallow subsurface soils (0 to 10 ft) within DSA#1 and DSA#2 at the Site.

Test pits were excavated with varying lengths and depths depending upon their location. The excavated materials were staged adjacent to the pit pending visual inspection by O'Brien & Gere and the NYSDEC and collection of samples. Three samples were collected for laboratory analysis for TCL/TAL parameters.

Supplemental RI Ground Water Screening Sampling

During the Supplemental RI, ground water screening samples (HB-GWS-01 through HB-GWS-09) were collected at the SYW-12 area to aid in the selection of monitoring well locations. Once first encountered ground water was observed, the boring was stopped at that depth, and a temporary well point was installed. The temporary well points were sampled using a peristaltic pump. The samples were submitted to O'Brien & Gere Laboratories for TCL/TAL analyses by USEPA SW846 methods.

Supplemental RI Ground Water Sampling

One round of ground water samples was collected from newly installed and existing wells. The round was collected in March 2007 during a time of high ground water elevations.

Supplemental RI Sediment Sampling (Harbor Brook Borings)

Three borings (HB-SB-82, HB-SB-83, and HB-SB-90) were advanced within Harbor Brook during the Supplemental RI.

Supplemental RI SYW-12 Surface Soil/Wetland Sediment Sampling

Surface soil samples were collected from 30 locations by boring with a manually driven 2-inch split spoon or hand auger. Sample locations were distributed throughout the SYW-12 area to evaluate conditions across the entire area. Samples were collected from the 0 to 6 inch, 6 to 12 inch, and the 12 to 24 inch depth intervals. Samples were not collected from 12 to 24 inches at locations HB-WSD-19 and HB-WSD-22 due to refusal.

1.2.2. Notes on Specific Analyses

Mercury and Mercury-High Resolution: In Exposure Areas where both mercury and mercury-high resolution were evaluated separately, these data were combined and integrated into a single “mercury” data set by retaining the analyte with the higher detected value.

Polychlorinated Biphenyls: Calculation of polychlorinated biphenyl (PCB) concentrations for use in exposure point concentrations combined individual Aroclors into three groups. Detected “less chlorinated” PCBs (Aroclors 1016, 1221, 1232, and 1242) were summed for screening (in the RAGS Table 2 Series against the screening values for Aroclor 1016) and for determination of the exposure point concentration. Detected “highly chlorinated” PCBs (Aroclors 1248, 1254, 1260, and 1268) were summed for screening (in the RAGS Table 2 Series against the screening values for Aroclor 1254) and for determination of the exposure point concentration. Lastly, “Total PCBs” combined all Aroclors detected and compared them to screening values of Aroclor 1254. The range of detection limits for less chlorinated PCBs is based on Aroclor 1016 and the range of detection limits for highly chlorinated PCBs is based on Aroclor 1254.

Dioxin/Furans: At each sample location, dioxin/furan congeners were related to 2,3,7,8-TCDD equivalents using World Health Organization toxicity equivalency factors (TEF; Van den Berg *et al.*, 2006). Screening and risk evaluations were performed on the derived 2,3,7,8-TCDD toxicity equivalent (TEQ) values. Where congeners were non-detect, one-half of the reporting limit was used for deriving TEQ values. In cases where a large proportion of congeners are non-detect and/or where reporting limits for non-detects are elevated, this approach may lead to overestimation of TEQ values. Uncertainties related to reporting limits for dioxin/furan congeners are discussed in Sections 7.1 and 7.1.1.4.

Chlordanes: The data set contains samples of chlordane, constituents of chlordane (alpha, beta, and gamma), and alpha chlordane. Where both chlordane and constituents of chlordane were measured in the same sample, they were summed to give a total chlordane value. If several chlordane compounds were detected, non-detect compounds were excluded from the sum (treated as zero). If all chlordane compounds were non-detect, one-half of the reporting limits are summed as the total chlordane value. In some samples, constituents of chlordane (alpha, beta, and gamma) and alpha chlordane were both measured. If both measurements were detects, only constituents of chlordane is used as total chlordane. If one compound is non-detect, the detected compound is used as total chlordane. For other related groups of pesticides (*e.g.*, endrin compounds, endosulfan compounds), total group values were not estimated because the individual compounds within the group were not often detected in the same sample.

Xylenes: Some samples include a measurement of total xylenes, while others include separate measurements of o-xylene and m&p-xylene. In cases where only o-xylene and m&p-xylene are available, the sum will provide the total xylene value. When one xylene constituent is non-detect and another is detect, the non-detect is excluded from the sum. If both o-xylene and m&p-xylene are non-

detect, one-half of the reporting limits are summed as the value for total xylene. All total xylene measurements were combined to calculate screening and EPC values.

1.3. Risk Assessment Approach

The approach to the risk assessment is presented as outlined below:

- Section 2 – This section presents the human health conceptual site model through which the most significant potential exposure pathways are identified.
- Section 3 – This section presents database definitions, media specific considerations, the approach used to identify Constituents of Potential Concern (COPCs) in the screening process, and the results of constituent screening.
- Section 4 – This section presents the human receptors selected for evaluation as well as the exposure pathways, grouping of exposure units, and development of exposure point concentrations. This section also contains details relating to exposure assumptions, values, and equations used in risk/hazard estimation.
- Section 5 – Non-cancer and cancer toxicity data, including oral, dermal, and inhalation parameters are presented in this section.
- Section 6 – In Section 6, the characterization of risk and hazards for reasonable maximum exposure and central tendency scenarios is presented.
- Section 7 – Uncertainties in the estimates of risk associated with various elements of the risk assessment process are presented in this section.
- Section 8 – Conclusions regarding potential population exposures are presented in Section 8.
- Section 9 – References are provided in Section 9.

2. Human Health Site Conceptual Model

This section identifies the most significant potential exposure pathways through which individuals may be exposed to the contaminants of concern at Wastebed B/Harbor Brook Site. An exposure pathway analysis describes the transport of a chemical from the source of release to the exposed individual. An exposure pathway links the sources, locations, and types of environmental patterns to determine significant pathways of human exposure. As defined in USEPA's Risk Assessment Guidance for Superfund (RAGS), an exposure pathway has four elements:

- A source and mechanism of chemical release to the environment.
- An environmental transport medium (*e.g.*, ground water) for the released chemical and/or mechanism of transfer of the chemical from one medium to another.
- A point of potential human contact with the contaminated medium (exposure point).
- Exposure route at the contact point (*i.e.*, ingestion, inhalation, or dermal contact).

The identification of potential release mechanisms and receiving media were determined utilizing site histories and data from existing reports. The fate and transport of the chemicals from release media were also considered to identify media that may receive site-related chemicals. Points of potential contact with chemically contaminated media (or sources) by human receptors were then considered and defined based on current and potential future uses of the site. The area demography and land use characteristics were taken into consideration when the pathways were developed. If a pathway potentially could be complete between the source of contamination and a human receptor, it was retained for further quantitative evaluation. This risk assessment identified exposure pathways assuming that no site remediation occurs and that no additional restrictions to site access or use exist. The goal was to establish whether it is feasible for individuals to engage in activities resulting in exposure to site-related contaminants. **Figures 8 and 9** summarize the Site Conceptual Model both Site-wide and for SYW-12, respectively.

This document utilizes the Exposure Unit (EU) concept to refine estimates of quantitative risk. An EU is defined as an area over which receptors are expected to integrate exposure when routinely present at the Site. For example, if a current or future construction worker has been identified as a potential receptor, that worker is assumed to be exposed randomly to Site media in an area equal to the area over which construction is possible. This area may include more than one of the defined sub-areas (exposure areas) of the Site (*e.g.*, Penn-Can Property, Railroad Area, *etc.*). As such, each receptor is associated with an EU that accounts for their potential exposure in all areas where they may be expected to come in contact with environmental media.

The following sections describe the possible sources, receptors, and exposure pathways relevant to the Site considering both current and potential future land use. An identified pathway does not imply that exposures are actually occurring, only that the potential exists for the pathway to be complete.

This section is comprised of the following subsections:

- In Section 2.1, potential human receptors that may be currently active at the Wastebed B/Harbor Brook Site and SYW-12 are identified and described. Receptors associated with potential future land use scenarios are also discussed in this subsection.

- In Section 2.2, potential exposure pathways for each Exposure Unit/receptor combination are identified.

2.1. Exposure Setting and Receptor Populations

The first step in evaluating the potential human exposure at a Site is to characterize the Site with respect to its physical characteristics, current and potential land uses, and human populations on or near the Site. A detailed description of this information is provided in Section 1 of this HHRA and is summarized below as well as in **RAGS Tables 1.1 through 1.9**, provided in Attachment A. This information was used to identify possible exposure pathways for potentially exposed populations and to determine appropriate exposure intake parameters to quantify exposure.

2.1.1. Current Land and Site Use

The Site is currently separated geographically into two main areas that include the properties along southwest corner of Onondaga Lake and the property along the northeast corner of Onondaga Lake adjacent to the mouth of Ley Creek. These areas are depicted in the Site Plan (**Figure 2**), which includes a representation of features (*e.g.*, fenceline, highway, access road, etc.) that would affect potential receptor access to the areas. Specific discussion of potential receptor access is presented in Section 1.1

The southwest corner properties include the following areas:

- Lakeshore Area (including Wastebed B)
- Penn-Can Property
- Railroad Area
- AOS #1
- AOS #2
- East Flume
- Harbor Brook

The northeast corner property includes wetland area SYW-12 that is currently owned by Onondaga County. Due to both areas proximity to the lake, there is an opportunity for individuals at the Site (trespassers and/or recreators) to participate in water-oriented recreational activities, including fishing.

The southwest corner is bisected by I-690 and several railroads. A ditch runs parallel to the westbound lane of I-690 at the southern border of the Lakeshore Area. The NYSDOT occasionally removes accumulated sediments and vegetation from this drainage ditch. Currently the NYSDOT recently replaced the I-690 bridge that is located along the western boundary of the Penn-Can property. Also, the Penn-Can property contains several buildings including an office building and several garages. Currently, the property is being used by Spano Container Corporation for the storage of equipment and their container rental operations. The CSX and New York, Susquehanna, and Western Railways are currently active and trains use the tracks that run through the Site on a daily basis. Also, several sewer lines and other buried utilities run through the Site at depths of less than or equal to 10 ft bgs. These utilities may need to be accessed on occasion for maintenance.

The northeast corner (SYW-12) is bisected by several CSX rail lines. Several sewer lines and other buried utilities run through SYW-12 at depths of less than or equal to 10 ft bgs. This area is currently a vacant lot other than the rail lines and utilities.

Currently ground water at the Site (including SYW-12) is not used for any purpose; however, utility workers may inadvertently come into contact with shallow ground water during the course of their excavations.

2.1.2. Potential Current Receptors

Under current conditions, the most likely potential receptors for the Wastebed B/Harbor Brook Site are as follows:

- *Adult and Older Child Trespasser* – Trespassers can access many of the areas at this Site.
- *Surveillance Worker* – All areas owned by Honeywell are subject to routine surveillance.
- *Utility Worker* – As part of installing or repairing underground utilities that exist in this area, the utility worker is evaluated.
- *Drainage Ditch Worker* – Periodic maintenance of the drainage ditches is needed to ensure functionality. Therefore, the drainage ditch worker is evaluated.
- *Railroad Worker* – Active rail tracks bisect areas of the Site and railroad workers currently access these areas to perform job functions and as such, this pathway is evaluated.
- *Commercial Industrial Worker* – Commercial/industrial facilities currently exist in this area of the Site. Therefore, this receptor is evaluated in a current scenario.

Potential current receptors and their associated Exposure Units are summarized below in **Table 2.1**.

Table 2.1. Current Exposure Scenarios.

Exposure Unit ^{a,b}	Receptors	Rationale
Exposure Unit 1: Site-Wide (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)	Older Child Trespasser, Adult Trespasser, Utility Worker	Currently, these receptors may access all exposure areas of the Site.
Exposure Unit 2: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2	Surveillance Worker ^c	The surveillance worker may access all exposure areas of the Site owned by Honeywell.
Exposure Unit 3: I-690 Storm Sewer and Drainage Ditch	Drainage Ditch Worker	The drainage ditch worker may access the I-690 storm sewer and drainage ditch.
Exposure Unit 4: Railroad Area	Railroad Worker	The railroad worker may access the railroad area, through which railroad tracks pass.
Exposure Unit 5: Penn-Can Property	Commercial/Industrial Worker	Businesses currently occupy only the Penn-Can property.
Exposure Unit 9: State Wetland SYW-12	Adult Recreator, Child Recreator, Utility Worker, Railroad Worker	Currently, these receptors may access all portions of this Exposure Unit.

Table 2.1. Current Exposure Scenarios.

Exposure Unit ^{a,b}	Receptors	Rationale
Notes: a = AOS#1 was formerly called SYW-19 b = For the HHRA, the Lakeshore Area is defined as Wastebed B and wetlands along the shore of Onondaga Lake. c = Surveillance worker constrained to Honeywell-owned exposure areas.		

2.1.3. Future Land and Site Use

Future land use at this Site is likely to include all of the activities outlined above. In addition, several future additional land use activities have the potential to occur at the Site. It is possible that additional industrial or commercial properties will be present on the Site, and the exposure areas located north of I-690 and near Onondaga Lake may be used for recreation.

While not expected or likely, it is possible that residential use of the Site could occur in the future. Given the availability and current use of municipal water, it is unlikely, though possible, that any future residents and commercial/industrial workers could use Site ground water as potable water.

2.1.4. Potential Future Receptors

Under potential future conditions, the most likely receptors for the Wastebed B/Harbor Brook Site are as follows:

- *Adult and Older Child Trespasser* – Trespassers are likely to continue to have access to many of the areas at this Site in the future.
- *Surveillance worker* – All areas owned by Honeywell will be subject to routine surveillance.
- *Utility Worker* – A utility worker is likely to be exposed to Site constituents during future installation or repair of underground utilities in this area.
- *Construction Worker* – Future construction in many areas of the Site is possible, therefore this receptor is selected for evaluation.
- *Adult and Child Recreator* – Adult recreators visiting this area may not be restricted in the future. Children accompanied by adults are also evaluated for potential exposure.
- *Adult and Child Resident* – Although residential use of the Site is not anticipated, it is possible that portions of the Site may be redeveloped for residential housing. As such, this pathway is evaluated as a potential future scenario.
- *Drainage Ditch Worker* – Periodic maintenance of the drainage ditches will be needed in the future to ensure functionality. Therefore, the drainage ditch worker is evaluated.
- *Railroad Worker* – Active rail tracks are likely to continue to operate in some areas of the Site in the future. Therefore, the railroad worker is evaluated.
- *Commercial Industrial Worker* – In the future, additional businesses could be developed on this Site. Therefore, commercial/industrial workers may be exposed to Site-related constituents and are evaluated.

Potential future receptors and their associated Exposure Units are summarized below in **Table 2.2**.

Table 2.2. Future Exposure Scenarios.

Exposure Unit^{a,c}	Receptors	Rationale
Exposure Unit 1: Site-Wide (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area)	Older Child Trespasser, Adult Trespasser, Utility Worker, Construction Worker	In the future, these receptors may access all exposure areas of the Site.
Exposure Unit 2: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2	Surveillance Worker ^b	The surveillance worker may access all exposure areas of the Site owned by Honeywell.
Exposure Unit 3: I-690 Storm Sewer and Drainage Ditch	Drainage Ditch Worker	The drainage ditch worker may access the I-690 storm sewer and drainage ditch.
Exposure Unit 4: Railroad Area	Railroad Worker	The railroad worker may access the railroad area, through which rail tracks pass.
Exposure Unit 5: Penn-Can Property	Commercial/Industrial Worker	In the future, businesses could be present in this exposure unit.
Exposure Unit 6: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1	Adult Recreator, Child Recreator, Adult Resident, Child Resident	These exposure areas located north of I-690 and near Onondaga Lake are suitable for recreation. Although residential development is not expected, exposures to potential residents will be evaluated for these exposure areas
Exposure Unit 7: Penn-Can Property, Lakeshore Area, DSA #1, DSA #2, AOS #1, AOS #2	Commercial/Industrial Worker	In the future, businesses could be present in these exposure areas.
Exposure Unit 8: Site-Wide Ground Water (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area, SYW-12)	Commercial/Industrial Worker, Adult Resident, Child Resident	Although future use of ground water for potable water is not expected, potential exposures to commercial/industrial workers and residents will be evaluated
Exposure Unit 9: State Wetland SYW-12	Adult Recreator, Child Recreator, Railroad Worker, Utility Worker, Construction Worker, Adult Resident, Child Resident, Commercial/Industrial Worker	In the future, these receptors may access all areas of this Exposure Unit.
Notes: a = AOS#1 was formerly called SYW-19 b = Surveillance worker constrained to Honeywell-owned exposure areas. c = For the HHRA, the Lakeshore Area is defined as Wastebed B and wetlands along the shore of Onondaga Lake.		

2.2. Selection of Exposure Pathways

This section identifies potential exposure pathways for receptors and constituents selected for evaluation at the Site under current conditions and the recognized scope of reasonably foreseeable future planned use of the Site. An exposure pathway is the course a constituent takes from a source to an exposed receptor. As noted above, a complete exposure pathway consists of the following four elements:

- A source for the constituent (*i.e.*, affected media)
- A mechanism of release, retention, or transport of a contaminant in a given medium (*e.g.*, air, water, soil)
- A point of human contact with the medium (*i.e.*, exposure point)
- A route of exposure at the point of contact (*e.g.*, incidental ingestion, dermal contact)

If any one of these elements are missing, the pathway is considered incomplete and does not present a means of exposure. The RAGS Table 1 Series shows the conceptual model used to identify exposure pathways evaluated in this HHRA.

2.2.1. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 1

Exposure Unit 1 (EU-1) consists of a Site-wide scenario with exposure related to the following exposure areas: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Storm Sewer and Drainage Ditch, Penn-Can Property, and Railroad Area. The scenarios considered for this EU are the current/future older child trespasser, adult trespasser, and utility worker. These receptors may be exposed to surface soil (ingestion, dermal contact, fugitive dust or volatile emissions), and sediment and surface water where present (*i.e.*, Harbor Brook). The utility worker may also be exposed to subsurface soil to a depth of less than or equal to 10 feet (ingestion, dermal contact, fugitive dust or volatile emissions) and shallow ground water that may be present during excavations necessary for utility work. The older child trespasser and adult trespasser may also be exposed to fish tissue from Onondaga Lake (ingestion). A future construction worker is also considered for this Exposure Unit. This receptor may be exposed to surface and subsurface soil to a depth of less than or equal to 10 feet (ingestion, dermal contact, fugitive dust or volatile emissions), and shallow ground water that may be present during excavations necessary for construction activities. Exposure to sediment and surface water is also considered for this receptor, (up to a depth of less than or equal to 10 feet for sediment) due to the current I-690 bridge replacement work over Harbor Brook. The locations of the Harbor Brook sediment samples are shown on **Figures 6 and 6D**.

It should be noted that EU-1 is referred to as Site-wide exposure; however, EU-1 does not include the SYW-12 exposure area, which is removed from the Wastebed-B area and is evaluated as a stand-alone exposure area. Wetland area SYW-12 is included as Exposure Unit 9.

2.2.2. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 2

Exposure Unit 2 (EU-2) reflects those areas associated with Honeywell-owned property that are subject to the activities of a surveillance worker. Therefore, EU-2 is comprised of the following exposure areas: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2. Security surveillance is conducted on these parcels via visual observation of disturbance from fully enclosed vehicles. The surveillance worker is not expected to contact the media present in this exposure unit except in instances where the worker must exit the vehicle to conduct activities such as locking or unlocking a gate. Therefore, surface soil (0 to 2 ft bgs) is the only relevant media to this receptor and is evaluated

in both a current and future scenario. Surface soil throughout the entire exposure area is used to evaluate this receptor, and data are not restricted to samples located near vehicle paths or roadways. Pathways evaluated for this scenario include incidental ingestion, dermal contact, and inhalation of both particulate dust and volatile emissions from surface soil.

2.2.3. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 3

Exposure Unit 3 (EU-3) is comprised of only one exposure area, the Interstate 690 Storm Sewer and Drainage Ditch. Due to the periodic maintenance necessary to ensure the function of the drainage ditch, the receptor evaluated for EU-3 is the drainage ditch worker in both current and future settings. Surface water present in the ditch as a result of storm water runoff is evaluated for dermal contact with the ditch worker; incidental ingestion of surface water is expected to be *de minimis* and, therefore, not evaluated quantitatively. Due to the ephemeral nature of the surface water in this ditch, instances occur when sediment is exposed. During periods of time when sediment is exposed, inhalation of volatile emissions may be possible and is, therefore, evaluated. Damp or wet sediment is not expected to generate fugitive dust and, therefore, is not a complete pathway. Ingestion and dermal exposure to sediment are also evaluated.

2.2.4. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 4

Exposure Unit 4 (EU-4) is comprised of the Railroad Area. A current/future exposure scenario for the areas of the Site containing railroad tracks is the evaluation of railroad worker. A railroad worker is expected to be exposed to surface soil present in the Railroad Area. Incidental ingestion and dermal contact with soil are evaluated in this scenario as are the inhalation of fugitive dust and vapor emissions originating from surface soil. The railroad worker is not expected to contact any other media during the course of his/her activities.

2.2.5. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 5

Exposure Unit 5 (EU-5) is comprised of the Penn-Can Property. Current/future exposure scenarios for the Penn-Can Property are restricted to the commercial/industrial worker receptor. Current zoning of this property and conditions at the Site (presence of buildings, current businesses) dictate that this pathway be evaluated. The commercial/industrial worker may have incidental ingestion or dermal contact with exposed surface soil in the area. During the course of this receptor's activities, inhalation of particulate dust or volatile emissions from surface soil is also possible. Commercial/industrial workers are not expected to contact surface water or sediment in the area. Inhalation of vapors in the occupation workspace arising as a result of vapor intrusion is also a viable exposure pathway that is considered in this assessment.

2.2.6. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 6

Exposure Unit 6 (EU-6) is comprised of exposure areas north of Interstate 690 and near Onondaga Lake, and include: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1. These areas are considered in the future scenario for the recreational adult and child receptors. While residential use of the Site is not expected due to the highly industrialized nature and zoning of the Site, hypothetical adult and child residents are also considered for EU-6. Both types of receptors and age categories may be exposed to surface soil of these areas and are evaluated for incidental ingestion, dermal contact, and the inhalation of fugitive dust and volatile emissions.

Recreational visitors (adult and child) to EU-6 may also attend areas containing surface water or surface sediment. While incidental ingestion of surface water is considered *de minimis* and, therefore, was not evaluated quantitatively, dermal contact with surface water is considered a complete

exposure pathway. Incidental ingestion and dermal contact with surface sediment is also evaluated for this receptor. Recreation use of areas containing surface water includes the collection of game fish for meals. To evaluate this exposure scenario, a quantitative assessment was conducted by utilizing fish tissue concentrations measured and presented in the Onondaga Lake Human Health Risk Assessment (NYSDEC 2002). Any contact with surface water, surface sediment, or fish tissue by the adult or child resident is expected to be *de minimis* and is therefore not evaluated.

Hypothetical residents occupying dwellings could also inhale vapors that may originate from shallow ground water that has intruded into buildings. Ambient air criteria were obtained from Region 9 PRG (USEPA 2004a) and Region 3 RBC (USEPA 2007a), except for those for PCE, TCE, carbon tetrachloride, vinyl chloride, 1,1,1-TCA, 1,1-DCE and cis-1,2-DCE, which were obtained from NYSDOH (2007). Ground water data was evaluated with respect to USEPA OSWER (2002a) ground water-to-indoor criteria. The table below presents the sources of data that were used to evaluate the indoor air pathway for EU-6.

Table 2.3. Data Sets Used for the Vapor Intrusion Pathway in EU-6.		
Exposure Area	Data Used to Screen for Indoor Air Pathway	RAGS Table
Harbor Brook	None	None
Lakeshore Area	Shallow Ground Water	2.4
East Flume	None	None
DSA #1	None	None
DSA #2	Shallow Ground Water	2.30
AOS #1	Shallow Ground Water	2.34

2.2.7. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 7

Exposure Unit 7 (EU-7) is comprised of the following exposure areas: Penn-Can Property, Lakeshore Area, DSA #1, DSA #2, AOS #1, and AOS #2. EU-7 is evaluated solely for a future scenario when development at the Site may include commercial and industrial enterprises. For the areas listed above, the commercial/industrial worker may have incidental ingestion or dermal contact with exposed surface soil. During the course of this receptor's activities, inhalation of particulate dust or volatile emissions from surface soil is also possible. Commercial/industrial workers are not expected to contact surface water or sediment in EU-7. Inhalation of vapors in the occupation workspace arising as a result of vapor intrusion is also a viable exposure pathway that is considered in this assessment. Ground water data were evaluated with respect to USEPA OSWER (2002a) ground water-to-indoor air criteria, and where available, sub-slab vapor was evaluated with respect to the USEPA Region 2 chemical-specific matrix approach for evaluating vapor intrusion.

Table 2.4. Data Sets Used for the Vapor Intrusion Pathway in EU-7.		
Exposure Area	Data Used to Screen for Indoor Air Pathway	RAGS Table
Penn-Can Property	Sub-slab Vapor, Shallow Ground Water	2.9
Lakeshore Area	Shallow Ground Water	2.4
DSA #1	None	None
DSA #2	Shallow Ground Water	2.30
AOS #1	Shallow Ground Water	2.34
AOS #2	None	None

2.2.8. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 8

Exposure Unit 8 (EU-8) consists of ground water data for all areas of the Site, regardless of the depth interval from which the data was collected. Due to the use designation for the aquifer present at the Site is considered potable and the National Contingency Plan states the ground water must be returned to its most beneficial use, this pathway has been evaluated as a future scenario. The receptors affected by potable water at the Site include future adult and child residents as well as future commercial/industrial workers. For adult and child residents, ingestion of potable water and dermal contact with potable water are evaluated. Inhalation of potable water vapor originating during showering is evaluated for the adult and child residents. Commercial/industrial workers are not expected to have dermal contact with potable ground water; however, ingestion of potable water is considered for this receptor. EU-8 also incorporates SYW-12 as part of site-wide ground water; this is distinct from the treatment of SYW-12 throughout the remainder of this HHRA (see section 2.2.9).

2.2.9. Exposure Pathways, Receptors, and Media Evaluated for Exposure Unit 9

Because SYW-12 is not contiguous with the Wastebed B/Harbor Brook Site, it was incorporated into the HHRA as stand-alone Exposure Unit 9 (EU-9). The scenarios considered for EU-9 are the current/future child recreator, adult recreator, railroad worker, and utility worker. The recreational and railroad worker receptors may be exposed to surface soil via ingestion, dermal contact, fugitive dust or volatile emissions. The recreator scenarios (child and adult) are considered to be protective of the trespasser scenarios (older child and adult). The utility worker may be exposed to surface and subsurface soil to a depth of less than or equal to 10 feet via ingestion, dermal contact, fugitive dust or volatile emissions, and shallow ground water that may be present during excavations necessary for utility work.

The future scenario timeframe considers a construction worker, a commercial/industrial worker, and an adult and child resident. The resident and the commercial/industrial worker receptor may have incidental ingestion or dermal contact with exposed surface soil in the area. Also, during the course of their activities, inhalation of particulate dust or volatile emissions from surface soil is also possible. Inhalation of vapors in the occupational workspace or residence arising as a result of vapor intrusion is also a viable exposure pathway that is considered in this assessment.

A future construction worker is also considered for EU-9. This receptor may be exposed to surface and subsurface soil to a depth of less than or equal to 10 feet via ingestion, dermal contact, fugitive dust or volatile emissions, and shallow ground water that may be present during excavations necessary for construction activities.

3. Screening for Constituents of Potential Concern

To select compounds to evaluate further in the HHRA analysis, a conservative screening process is applied using methods consistent with the National Oil and Hazardous Substances Pollution Contingency Plan (NCP). The following sections present considerations and assumptions made relative to specific compound groups and media types, the approach used to select COPCs, and the results of the screening process.

3.1. Media Specific Considerations

This section describes the media that are relevant to this assessment. **Appendix B** provides a comprehensive list of samples used in this deliverable.

Surface Soil: Surface soil was defined as soil collected from 0 to 2 feet (ft) below ground surface (bgs). The soil database contained a start depth and an end depth for a given sample. Surface soil was sorted from the entire soil database by selecting samples with an end depth that was less than or equal to 2 ft. Thus, a sample collected from 1 ft (start depth) to 3 ft (end depth) would not have been included in the RAGS 2 Tables that evaluate surface soils.

Where SYW-12 surface soil was evaluated quantitatively, the data selected included soil with end depth less than or equal to 2 ft. In **Appendix A** (electronic copy of the Site data set), the sample type code for SYW-12 surface soil is WSD (wetland sediment). However, because SYW-12 wetland sediment is considered hydric soil, it was evaluated as soil. SYW-19 wetland sediment was also evaluated as soil.

Upper Soil (Surface and Subsurface Soil combined): Two exposure scenarios (construction worker and utility worker scenarios) required the evaluation of surface and subsurface soil combined. This exposure medium was defined as soil collected from 0 to 10 ft bgs. Surface and subsurface soil combined was sorted from the entire soil database by selecting samples with an end depth that was less than or equal to 10 ft bgs.

Shallow Ground Water: Two exposure scenarios (construction and utility worker scenario) required the evaluation of direct exposure to shallow ground water. This exposure medium was defined as ground water samples collected from monitoring wells that contained a depth to water from 0 to 10 ft bgs. Shallow ground water was sorted from the Site database by selected data with a start depth less than or equal to 10 ft bgs. The start depth was used rather than the end depth to select for shallow ground water, because of the abundance of ground water samples with start depth less than or equal to 10 ft bgs but an end depth greater than 10 ft bgs.

Shallow ground water data were also used to evaluate the ground water-to-indoor air vapor intrusion pathway when no soil vapor data were available for a particular exposure unit.

Surface Sediment: Surface sediment was evaluated for the following exposure areas: AOS #2, East Flume, Harbor Brook, I-690 Drainage Ditch, Penn-Can Property, and the Railroad Area. Generally, surface sediment was defined as sediment with an end depth that was less than or equal to 1 ft. In some instances, data were collected from an interval that began at 0 ft but extended below 1 ft. For example, sediment samples collected over the 0 to 1.3 ft and 0 to 1.5 ft intervals for the Harbor Brook exposure area were included as surface sediment. Likewise, surface sediment samples collected over

the 0 to 1.5 ft, 0 to 2 ft, 0 to 2.25 ft, and 0 to 3 ft intervals for the East Flume exposure area were included as surface sediment.

Subsurface Sediment: Subsurface sediment was only evaluated for the Harbor Brook exposure area in this assessment, in anticipation of the I-690 bridge replacement work over Harbor Brook. These data were defined as sediment data with a beginning depth greater than 1 ft bgs and an end depth less than or equal to 10 ft bgs.

Surface Water: Surface water present in water bodies (Harbor Brook, East Flume, Railroad Area, Penn-Can Property) as well as small collections of surface water present within exposure areas of the Site were evaluated in this assessment. Storm water collected at the Interstate 690 Drainage Ditch and Lakeshore Area seeps were considered surface water for the purposes of this evaluation.

Site-Wide Ground Water: One exposure scenario (hypothetical drinking water scenario) required the evaluation of all Site ground water collected from monitoring wells, regardless of depth. This scenario was evaluated for the adult and child resident as well as the commercial/industrial worker (adult), with regard to potential use of ground water as potable water. SYW-12 was included in the analysis of site-wide ground water.

Soil Vapor: Where soil vapor was evaluated quantitatively with respect to a potential vapor intrusion exposure pathway, the data selected included soil vapor collected at all depths (all samples have end depth less than or equal to 12 ft).

3.1.1. Indoor Air Pathway

The vapor intrusion pathway was evaluated in the HHRA for a current or future commercial/industrial worker and a future resident (adult and child). The RAGS Table 2 Series screening for the indoor air exposure was conducted in one of two ways. In situations where sub-slab soil vapor data were available (Penn-Can Property), these data were evaluated with respect to the USEPA Region 2 chemical-specific matrix approach for evaluating vapor intrusion. The screening resulted in a decision of either “no action” or a recommendation to sample indoor air.

In areas where shallow ground water data exist (Lakeshore, Penn-Can, DSA #2, AOS #1, and SYW-12), the constituents for the vapor intrusion pathway were screened against USEPA OSWER (2002a) ground water to indoor air criteria. Screening the vapor intrusion pathway using ground water data is based on the simplified assumption that soil gas attenuates by a factor of 1000 when migrating to indoor air and that partitioning across the water table obeys Henry's Law. Based on empirical data (USEPA 2008a), attenuation factors are variable and can span over two orders of magnitude.

3.2. Identification of Constituents of Potential Concern

Unlike RAGS Table Series 1 and 3 that are organized by Exposure Units, the RAGS Table 2 series, which identifies COPCs, is organized by individual exposure areas (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Storm Sewer and Drainage Ditch, Penn-Can Property, and Railroad Area). This was done to increase the resolution for determining specific areas that drive risk at this Site. For example, knowing that the maximum concentration of a constituent is located in the Lakeshore area is more useful for risk management decisions than knowing that the maximum value is somewhere in Exposure Unit 1. This approach also facilitates the examination of potential hot spots. Hot spots are discussed in greater detail in Sections 7.5 and 7.6.

Consistent with USEPA guidance (USEPA, 1989), a conservative screening process was applied to the selection of constituents of potential concern (COPC). To develop the COPC list, the maximum detected concentrations of the detected constituents in surface soil, combined surface and subsurface soil, surface water, surface sediment, shallow ground water, and all ground water were compared to conservative screening values for the protection of human health.

The screening values utilized were the lowest of the USEPA Region 9 Preliminary Remediation Goals (PRGs) (USEPA 2004a) or the USEPA Region 3 Risk-Based Concentrations (RBCs) (USEPA 2007a). RBCs and PRGs for tap water were applied to screen surface water and ground water detected concentrations. RBCs and PRGs for residential soils were applied to screen the soil and sediment detected concentrations. RBCs and PRGs utilized in the screening process corresponded to a cancer risk of 10^{-6} or a hazard quotient of 0.1.

Other Applicable or Relevant and Appropriate Requirements (ARAR) were included in the RAGS Table 2 Series for surface and subsurface soils (New York Subpart 375-6 Soil Cleanup Objectives) and for surface water and ground water [USEPA (2008b) National Primary and Secondary Drinking Water Regulations]. These ARARs were included for informational purposes and were not used to screen constituents in or out of the HHRA.

If the maximum detected concentration was less than the identified screening values, it was concluded that exposure to the constituent does not represent an unacceptable risk to human health, and no further evaluation of this constituent was necessary. If the maximum detected concentration exceeded the selected screening value, the constituent was selected as a COPC and retained for further evaluation in this assessment.

Naturally occurring compounds were eliminated from the COPC list if they were essential nutrients. Based on this consideration, calcium, magnesium, potassium, and sodium were not carried forward as COPCs for the risk assessment. Wet chemistry analytes and geochemical parameters were not included in the risk assessment (*e.g.*, chloride, nitrogen, and total organic carbon).

Constituents detected in media that do not have established RBC or PRGs were carried forward for further evaluation in the risk assessment. Compounds that were not detected at any of the locations sampled were not included in the quantitative evaluation.

All detected Group A carcinogens (arsenic, benzene, chromium, and vinyl chloride) were retained as COPCs even if their maximum detected concentration did not exceed their respective screening criteria. The unspiciated chromium was evaluated as hexavalent chromium.

The constituent 3&4-methylphenol was screened against the 4-methylphenol RBC criteria.

Chlordane constituents were summed as described in section 1.2.2 and screened against the chlordane RBC and technical chlordane PRG criterion.

3.3. Screening Results

Results of the constituent screening are presented below.

3.3.1. Site-Wide – All Ground Water (Potable Water Scenario)

Analytical results of detected concentrations of Site-wide ground water samples are presented in RAGS Table 2.1. Approximately 160 samples were analyzed for 115 chemical constituents, of which 87 chemical constituents were screened in as COPCs, with 72 constituents above screening levels and 11 constituents screened in because there was no toxicity information (endosulfan II, endosulfan sulfate, 2-nitrophenol, 4-chloro-3-methylphenol, 4-nitrophenol, acenaphthylene, benzo(g,h,i)perylene, phenanthrene, 1,2,3-trichlorobenzene, 2-hexanone, and p-isopropyltoluene). In addition, arsenic, chromium, benzene, and vinyl chloride were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients. It should be noted that “Site-Wide” refers to all exposure areas except SYW-12, which was evaluated separately.

3.3.2. Lakeshore Area

Surface Soil: Analytical results of detected concentrations of surface soil samples from the Lakeshore Area are presented in RAGS Table 2.2. Approximately 58 samples were analyzed for 90 chemical constituents, of which 36 COPCs were screened in, with 29 chemical constituents above screening levels, and four constituents screened in because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, phenanthrene, and p-isopropyltoluene). In addition, arsenic, chromium, and benzene were retained because they are classified as Class A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Soil: Analytical results of detected concentrations of subsurface soil samples from the Lakeshore Area are presented in RAGS Table 2.3. Approximately 77 samples were analyzed for 105 chemical constituents, of which 50 COPCs were retained, with 42 chemical constituents above screening levels and five constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, phenanthrene, 1,2,3-trichlorobenzene, and p-isopropyltoluene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water: Analytical results of detected concentrations of shallow ground water samples from the Lakeshore Area are presented in RAGS Table 2.5. Approximately 27 samples were analyzed for 84 chemical constituents, of which 60 COPCs were retained, with 50 chemical constituents above screening levels and six constituents being retained because there was no toxicity information (2-nitrophenol, 4-nitrophenol, acenaphthylene, phenanthrene, 2-hexanone, and p-isopropyltoluene). In addition, arsenic, chromium, benzene, and vinyl chloride were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Water: Analytical results of detected concentrations of surface water samples from the Lakeshore Area are presented in RAGS Table 2.6. Three samples were analyzed for 46 chemical constituents, of which 22 COPCs were retained, with 19 chemical constituents above screening levels, and two constituents were retained because there was no available toxicity information: acenaphthylene and phenanthrene. In addition, benzene was retained because it is a Group A carcinogen, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

3.3.3. Penn-Can Property

Surface Soil: Analytical results of detected concentrations of surface soil samples from the Penn-Can Area are presented in Table RAGS 2.7. Approximately 23 samples were analyzed for 69 chemical constituents, of which 26 COPCs were retained for evaluation, with 19 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, phenanthrene, and p-isopropyltoluene). In addition, arsenic, chromium, and benzene were retained because they are classified because they are Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Soil: Analytical results of detected concentrations of subsurface soil samples from the Penn-Can Area are presented in RAGS Table 2.8. Approximately 29 samples were analyzed for 69 chemical constituents, of which 34 COPCs were screened in, with 27 chemical constituents above screening levels and four constituents screened in because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, phenanthrene, and p-isopropyltoluene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subslab Vapor: Analytical results of detected concentrations of subslab vapor samples from the Penn-Can Area are presented in RAGS Table 2.9a. Four samples were analyzed for 26 chemical constituents, and all except one of them were VOCs. Three compounds (naphthalene, chloroform, and trichloroethene) were above screening levels, and one other (benzene) was retained because it is a Group A carcinogen. Another four compounds lacked available subslab vapor screening toxicity values and were thus flagged for investigation and/or remediation. The remaining 18 compounds were below the screening level, and no further action is required.

Shallow Ground Water: Analytical results of detected concentrations of shallow ground water samples from the Penn-Can Area are presented in RAGS Table 2.10. Approximately nine samples were analyzed for 63 chemical constituents, of which 32 COPCs were retained, with 26 chemical constituents above screening levels and three constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, and phenanthrene). In addition, arsenic, chromium, and benzene were retained because they are classified because they are Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Sediment: Analytical results of detected concentrations of surface sediment samples from the Penn-Can Area are presented in RAGS Table 2.11. Approximately seven samples were analyzed for 49 chemical constituents, of which 19 COPCs were screened in, with 13 chemical constituents above screening levels and four constituents screened in because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, phenanthrene, and p-isopropyltoluene). In addition, arsenic and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Water: Analytical results of detected concentrations of surface water samples from the Penn-Can Area are presented in RAGS Table 2.12. Approximately six samples were analyzed for 39 chemical constituents, of which 12 COPCs were retained, with seven chemical constituents above screening levels and two constituents retained because there was no toxicity information (acenaphthylene and phenanthrene). In addition, arsenic, chromium, and benzene were retained

because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

3.3.4. Railroad Area

Surface Soil: Analytical results of detected concentrations of surface soil samples from the Railroad Area are presented in RAGS Table 2.13. Approximately 19 samples were analyzed for 61 chemical constituents, of which 23 COPCs were retained, with 16 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, phenanthrene, and p-isopropyltoluene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Soil: Analytical results of detected concentrations of subsurface soil samples from the Railroad Area are presented in RAGS Table 2.14. Approximately 26 samples were analyzed for 71 chemical constituents, of which 23 COPCs were retained, with 16 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, phenanthrene, and p-isopropyltoluene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water: Analytical results of detected concentrations of shallow ground water samples from the Railroad Area are presented in RAGS Table 2.15. Approximately 12 samples were analyzed for 36 chemical constituents, of which 14 COPCs were retained, with nine chemical constituents above screening levels and two constituents retained because there was no toxicity information (phenanthrene and 1,2,3-trichlorobenzene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Sediment: Analytical results of detected concentrations of surface sediment samples from the Railroad Area are presented in RAGS Table 2.16. Approximately six samples were analyzed for 56 chemical constituents, of which 18 COPCs were retained, with 13 chemical constituents above screening levels and three constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, and phenanthrene). In addition, arsenic and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Water: Analytical results of detected concentrations of surface water samples from the Railroad Area are presented in RAGS Table 2.17. Approximately four samples were analyzed for 34 chemical constituents, of which 15 COPCs were retained, with ten chemical constituents above screening levels and two constituents retained because there was no toxicity information (benzo(g,h,i)perylene and phenanthrene). In addition, arsenic and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

3.3.5. Harbor Brook

Surface Sediment: Analytical results of detected concentrations of surface sediment samples from Harbor Brook are presented in RAGS Table 2.18. Approximately 30 samples were analyzed for 82 chemical constituents, of which 33 COPCs were retained, with 28 chemical constituents above

screening levels and three constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, and phenanthrene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Sediment: Analytical results of detected concentrations of subsurface sediment samples from Harbor Brook are presented in RAGS Table 2.19. Approximately 70 samples were analyzed for 86 chemical constituents, of which 46 COPCs were retained, with 37 chemical constituents above screening levels and five constituents retained because there was no toxicity information (2-hexanone, acenaphthylene, benzo(g,h,i)perylene, delta-BHC, and phenanthrene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Water: Analytical results of detected concentrations of surface water samples from Harbor Brook are presented in RAGS Table 2.20. Approximately 14 samples were analyzed for 50 chemical constituents, of which 14 COPCs were retained, with nine chemical constituents above screening levels and two constituents retained because there was no toxicity information (acenaphthylene and phenanthrene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

3.3.6. East Flume

Surface Sediment: Analytical results of detected concentrations of surface sediment samples from East Flume Area are presented in RAGS Table 2.21. Fifteen samples on average were analyzed for 86 chemical constituents, of which 36 COPCs were retained, with 24 chemical constituents above screening levels and nine constituents retained because there was no toxicity information (1,2,3-trichlorobenzene, 1,3,5-trichlorobenzene, 1-methylnaphthalene, acenaphthylene, benzo(g,h,i)perylene, endrin ketone, n-hexadecane, p-isopropyltoluene, and phenanthrene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, and sodium were eliminated because they are essential nutrients.

Surface Water (Outfall): Analytical results of detected concentrations of surface water outfall samples from East Flume Area are presented in RAGS Table 2.22. Two samples were analyzed for six chemical constituents, of which one COPC was retained because it was detected above its screening level. No Group A carcinogens or essential nutrients were detected in any environmental media.

3.3.7. I-690 Ditch

Surface Sediment: Analytical results of detected concentrations of surface sediment samples from I-690 Ditch Area are presented in RAGS Table 2.23. Approximately 14 samples were analyzed for 69 chemical constituents, of which 25 COPCs were retained, with 15 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, p-isopropyltoluene, and phenanthrene). In addition, arsenic, chromium, and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Water: Analytical results of detected concentrations of surface water samples from I-690 Ditch Area are presented in RAGS Table 2.24. Approximately seven samples were analyzed for 43 chemical constituents, of which 20 COPCs were retained, with 15 chemical constituents above screening levels and three constituents retained because there was no toxicity information (acenaphthylene, 2-methylnaphthalene and phenanthrene). In addition, chromium and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

3.3.8. DSA #1

Surface Soil: Analytical results of detected concentrations of surface soil samples from DSA #1 are presented in RAGS Table 2.25. Two samples on average were analyzed for 44 chemical constituents, of which 19 COPCs were retained, with 14 chemical constituents above screening levels and three constituents retained because there was no toxicity information (benzo(ghi)perylene, 1,2,3-trichlorobenzene, and phenanthrene). In addition, arsenic and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Soil: Analytical results of detected concentrations of subsurface soil samples from DSA #1 are presented in RAGS Table 2.26. Approximately seven samples were analyzed for 57 chemical constituents, of which 28 COPCs were retained, with 22 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, p-isopropyltoluene, 1,2,3-trichlorobenzene, and phenanthrene). In addition, chromium and benzene were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

3.3.9. DSA #2

Surface Soil: Analytical results of detected concentrations of surface soil samples from DSA #2 are presented in RAGS Table 2.27. Approximately four samples were analyzed for 60 chemical constituents, of which 22 COPCs were retained, with 15 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, benzo(ghi)perylene, 1,2,3-trichlorobenzene, and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Soil: Analytical results of detected concentrations of subsurface soil samples from DSA #2 are presented in RAGS Table 2.28. Approximately 10 samples were analyzed for 79 chemical constituents, of which 40 COPCs were retained, with 32 chemical constituents above screening levels and five constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, p-isopropyltoluene, 1,2,3-trichlorobenzene, and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water: Analytical results of detected concentrations of shallow ground water samples from DSA #2 are presented in RAGS Table 2.29. Approximately four samples were analyzed for 55 chemical constituents, of which 26 COPCs were retained, with 19 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, phenanthrene, 1,2,3-trichlorobenzene, p-isopropyltoluene). In addition, arsenic,

benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water - Vapor Intrusion Evaluation: Analytical results from shallow ground water samples in DSA #2 were evaluated to assess the potential for a complete vapor intrusion pathway. The results of this screening are presented in RAGS Table 2.30. On average, three samples were evaluated, and 21 of 36 detected chemical constituents were retained for further evaluation. Nine of these chemicals were retained as COPCs because their maximum detected concentration exceeded their relative soil vapor screening value, and benzene was retained as a COPC because it is classified as a Group A carcinogen. The remaining 11 chemicals were retained as COPCs for further evaluation because they lacked screening values. The remaining 16 chemical constituents had maximum chemical concentrations below their relative screening values and thus were not retained as COPCs.

3.3.10. AOS #1

Surface Soil: Analytical results of detected concentrations of surface soil samples from AOS #1 are presented in RAGS Table 2.31. Approximately 20 samples were analyzed for 69 chemical constituents, of which 29 COPCs were retained, with 22 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, benzo(ghi)perylene, dodecane, and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Soil: Analytical results of detected concentrations of subsurface soil samples from AOS #1 are presented in RAGS Table 2.32. Approximately 24 samples were analyzed for 79 chemical constituents, of which 32 COPCs were retained, with 25 chemical constituents above screening levels and four constituents retained because there was no toxicity information (acenaphthylene, benzo(ghi)perylene, dodecane, and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water: Analytical results of detected concentrations of shallow ground water samples from AOS #1 are presented in RAGS Table 2.33. Approximately nine samples were analyzed for 45 chemical constituents, of which 14 COPCs were retained, with 11 chemical constituents above screening levels and one constituent retained because there was no toxicity information (phenanthrene). In addition, benzene and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water - Vapor Intrusion Evaluation: Analytical results from shallow ground water samples in AOS #1 were evaluated to assess the potential for a complete vapor intrusion pathway. These samples (eight on average) for AOS #1 are presented in RAGS Table 2.34. Fifteen of 28 detected chemical constituents were retained as COPCs for further analysis. Benzene was retained because it is a Group A carcinogen, naphthalene had a concentration greater than its screening value, while the other 13 constituents were retained due to lack of screening values. The remaining 13 detected chemical constituents had maximum chemical concentrations below their relative water vapor screening value and were therefore eliminated from further analysis.

3.3.11. AOS #2

Surface Soil: Analytical results of detected concentrations of surface soil samples from AOS #2 are presented in RAGS Table 2.35. Two samples were analyzed for 31 chemical constituents, of which 13 COPCs were retained, with nine chemical constituents above screening levels and two constituents retained because there was no toxicity information (benzo(g,h,i)perylene and phenanthrene). In addition, arsenic and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Surface Sediment: Analytical results of detected concentrations of surface sediment samples from AOS #2 are presented in RAGS Table 2.36. Two samples on average were analyzed for 36 chemical constituents, of which 13 COPCs were retained, with eight chemical constituents above screening levels and two constituents retained because there was no toxicity information (benzo(g,h,i)perylene and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

3.3.12. SYW-12

Surface Soil (Wetland Sediment): Analytical results of detected concentrations of surface soil (wetland sediment) samples from wetland area SYW-12 are presented in RAGS Table 2.37. Approximately 88 samples were analyzed for 80 chemical constituents, of which 24 COPCs were retained, with 18 chemical constituents above screening levels and three constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Subsurface Soil: Analytical results of detected concentrations of subsurface soil samples from wetland area SYW-12 are presented in RAGS Table 2.38. Approximately 103 samples were analyzed for 89 chemical constituents, of which 29 COPCs were retained, with 23 chemical constituents above screening levels and three constituents retained because there was no toxicity information (acenaphthylene, benzo(g,h,i)perylene, and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water: Analytical results of detected concentrations of 18 shallow ground water samples from wetland area SYW-12 Area are presented in RAGS Table 2.39. Out of 68 chemical constituents, 37 COPCs were retained with 30 chemical constituents above screening levels and four constituents retained because there was no toxicity information (4-nitrophenol, acenaphthylene, benzo(g,h,i)perylene, and phenanthrene). In addition, arsenic, benzene, and chromium were retained because they are classified as Group A carcinogens, and calcium, magnesium, potassium, and sodium were eliminated because they are essential nutrients.

Shallow Ground Water - Vapor Intrusion Evaluation: Analytical results from shallow ground water samples in wetland area SYW-12 were evaluated to assess the potential for a complete vapor intrusion pathway. These samples (18 in all) are presented in RAGS Table 2.40. Twenty-four of 43 detected chemical constituents were retained for further analysis as COPCs. Naphthalene was retained as a COPC because its maximum detected concentration exceeded its relative soil screening toxicity value. Benzene was retained due to its classification as a Grade A Carcinogen. Twenty-two chemical constituents were retained because there is no vapor intrusion screening value available for

comparison. The remaining 19 detected chemical constituents had maximum chemical concentrations below their relative screening value and thus were not included in further Site analysis.



4. Exposure Assessment

The goal of the exposure assessment is to estimate intake levels of each of the COPCs for each potential receptor in a given exposure unit. This calculation requires estimates of:

- The concentration of the COPCs encountered by the receptors (the exposure-point concentration).
- The manner and frequency of exposure.
- Receptor characteristics (body weight, ingestion rate, etc.).

These factors were combined to estimate the average daily dose potentially received by receptors.

US EPA defines two types of exposure estimates for Superfund risk assessments: reasonable maximum exposure (RME) and central tendency exposure (CTE). The RME is defined as the highest exposure that reasonably could be expected to occur for a given exposure pathway at a site and is intended to account for both uncertainty in the chemical concentration and variability in the exposure parameters (such as exposure frequency or averaging time) (USEPA 1989b). The CTE is based on mean exposure parameters.

This section is comprised of the following subsections:

- In Section 4.1, the concentrations of the constituents in the various affected media that Site-related receptors may be exposed to are quantified. This subsection discusses the calculation of 95% UCL, the shower model, fish tissue, calculation of particulate emission factors, and volatilization factors, among other parameters
- In Section 4.2, the equations for the calculation of chronic daily intake are presented.
- Section 4.3 presents parameters for the quantitation of exposure to the various affected media, including among others, fish ingestion rates, exposure frequencies and duration, a incidental ingestion of soil and sediment.

4.1. Development of Constituent Exposure Point Concentrations

Exposure point concentrations (EPCs) were calculated for all constituents that were retained in the RAGS Table 2 screening process. An exposure point concentration was calculated for any constituent that was screened in for any one of the exposure areas comprising an exposure unit. For example, Exposure Unit 2 is comprised of five areas: 1) Harbor Brook, 2) Lakeshore Area, 3) East Flume, 4) DSA #1, and 5) DSA #2. If a hypothetical compound was not retained in four of the five exposure areas but was retained for the fifth, an exposure point concentration would still be calculated for that compound in Exposure Unit 2 using the data from all five of the component exposure areas.

4.1.1. General Approach for the Development of EPC Values

Statistical and procedural methods were applied to the data in order to develop an estimate of the EPC for COPCs selected for each Exposure Unit, on a medium-specific basis. The general approach was as follows: where a given data set contained less than three sample points or only one unique detected sample, the maximum value for each analyte in that data set was used as the EPC; for sets with four or more data points, and at least two unique detected samples, statistical methods were applied. In the

latter case, the ProUCL statistical software package (Version 4.0; USEPA 2007b) was used to examine the data distribution and develop an upper confidence level (UCL) on the arithmetic mean. ProUCL was run using Regression on Order Statistics (ROS), which is a method for accounting for non-detect samples in the data set. ROS infers values for non-detect samples based on the distribution of detected data, thus eliminating the influence of high detection limits. ProUCL recommends the most appropriate UCL to use given the distribution type. The UCL recommended by ProUCL was subsequently applied as the EPC. All ProUCL output files are contained in **Appendix C (Electronic)**.

It should be noted that in some cases the 95% UCL is less than the reported average concentration. This is because the arithmetic average reported in the RAGS Table 3 Series is the mean detected concentration. In instances where the detection frequency is low and non-detect samples largely outnumber detected samples, the 95% UCL recommended by ProUCL Version 4 can be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

4.1.2. Calculation of EPCs for Soil, Sediment, Surface Water, and Ground Water

For these media, the approach outlined in Section 4.3.1 was utilized.

4.1.3. Calculation of EPCs for Shower Scenario

The inhalation of volatiles while showering or bathing was quantitatively evaluated for the child and adult resident in the Site-Wide Ground Water exposure scenario for Exposure Unit (EU-8). The Andelman model, as modified by Schaum *et al.* (1994) was used to derive the exposure point concentrations for this pathway (**Appendix D**).

The maximum air concentration in the bathroom ($C_{a \max}$) was derived by applying the following equation from Schaum *et al.* (1994):

$$C_{a \max} = \frac{C_w f F_w t_1}{V_a}$$

The concentration of contaminant in the air (C_a) was derived by applying the following equation from Schaum *et al.* (1994):

$$C_a = \frac{(C_{a \max} / 2) t_1 + C_{a \max} t_2}{t_1 + t_2}$$

Where (all scenarios): Fraction volatilized (f) = 1, C_w = constituent/exposure unit-specific ground water concentration, water flow rate (F_w) = 750 L/day, bathroom volume (V_a) = 12 m³

Where (adult scenarios): time of shower (t_1) = 0.25 hr (RME), 0.1 hr (CT); time after shower (t_2) = 0.33 hr (RME), 0.15 hr (CT)

Where (child scenarios): time of shower (t_1) = 0.45 hr (RME), 0.14 hr (CT); time after shower (t_2) = 0.55 hr (RME), 0.19 hr (CT)

4.1.4. Calculation of EPCs for Fish Tissue

Recreational use of areas containing surface water includes the collection of game fish for consumption. To evaluate this exposure scenario, a quantitative assessment was conducted by utilizing fish tissue exposure point concentrations derived in the *Onondaga Lake Human Health Risk Assessment* (NYSDEC 2002).

4.1.5. Calculation of EPCs for Ambient Air Exposure

The inhalation of air particulates and volatile compounds generated from Site soils (and I-690 drainage ditch sediment for volatile emissions) was evaluated in the Wastebed B/Harbor Brook HHRA. The calculation of the Particulate Emission Factor (PEF) and the Volatilization Factor (VF) are discussed in this section.

Soil constituents that were eliminated in the RAGS Table 2 screening process were not considered to be constituents of concern for these air pathways, because the PRG screening criteria utilized are protective of multi-pathway exposure to soil. Of those soil constituents that were retained, volatile organic compounds were evaluated using the soil-to-air volatilization factor (**Appendix E**). Other types of constituents (metals, PCBs, pesticides, SVOCs, dioxins, and others) were evaluated as particulate emissions (**Appendix F**). These two pathways are discussed below.

Inhalation of Fugitive Dust

The particle emissions factor (PEF) is required to calculate the constituent concentration in fugitive dust. A separate PEF was calculated for each exposure unit based on the size and percent vegetative cover for each exposure area comprising the exposure unit.

The following equation was used to derive concentrations of inorganics, semivolatiles, PCBs, pesticides, dioxins, and compounds not fitting in these categories (designated as “other”) in outdoor air for inhalation exposure pathways (refer to **Appendix E**, Table 1 for the proposed dust constituent list):

$$C_{\text{air}} = \left(\frac{C_{\text{soil}}}{\text{PEF}} \right)$$

where: C_{air} : Concentration of inorganic particulates in air (mg/m^3), C_{soil} : Concentration in soil (UCL, mg/kg), and PEF: Particle emission factor (m^3/kg)

The particle PEF converts concentrations of constituents in soil to concentrations in dust particles in the air as a result of fugitive dust emissions from bare surface soils. USEPA provides the methodology required to calculate the PEF in Appendix D of *Soil Screening Guidance: Technical Background Document* (USEPA 2002b). Separate PEFs for each exposure unit were calculated in this assessment, given that acreages and estimates of vegetative cover differ between them. Equation 5-5 in USEPA (2002b) was used to derive a PEF for the construction worker and utility worker scenario and Equation 4-5 in of *Soil Screening Guidance: Technical Background Document* (USEPA 2002b) was used to calculate the PEF for the remainder of the fugitive dust scenarios. The details of these calculations can be found in **Appendix F**.

Inhalation of Volatile Compounds

The following equation was utilized to derive concentrations of volatile compounds in outdoor air for inhalation exposure pathways:

$$C_{\text{air}} = \left(\frac{C_{\text{soil}}}{\text{VF}} \right)$$

where: C_{air} : Concentration of volatiles in air (mg/m^3), C_{soil} : Concentration in soil (UCL, mg/kg), and VF: Soil-to-air volatilization Factor (m^3/kg)

The volatilization factor is used for defining the relationship between the concentration of volatile organic constituents in soil and the volatilized constituents in outdoor air. A VF is specific to each volatile compound and each exposure area. VFs for this assessment were calculated using Equation 4-8 from of *Soil Screening Guidance: Technical Background Document* (USEPA 2002b) and can be found in **Appendix E**.

4.1.6. Calculation of EPCs for PCDD/PCDFs and Use of TEFs

An objective of the exposure assessment is to estimate exposure concentrations or doses that can be related to the toxicity of the compounds. Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/PCDFs) present a special challenge when it comes to achieving this objective. PCDD/PCDFs are a group of 210 structurally related chlorinated chemicals that are ubiquitous in the environment. Although PCDD/PCDFs are typically present in the environment as a mixture of many individual compounds, the toxicity of the vast majority of these compounds has not been evaluated. The Toxic Equivalency Factors (TEFs) approach is used to facilitate the evaluation of mixtures of PCDD/PCDFs. The TEF approach has been widely accepted and used by the scientific and regulatory communities in many parts of the world (USEPA 1989a; USEPA 1989b; WHO 1998).

The TEF approach is based on the fact that 2,3,7,8-TCDD is the most widely studied chlorinated dioxin. Available data indicates that some of the congeners, which are substituted with chlorine at the 2,3,7, and 8 positions, may display toxic properties, even though they are less potent than 2,3,7,8-TCDD. Basically, the TEF approach assigns a relative potency factor (relative to 2,3,7,8-TCDD) for certain PCDD/PCDF congeners. The TEFs are based on observed structure activity relationships for PCDD/PCDF compounds, receptor binding affinity, or enzyme induction (Van den berg *et al.* 2006).

Dioxin congeners from a given sample location are each multiplied by their respective TEF to derive the 2,3,7,8-TCDD toxic equivalent concentration (TEQ) for that congener (**Table 4.1** below). TEQs were derived for all congeners at each sample location and summed to derive one TEQ for each location. For non-detect congeners, half the detection limit was used to estimate the TEQ. Statistical analyses (95% UCL, distribution tests) were completed for the collection of location-specific TEQs for each exposure unit.

Table 4.1. Sample Derivation of Toxic Equivalent Concentrations for PCDD/PCDFs.

Congener	Detect Y/N	Reported Value	Concentration for TEF Derivation	Dioxin TEF	Calculated Dioxin Equivalency Concentration (ng/kg)
1,2,3,4,6,7,8-HPCDD	Y	4.36	4.36	0.01	0.044
1,2,3,4,6,7,8-HPCDF	Y	0.537	0.537	0.01	0.005
1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	0.01	0.013
1,2,3,4,7,8-HXCDD	N	2.5	1.25	0.1	0.125
1,2,3,4,7,8-HXCDF	N	2.5	1.25	0.1	0.125
1,2,3,6,7,8-HXCDD	N	2.5	1.25	0.1	0.125
1,2,3,6,7,8-HXCDF	N	2.5	1.25	0.1	0.125
1,2,3,7,8,9-HXCDD	N	2.5	1.25	0.1	0.125

Table 4.1. Sample Derivation of Toxic Equivalent Concentrations for PCDD/PCDFs.

Congener	Detect Y/N	Reported Value	Concentration for TEF Derivation	Dioxin TEF	Calculated Dioxin Equivalency Concentration (ng/kg)
1,2,3,7,8,9-HxCDF	N	2.5	1.25	0.1	0.125
1,2,3,7,8-PECDD	N	2.5	1.25	1	1.250
1,2,3,7,8-PECDF	N	2.5	1.25	0.03	0.038
2,3,7,8-TCDD	N	1	0.5	1	0.500
2,3,7,8-TCDF	Y	1	1	0.1	0.100
OCDD	Y	21.764	21.764	0.0003	0.007
OCDF	N	5	2.5	0.0003	0.001
Sample Location TEQ =					2.7

4.1.7. Calculation of EPCs for Polychlorinated Biphenyls

Calculation of polychlorinated biphenyl (PCB) concentrations for use in exposure point concentrations combined individual Aroclors into two groups. The concentrations of “Less chlorinated” PCBs (Aroclors 1016, 1221, 1232, and 1242) were combined for each sample, screened in RAGS Table 2 against the screening values for Aroclor 1016, and used to calculate the 95% UCL for the exposure point concentration. “Highly chlorinated” PCBs (Aroclors 1248, 1254, 1260, and 1268) were combined for each sample, then screened in RAGS Table 2 against the screening values for Aroclor 1254 and used to calculate the 95% UCL.

4.2. Quantitation of Exposure

The next step in the exposure assessment was to generate estimates of chronic daily intake (CDI) based on the magnitude, frequency, and duration of exposure for each identified complete exposure pathway. In accordance with *Risk Assessment Guidance for Superfund Vol. 1: Human Health Evaluation Manual* (USEPA 1989a), exposure factors were applied to estimate the CDI from incidental ingestion, dermal contact, and inhalation with Site media for the receptor populations.

Chronic daily intake values were calculated for an RME and CT scenario. The RME scenario provides a conservative estimate of potential health risk related to exposure to constituents in Site media. The RME relies on estimated upper bound values for specific exposure parameters as a conservative and health protective measure. A more representative estimate of risk may be developed based on the average exposure values for a specific parameter. Estimates of health risks and hazards based on the less conservative exposure approximations are presented in the CT scenario.

4.2.1. Intake equations and parameter estimates

The intake equations for application in the assessment are presented below. The specific variables used in each calculation and their values are defined in Section 4.5 and **Appendix G**.

Incidental ingestion of COPC in surface water

$$CDI_{sw} = \frac{C_{sw} \times IR \times EF \times ED}{BW \times AT}$$

Dermal uptake of COPC in surface water

$$DAD_{sw} = \frac{C_{sw,pw} \times SA \times PC \times ET \times EF \times ED \times 10^{-3} \text{ L/cm}^3}{BW \times AT}$$

Incidental ingestion of COPC from soil and sediment

$$CDI_{soil, sediment} = \frac{C_{soil} \times IR \times FI \times EF \times ED \times (1 \times 10^{-6} \text{ kg/mg})}{BW \times AT}$$

Dermal uptake of COPC from soil and sediment

$$DAD_{soil, sediment} = \frac{C_{soil} \times SA \times ABS \times AF \times EF \times ED \times (1 \times 10^{-6} \text{ kg/mg})}{BW \times AT}$$

Inhalation of airborne constituents in fugitive dust

$$CDI_{air} = \frac{C_{air} \times InR \times ET \times EF \times ED}{BW \times AT}$$

where:

ABS:	Dermal absorption factor	(unitless)
AF:	Soil to skin adherence factor	(mg/cm ²)
AT:	Averaging time	(days)
BW:	Body weight	(kg)
C _{air} :	COPC concentration in air	(mg/m ³)
C _{soil} :	COPC concentration in soil	(mg/kg)
C _{sed} :	Concentration of each constituent in sediment	(mg/kg)
C _{sw} :	Concentration of each constituent in surface water	(mg/L)
CDI:	Chronic daily intake	(mg/kg-day)
DAD:	Dermally absorbed dose	(mg/kg-day)
ED:	Exposure duration	(years)
EF:	Exposure frequency	(days/year)
ET:	Exposure time	(hours/day)
FI:	Fraction ingested from contaminated source	(unitless)
IR:	Ingestion rate for soil (mg/day) or water	(L/day)
InR:	Inhalation rate	(m ³ /hour)
PC:	Permeability Coefficient	(cm/hour)
SA:	Skin surface area for dermal absorption	(cm ²)

4.3. Exposure Parameter Estimates

Values selected and assumptions made for the RME and CT scenarios are presented in the RAGS Table 4 Series and discussed below.

4.3.1. Age Dependent Adjustment for Chemicals with Mutagenic Mode of Action

Those constituents listed in the USEPA's 2006 memorandum (USEPA 2006) as having a Mutagenic Mode of Action (MMOA) are subject to adjustment by an age-dependent adjustment factor using Age Dependent Adjustment Factors (ADAFs) as described in *Supplemental Guidelines for Assessing Susceptibility From Early Life Exposure to Carcinogens - Supplemental Guidance* (USEPA 2005). This ADAF evaluation required the modification of the RAGS Table 4 Series to include the specific age bins identified in the Supplemental Guidance (0 to 2 years, 2 to 16 years, >16 years, and all

subgroups within these age bins). This ADAF evaluation was derived specifically for this assessment by using the *Wastebeds 1 through 8 Bike Trail HHRA* (USEPA 2007c) as an example.

There are no ADAF exposure factors for the ingestion of fish tissue included in the RAGS Table 4 Series. This is because the EPCs for fish tissue were taken from the Onondaga Lake HHRA, and these EPCs did not include any constituents that exhibit a MMOA.

It should be noted that other PAHs considered toxicologically related to benzo(a)pyrene, based on the *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons* (USEPA 1993), are not included on the list of chemicals with a MMOA (USEPA 2006) but are subject to an ADAF as well.

Vinyl chloride is listed in USEPA's 2006 memorandum (USEPA 2006), but this constituent is a special case with respect to age adjustment. The USEPA has published a carcinogenicity assessment for exposure to vinyl chloride from birth in addition to adulthood (IRIS Vinyl Chloride guidance). As indicated in this assessment, the cancer slope factor (CSF)/unit risk for vinyl chloride is simply given a two-fold adjustment when exposure occurs from birth as opposed to from adulthood. Therefore, within RAGS Table 6 there are two CSF/unit risk values presented for vinyl chloride; one for exposure from birth and one for exposure from adulthood. No further age-adjustment of vinyl chloride is necessary.

4.3.2. Dermal Adsorption Factor

The dermal absorption factor (ABS, unitless) represents the fraction of the soil constituent that may be absorbed through the skin over each exposure event. In general, metals are poorly absorbed through the skin where as organic constituents may be absorbed more readily. Constituent-specific values were obtained from USEPA Risk Assessment Guidance (RAGS Part E, USEPA 2004b, Exhibit 3-4). Table 2 of that document presents the specific values for each constituent. If chemical-specific data for dermal absorption were not available, 100% dermal absorption was assumed.

4.3.3. Soil to Skin Adherence Factor

Soil to skin adherence factors (AF, mg/cm²) represent the average mass of soil that adheres to the skin over each exposure event. The AF depends on the specific activity being conducted and is higher for body parts with greater exposure to the soils. For example, the AF is higher for a construction worker than for an industrial worker, with greater adherence to the hands as compared with less exposed parts such as the head. AFs are therefore derived as the body part weighted average estimates for each receptor, considering the specific activities in which each receptor group is likely to participate. The specific RME and CT AFs obtained from USEPA Risk Assessment Guidance (RAGS Part E, USEPA 2004b, Exhibit 3-3) and applied for each receptor group as summarized below.

- For an older child trespasser or younger child recreator, the RME AF value is 3 mg/cm² and the CT value is 0.2 mg/cm². For an adult trespasser or recreator, the RME and CT values are 0.3 mg/cm² and 0.15 mg/cm², respectively.
- For utility workers exposed to soil, the RME AF is 0.3 mg/cm² for consistency with the Wastebeds 1 through 8 Site, while the CT value is 0.2 mg/cm², the geometric mean for utility workers (RAGS Part E, USEPA 2004b, Exhibit 3-3). For sediment, the RME value is 0.9 mg/cm², the 95th percentile for construction workers, and the CT value is 0.2 mg/cm². These sediment AF values also apply to drainage ditch workers.

- The RME and CT values for a surveillance worker are 0.07 mg/cm² and 0.01 mg/cm², respectively.
- For railroad workers, the RME and CT values are based on the 95th percentile and geometric mean, respectively, for staged activity: pipe layers (dry soil). The RME value is 0.2 mg/cm², and the CT value is 0.07 mg/cm².
- In the construction worker scenario, the RME value is 0.3 mg/cm² and the CT value is 0.1 mg/cm². These values correspond to the 95th percentile and geometric mean values for construction workers. The same values are used for commercial/industrial workers.
- For the child resident scenario, an RME AF of 0.2 mg/cm² is applied, consistent with USEPA guidance for a child resident (USEPA 2004b, Exhibit 3-3, page 3-14). The CT value is 0.04 mg/cm², consistent with the geometric mean for children playing in dry soil. For adult residents, RME and CT values of 0.07 and 0.01 mg/cm², respectively, are applied. The RME value is based on the geometric mean for gardeners as recommended on page 3-14 of RAGS Part E Exhibit 3-3 (USEPA 2004b).

4.3.4. Averaging Time

The averaging time (AT, days) is the time period over which exposure is averaged. In accordance with USEPA guidance (USEPA 1989a, Exhibits 6-11 through 6-16), the averaging time for exposure to potential carcinogenic compounds (AT-C) is 25,550 days. This accounts for exposure to a carcinogenic substance over a 70-year lifetime. For exposure to non-carcinogens, the averaging time (AT-NC) is calculated as the exposure duration (years) multiplied by 365 days per year (USEPA 1989a, Exhibits 6-11 through 6-16). The averaging time for exposure to non-carcinogenic substances therefore varies for receptors depending on their exposure duration.

4.3.5. Body Weight

The body weight (BW, kg) estimates are receptor-specific for adults, older children, and younger children. For adults, a default body weight of 70 kg was applied (USEPA 1991, Section 8.0 Summary Table). This is slightly less than the mean body weight for men and women aged 18 to 74 years in the United States (71.8 kg). For older children (ages 12 to <18 years), a body weight of 56 kg was used based on values for 12 to 17 year old boys and girls reported by USEPA, 1997a (*Exposure Factors Handbook*, USEPA 1997a, Table 7.3) averaged over the age range. A body weight of 15 kg was used for younger children (less than 6 years old), the default given in USEPA risk assessment guidance (RAGS vol. 1, USEPA 1991, Section 8 Summary Table).

4.3.6. Exposure Duration, Frequency, and Time

Values for exposure duration (ED), exposure frequency (EF), and exposure time (ET) for each exposure scenario are summarized in **Appendix G** and explained below.

Exposure duration (in years) is an estimate of the time period over which a receptor is exposed. This parameter is receptor-specific:

- For the current/future trespasser and recreator scenarios (Sections 2.1.2 and 2.1.4) and the future recreator and resident scenarios (Sections 2.1.2 and 2.1.4), the exposure duration was assumed to be 6 years for an older child and 9 years for an adult in the CT scenario. In the RME scenario, the older child and adult EDs were assumed to be 6 years and 30 years, respectively. These are USEPA recommended values for water contact in residential scenarios (RAGS Part E, USEPA 2004b, Exhibit 3-2).
- The current/future utility, surveillance, drainage ditch, railroad, and commercial/industrial worker scenarios (Sections 2.1.2 and 2.1.4) and future commercial/industrial worker scenario (Section 2.1.4) use ED values of 9 years in the CT scenario and 25 years in the RME scenario, based on USEPA values for soil contact in industrial scenarios (RAGS Part E, USEPA 2004b, Exhibit 3-5).
- For a future construction worker (Section 2.1.4), the exposure duration is estimated to be 1 year. This value is based on professional judgment, assuming that 1 year is a conservative estimate of the duration of a typical construction project.

Exposure frequency (in days/year) is a receptor-specific parameter that estimates how frequently the receptor exposure occurs:

- For current/future adult and older child trespassers, as well as current/future adult and younger child recreators, EF values are based on professional judgment. The CT EF is 32 days/year; the RME values are 42 days/year for an older child and 42 days/year for an adult. The 42-day EF is based on the assumption that the trespassing occurs twice per week during the ten summer weeks and once per week during the 22 weeks when the temperature is above 50°F (USEPA correspondence, April 25, 2008). However, for the case of exposure via ingestion of fish tissue, the current/future adult and older child trespassers are given EF values of 365 days/year for the CT and RME scenarios, following the guidance of USEPA (*Exposure Factors Handbook*, Vol. 2, USEPA 1997a, pages 10-26).
- The RME exposure frequency for a current/future utility worker is 20 days/year, based on best professional judgment. The CT exposure frequency is 5 days/year based on professional judgment.
- For a current/future surveillance worker, the EF is calculated to be 37 days/year in both the CT and RME scenarios. This assumes that surveillance occurs once per week, with two weeks of annual vacation, and a 25% reduction due to snow cover.
- Note that the snow cover percentage of 25% is derived from a 30 year (1971-2000) temperature and snowfall record for Syracuse, NY from the National Weather Service (http://www.erh.noaa.gov/bgm/climate/syr/syr_normals.shtml). During the months of December, January, and February, the average measurable snowfall is 28.6, 33.2, and 24.0 inches, respectively, and average temperatures are below freezing (28.6, 22.7, and 24.5 °F, respectively). Therefore, it can be assumed that the snow that falls during these months does not readily melt and provides continuous snow cover. Since the three months represent one-quarter of the year, a snow cover percentage of 25% is utilized. This percentage is conservative in that the months of March and November are not included. March and November on average have measurable snowfall (18.8 and 11.1 inches, respectively), but also have average temperatures above freezing (33.6 and 39.7 °F, respectively). The “Climate of New York” issued by the New York State Climate Office (<http://nysc.eas.cornell.edu/>) also describes a three month period of snow cover

for the Syracuse area: “The Southern Plateau, Great Lakes Plain in southern portions of western upstate New York, and the Hudson Valley experience a continuous snow cover from about mid-December to mid-March, with maximum depths usually occurring in February.”

- In the current/future drainage ditch worker scenario, a CT EF of 5 days/year and a RME EF of 10 days/year is applied based on professional judgment.
- The EFs for the railroad worker are 164 days/year in the CT scenario and 188 days/year in the RME scenario. The CT value assumes 219 work days per year, while the RME values assumes 250 work days per year. Both values are then reduced by 25% to account for snow cover.
- For the current/future commercial/industrial worker scenario, a CT EF of 219 days/year is applied (RAGS Part E, USEPA 2004b, Exhibit 3-5). The RME EF is 250 days/year (RAGS Vol. 1, USEPA 1991).
- The EF for a future construction worker is estimated to be 125 days/year for the CT scenario and 250 days/year for the RME scenario based on professional judgment. Given an exposure duration (ED) for the construction worker of 1 year for both scenarios, 250 work days assumes 12 months on-site and 125 work days assumes 6 months on-site.
- For future adult and younger child recreators, a CT and RME EF values of 32 and 42 days/year, respectively, are typically applied, which is consistent with the assumptions for current recreators. However, for the case of exposure via ingestion of fish tissue, the future adult and younger child recreators are given EF values of 365 days/year for the CT and RME scenarios, following the guidance of USEPA (*Exposure Factors Handbook*, Vol. 2, USEPA 1997a, pages 10-26).
- The CT and RME EF value for future adult and younger child residents is 350 days/year, which is consistent with the USEPA recommendation for residential water contact scenarios (RAGS Part E, USEPA 2004b, Exhibit 3-2).

Exposure time (in hours/day) is a receptor-specific parameter applies to inhalation exposure and describes the length of time for which exposure occurs.

- For the current/future adult or older child trespasser scenario, the ET is estimated to be 4 hours/day or 2 hours/day in the RME or CT scenarios, respectively. These values are based on professional judgment.
- For current/future utility, drainage ditch, or commercial/industrial worker and future construction and commercial/industrial worker scenarios, an ET of 8 hours/day, which is the length of a typical work day, is applied in both the CT and RME scenarios.
- The ET values uses for a current/future surveillance worker are 8 hours/day in the RME scenario and 1 hour/day in the CT scenario. For the latter value, it is assumed that a surveillance worker spends most of the shift inside a vehicle.
- For a current/future railroad worker, an EF value of 2 hours/day is applied in the CT and RME scenarios and is consistent with 25% of the work day being spent on-site.

- For the current/future and future adult or younger child recreator scenarios, a CT EF value of 2 hours/week and a RME EF value of 4 hours/week are applied.
- In the future resident scenario, the EF values are 16 hours/day for an adult resident and 24 hours/day for a younger child resident in both the CT and RME scenarios. This conservatively assumes that a young child may spend all day in the residence.

4.3.7. Ingestion Rate

Ingestion rate values for incidental ingestion of soils and ingestion of drinking water are presented below. Exposure parameters for all the receptors considered in this Risk Assessment, including those discussed below, are summarized in **Appendix G**.

IRsoil: Incidental ingestion rate for soil (mg/day).

- The *IRsoil* for a current/future trespasser is assumed to be 100 mg/day for an older child and 50 mg/day for an adult, consistent with USEPA recommendations (*Exposure Factors Handbook*, USEPA 1997a, Table 4.23). The RME and CT values are identical.
- For a current/future utility worker, the RME ingestion rate is 330 mg/day and the CT ingestion rate is 100 mg/day. These values are consistent with EPA guidance for the *IRsoil* for construction workers and non-residential outdoor workers, respectively (*Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, USEPA 2002, Exhibit 1-2).
- The *IRsoil* for a current/future surveillance or railroad worker is 100 mg/day for both CT and RME scenarios, which is consistent with EPA guidance for non-residential outdoor workers (*Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, USEPA 2002, Exhibit 1-2).
- In the current/future drainage ditch worker scenario, the *IRsoil* value is 330 mg/day for the CT and RME scenarios. This is consistent with EPA guidance (*Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, USEPA 2002, Exhibit 1-2) and the Onondaga Lake HHRA (*Onondaga Lake Human Health Risk Assessment*, NYSDEC 2002).
- For a current or future commercial/industrial worker, the RME *IRsoil* value is 100 mg/day and is consistent with EPA guidance for non-residential outdoor workers (*Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, USEPA 2002, Exhibit 1-2). The CT *IRsoil* is 50 mg/day, based on guidance from USEPA (*Risk Assessment Guidance for Superfund*, USEPA 1991, Section 8 Summary Table).
- The *IRsoil* value for a current/future or future adult recreator or resident is 50 mg/day in both the RME and CT scenarios. For a younger child recreator or resident, the CT value is 100 mg/day. These values are consistent with EPA recommendations (*Exposure Factors Handbook*, USEPA 1997a, Table 4.23). An RME value of 200 mg/day for a younger child recreator or resident is applied in the RME scenario following the USEPA recommendation that 200 mg/day may be used as a conservative estimate of the mean (*Exposure Factors Handbook*, USEPA 1997a, Table 4.23).

- For a future construction worker, the IR_{soil} value is 330 mg/day in both the CT and RME scenarios and is consistent with EPA guidance for construction workers (*Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites*, USEPA 2002, Exhibit 1-2).

From the soil ingestion rates above, we observe that the commercial/industrial worker has a lower CT ingestion rate of soil than the surveillance and railroad workers, because the commercial/industrial worker is an indoor worker, whereas the other receptors are outdoor workers. As an outdoor worker, the utility worker has the same CT ingestion rate of soil as the surveillance and railroad workers. The construction worker has a higher ingestion rate of soil than the other receptors because USEPA (2002b) *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites* (Exhibit 1-2) provides specific factors for construction.

IR_{water} (potable): Ingestion rate for drinking water (L/day). Site-wide ground water is considered potable water. Consequently, water intake is assumed to be 2 L/day for adult residents and 1 L/day for younger child residents and is consistent with USEPA guidance (RAGS Part A, USEPA 1989a, Exhibit 6-11). The RME and CT IR_{water} values are identical.

4.3.8. Inhalation Rate

The inhalation rate (InR, m³/hour) depends on individual characteristics such as age, gender, weight, health, and activity level. The receptor-specific values are described below:

- For a current/future older child trespasser, an InR of 1.2 m³/hour is applied and is consistent with USEPA recommendations for children engaged in moderate activity (*Exposure Factors Handbook*, USEPA 1997a, Table 5-23). The CT and RME InR values are identical. For an adult trespasser, the RME value is 1.6 m³/hour and the CT value is 1.0 m³/hour based on USEPA recommendations for adults engaged in moderate and light activity levels, respectively (*Exposure Factors Handbook*, USEPA 1997a, Table 5-23).
- In the current/future utility and drainage ditch worker scenario, an InR of 1.5 m³/hour is used that is consistent with USEPA recommendations for an outdoor worker during moderate activity (*Exposure Factors Handbook*, USEPA 1997a, Table 5-23). The CT and RME InR values are identical.
- A current/future surveillance worker would likely be involved in activities with less physical exertion than a utility worker. Therefore, an InR value of 1.0 m³/hour is applied in both the RME and CT scenarios, following USEPA recommendations for adults engaged in light activities (*Exposure Factors Handbook*, USEPA 1997a, Table 5-23).
- For a current/future railroad worker, the CT value is 1.5 m³/hour and the RME value is 2.5 m³/hour based on USEPA recommendations for outdoor workers engaged in moderate and heavy activity, respectively.
- The InR for a current or future commercial/industrial worker is 1.6 m³/hour and is consistent with USEPA recommendations for adults at moderate activity levels (*Exposure Factors Handbook*, USEPA 1997a, Table 5-23). The CT and RME InR values are identical.

- For a current/future or future adult recreator, the RME value is 1.6 m³/hour and the CT value is 1.0 m³/hour, which is consistent with USEPA recommendations for adults engaged in moderate and light activity, respectively. For a current/future younger child recreator, the RME value is 1.2 m³/hour and the CT value is 1.0 m³/hour, following USEPA recommendations for children engaged in moderate and light activity, respectively levels (*Exposure Factors Handbook*, USEPA 1997a, Table 5-23).
- For the future construction worker scenario, an RME InR value of 3.2 m³/hour and a CT InR value of 1.6 m³/hour is applied based on USEPA recommendations for adults engaged in heavy and moderate activity, respectively (*Exposure Factors Handbook*, USEPA 1997a, Table 5-23).
- The InR values for a future adult and child resident are 0.8 m³/hour and 0.42 m³/hour, respectively. These are conservative estimates based on USEPA recommendations (*Exposure Factors Handbook*, USEPA 1997a, Table 5-11; USEPA 2002, Exhibit 1-2). The RME and CT InR values are identical.

4.3.9. Permeability Coefficient

The permeability coefficient (K_p , cm/hour) represents the rate at which dissolved constituents in water migrate across the skin into the bloodstream. Chemical-specific dermal permeability coefficients from USEPA (*RAGS Part E*, USEPA 2004b, Exhibits B-3 and B-4) were applied. The values for each constituent are presented in **Table 2** (attached).

4.3.10. Skin Surface Area Estimates

Skin surface area (SA) for dermal absorption from water (cm²) and soil (cm²/day). This parameter represents the exposed surface area of the skin, which may contact water or soil. The receptor and media specific values are summarized in **Appendix G**.

- For an older child trespasser, a SA value of 5400 cm² is applied and is consistent with NYSDEC guidance (NYSDEC 2002, *Onondaga Lake HHRA*). For an adult trespasser, the value is 5700 cm² consistent with NYSDEC and USEPA guidance (NYSDEC 2002, *Onondaga Lake HHRA*; USEPA 2004b, Exhibit C-1). The RME and CT SA values are identical.
- The SA value for a utility, drainage ditch, railroad, commercial/industrial, or construction worker is 3300 cm², based on USEPA guidance for construction and outdoor workers (USEPA 2002, Exhibit 1-2). The RME and CT SA values are identical.
- For the surveillance worker scenario, the SA RME value is 2480 cm², which is consistent with guidance from USEPA (USEPA 2004b, Exhibit C-1). This assumes that hands, forearms, and face are exposed. The SA CT value of 1930 cm²/day assumes that only the head and hands are exposed because a worker would be wearing long sleeves for much of the year.
- For a younger child recreator, a SA value of 2800 cm² is applied, and for an adult recreator a SA value of 5700 cm² is used. These values are consistent with guidance from the USEPA (USEPA 2002, Exhibit 1-2). The adult value includes exposure of the head, hands, forearms, and lower legs, while the younger child value includes exposure of the head, hands, forearms, lower legs, and feet. The RME and CT SA values are identical.

- The younger child and adult resident scenarios assume that the entire body is exposed during showering or bathing. Consequently, the SA values for water exposure are 6600 cm² and 18,000 cm² for a child and adult, respectively, based on guidance from the USEPA for residential scenarios (USEPA 2004b, Exhibit 3-2). For contact with soil, the exposed surface area is limited to areas not covered by clothing. The SA values for soil exposure are 2800 cm² and 5700 cm², consistent with the values for a child recreator and USEPA guidance (USEPA 2002, Exhibit 1-2). The RME and CT SA values are identical.

4.3.11. Event Duration and Frequency

The event duration and frequency together describe the amount of time during which the receptor is in contact with water.

t_{event} : Event duration (hours/event). The receptor-dependent event duration is described below.

- For an adult or older child trespasser, and adult or younger child recreator, the RME event duration is 4 hours/event and the CT event duration is 2 hours/event. These RME and CT values are based on the assumption of 4 or 2 hours/day, respectively, being spent on recreation.
- The event duration for a utility, drainage ditch, or construction worker is 8 hours/day, based on a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers). The RME and CT values are identical.
- The event duration for residents is based on the amount of time spent showering or bathing. For a younger child resident, the RME value is 1 hour/event and the CT value is 0.33 hour/event. For an adult resident, the RME and CT values are 0.58 and 0.25 hour/event, respectively. These values are consistent with USEPA guidance (USEPA 2004b, Exhibit 3-2).

EV: Event frequency (events/day). In this study, the event frequency for all relevant receptors is once per day.

4.3.12. Fraction Absorbed

FA: Fraction absorbed water (unitless). Chemical specific values for FA are based on USEPA guidance (USEPA 2004b, Exhibits B-3 and B-4) and summarized in **Table 2** (attached).

4.3.13. Lag Time per Event

τ_{event} : Lag time per event (hours/event). The chemical-dependent values for lag time are based on USEPA guidance (USEPA 2004b, Exhibits B-3 and B-4) and summarized in **Table 2** (attached).

4.3.14. Beta Constant

B: Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve) (unitless). B values are chemical specific and based on guidance from EPA (USEPA 2004b, Exhibits B-3 and B-4). **Table 2** (attached) reports the B values for this study.

4.3.15. Time to Reach Steady State

t^* : Time to reach steady state (hours). The chemical-dependent values for t^* are based on USEPA guidance (USEPA 2004b, Exhibits B-3 and B-4) and summarized in **Table 2** (attached).

5. Toxicity Assessment

The purpose of the toxicity assessment is to evaluate available information regarding the potential for Site-related chemical residues of potential concern to cause adverse effects in exposed individuals. The potential toxicological effects resulting from a given dose of a chemical are classified according to two criteria, consisting of non-cancer effects (hazards) and cancer effects (risks). The toxicity assessment presented herein was completed according to USEPA guidance (USEPA 1989a). In particular, toxicity values were obtained from a hierarchy of sources, described in Section 5.3. The hierarchy consists of Tier 1 - EPA's Integrated Risk Information System (IRIS); Tier 2 - Provisional Peer-Reviewed Toxicity Values (PPRTV) used in USEPA's Superfund Program; and Tier 3 - other peer-reviewed toxicity values.

5.1. Non-Cancer Effects

A non-cancer health effect occurs as a result of damage to cells in one or more human organs, which causes the organ(s) to function less efficiently (or not at all). Due to the body's ability to cope with small doses of most substances, a non-cancer health effect will not occur if intake of a chemical is less than a certain threshold dose. This threshold dose is referred to as a "no observed adverse effect level" (NOAEL) for a substance. From a NOAEL, a reference dose (RfD) is calculated and compared with the calculated intake of a constituent. If the calculated intake in a given species is less than the RfD for a constituent, then no adverse non-cancer health effects are expected as a result of that exposure.

The specific non-carcinogenic toxic effects that may be elicited depend on the exposure concentration and the duration of exposure. If an individual is exposed to very high concentrations of a substance, severe organ dysfunction can occur in a short period of time. This is referred to as an acute toxic effect. If an individual is exposed to lower levels of a substance regularly for a long period of time, smaller amounts of repeated damage to an organ can accumulate and cause the organ to malfunction. These are termed sub-chronic and chronic toxic effects (depending on the exposure duration).

A brief discussion of the methods by which RfDs are derived is presented below. For some constituents, RfDs are derived directly from data on human exposures. This may include data relating to occupational exposures, normal dietary levels of certain constituents (*e.g.*, magnesium), therapeutic doses of certain constituents (*e.g.*, silver), and epidemiological data relating to populations with background exposures (*e.g.*, selenium) or accidental exposures (*e.g.*, mercury).

For most constituents, the USEPA derives RfDs based on laboratory studies in which experimental animals were exposed to different concentrations of a constituent, and a NOAEL is identified or estimated. If data from several animals' studies are available, USEPA seeks to identify the species that is most comparable to humans, based on knowledge of specific biological properties. However, if adequate comparative data is not available, USEPA selects the study on the most sensitive animal species as the critical study for the basis of the NOAEL. The NOAEL is then used to derive a RfD for potential adverse effects in human populations.

In most cases, there is considerable uncertainty regarding the extension of toxicological data from animal studies to humans (see Section 7). In other words, the actual RfD for humans or sensitive sub-populations of humans (*e.g.*, children, the elderly) would not be precisely known based on data from laboratory studies with animals. This uncertainty arises because there may be differences between the

animal and human species regarding factors such as the metabolism of the constituent, the distribution and clearance rate of the constituent from the body, and the sensitivity of the specific organ systems to the constituent. Therefore, the USEPA derives RfDs that are designed to be protective of the public at large, including sensitive sub-populations.

To accomplish this, the USEPA applies a series of uncertainty factors to calculate a final, conservative RfD value. Depending on many parameters of the study/studies reviewed, the NOAEL may be divided by an uncertainty factor ranging from 0 to 10,000. This means that the reported no observed adverse effect level for the given test is then divided by several orders of magnitude. For human data an uncertainty factor of 10 is usually applied for the application of data from the public at large to sensitive sub-populations. For animal data the uncertainty factor of 100 (10 for sensitive sub-populations and 10 for animal to human extrapolation) is applied for deriving the human RfD.

5.2. Cancer Effects

To evaluate cancer risks, the USEPA has developed cancer slope factors (CSFs), which are expressed as risks per $(\text{mg/kg-day})^{-1}$. The CSFs are derived using a low-dose extrapolation procedure, which assumes that there is no threshold for the induction of cancer (as opposed to non-cancer toxicity, where it is assumed that certain doses will not produce adverse health effects). COPCs operating with a mutagenic mode of action were evaluated following USEPA (2006) guidance on age dependent adjustment factors. Section 4.3.1 provides a more detailed discussion of the treatment of chemicals with an MMOA. For vinyl chloride, EPA's IRIS provides two different values of the CSF, one representing lifetime exposure from birth and one representing lifetime exposure during adulthood. The HHRA uses the exposure from birth CSF for receptors under age 18 and the exposure during adulthood CSF for adult receptors.

Weight of evidence – USEPA classifies substances according to their potential to induce cancer in humans. The USEPA reviews and evaluates available data regarding the potential carcinogenic effects of a constituent, and assigns a “carcinogenicity” classification according to a weight of evidence classification scheme (49 CFR 462394). A constituent may be classified into one of five groups with respect to the weight of evidence for human carcinogenicity. The categories are:

- Group A – Known Human Carcinogen. A constituent is classified in Group A if there is sufficient evidence from human observations (epidemiological studies) to support an association between exposure to a chemical agent and cancer in humans
- Group B1 – Probable Human Carcinogen. A constituent is classified as a B1 carcinogen if there is sufficient evidence for carcinogenicity based on animal studies and limited (suggestive but not conclusive) evidence based on human observations.
- Group B2 – Probable Human Carcinogen. A B2 carcinogen is a constituent for which there is sufficient evidence for carcinogenicity in animals and inadequate evidence for carcinogenicity in humans.
- Group C – Possible Human Carcinogen. A constituent is classified as a Group C carcinogen if there is limited evidence for carcinogenicity in animals and inadequate evidence for carcinogenicity in humans.

- Group D – A constituent is classified as a Group D agent if there is insufficient data available with which to evaluate the carcinogenicity of the constituent.

Slope Factors – For Group A, B, or C chemicals, USEPA derives chemical-specific CSFs. A CSF is a number which, when multiplied by the estimated chemical-specific CDI, provides an estimate of the “excess cancer risk” associated with that exposure. Theoretically, the excess cancer risk represents the lifetime probability (greater than background) that a carcinogenic event would occur in an individual as a result of a given exposure or pattern of exposures. It is important to note that for many chemicals, the excess cancer risk as calculated by USEPA’s procedure is likely to result in a conservative and health protective overestimate of the potential cancer risk.

5.3. Derivation of Toxicity Values – Hierarchy

For each constituent that was retained as a COPC, a brief synopsis of the human toxicological effects, including chronic RfDs and CSFs was compiled from the following hierarchy of sources listed below:

- Tier 1 - EPA’s Integrated Risk Information System (IRIS).
- Tier 2 - Provisional Peer-Reviewed Toxicity Values (PPRTV) used in USEPA’s Superfund Program.
- Tier 3 - Other (peer-reviewed) toxicity values, including:
 - Minimal Risk Level produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - California Environmental Protection Agency (CalEPA) values, and
 - EPA Health Effects Assessment Summary Table (HEAST) values.

Third tier toxicological values were not used in this assessment unless these values were supplied by the USEPA Superfund Technical Support Center (STSC).

The non-cancer toxicity data applied in the risk characterizations of oral/dermal exposures evaluated in this report are presented in RAGS Table 5-1. Non-cancer toxicity data applied for the inhalation of outdoor air is presented in RAGS Table 5-2. The cancer toxicity data applied in the risk characterizations of oral/dermal exposures evaluated in this document are presented in RAGS Table 6-1. Cancer toxicity data applied for the inhalation of outdoor air is presented in RAGS Table 6-2. All values in RAGS Tables 5 and 6 were taken either from the IRIS or were supplied by the STSC.

The values provided by the STSC can be divided into two groups. The first group of toxicity values provided by the STSC is labeled as PPRTV on the subject RAGS Tables 5 and 6. The PPRTV label indicates that the value was presented in a Provisional Peer Reviewed Toxicity Information report supplied to Honeywell by the USEPA. The date associated with the PPRTV value is the date of the specific report for that constituent (*e.g.*, RfC for Aluminum, PPRTV report dated October 23, 2006).

The second group of toxicity values provided by the STSC is labeled according to their original source on the subject RAGS Tables 5 and 6 (ATSDR, HEAST, CalEPA, *etc.*). The use of these toxicity values was approved by the USEPA in electronic mail communications to Honeywell but there are no Provisional Peer Reviewed Toxicity Information reports associated with these toxicity values. For example, a March 27, 2008 email from R. Nunes (US EPA Region II) to T. Conklin and P. Sinha (O'Brien & Gere) contained a spreadsheet that endorsed values for several constituents from these sources (ATSDR, HEAST, CalEPA). This spreadsheet lists a CalEPA value for the arsenic RfC. The source of the RfC is listed as CalEPA (STSC) on the subject RAGS Table 5.2 to indicate that this value originated from the CalEPA website and was approved by the STSC as per the March 27, 2008 email. The dates listed on RAGS Table 5 and 6 for the toxicity values selected from these sources follows USEPA protocol (current dates for electronic sources [CalEPA] and date of publication for non-electronic sources).

Appendix H (electronic) includes all of the agency email communications related to toxicity values that were from sources other than IRIS or an official PPRTV reports (ATSDR, HEAST, CalEPA, *etc.*).

5.4. Adjustment for Dermal Toxicity

Assessing toxicity associated with dermal exposure to constituents in soil and water requires special considerations. Dermal toxicity of a substance depends on factors including the analyte concentration in contact with the skin, the potential dose, the area of skin surface exposed, the exposure duration, the absorption of the analyte through the skin, the internal dose, and the amount of analyte that can be delivered to a target organ (*i.e.*, biologically effective dose) (USEPA 1997a).

In most instances, it was necessary to use oral toxicity data to estimate dermal toxicity. The dermal CDI represents the absorbed dose of the analyte. However, for many constituents, the oral toxicity data is based on the administered dose rather than the absorbed dose. Therefore, in order to assess dermal exposures, the oral toxicity data was adjusted to reflect the absorbed dose in accordance with USEPA guidance (USEPA 2004b) as follows:

$$RfD_{\text{dermal}} = RfD_{\text{oral}} \times \text{Gastrointestinal absorption efficiency (ABS}_{\text{GI}})$$

$$CSF_{\text{dermal}} = CSF_{\text{oral}} / \text{Gastrointestinal absorption efficiency (ABS}_{\text{GI}})$$

The gastrointestinal absorption efficiency data used for evaluating dermal exposures were obtained from Exhibit 4-1, USEPA (2004b). The RfD_{oral} and the CSF_{oral} were calculated using the above equations for constituents with an ABS_{GI} of less than 50 percent. Otherwise, no absorption adjustment was made (USEPA 2004b).

5.5. Chemical Specific Summaries and Toxicology Values for Risk Drivers

Toxicological summary information is provided below for all the constituents identified as risk drivers in Section 6 of this report.

5.5.1. Benzene

Benzene potentially contributes to Site-wide carcinogenic risk to commercial/industrial workers exposed to ground water (as potable water) in the future scenario. Benzene also potentially contributes to the non-carcinogenic hazard for three receptor populations exposed to Site-wide ground water (as potable water) in the future exposure scenarios – commercial/industrial workers, child resident, and adult resident (Sections 6.1.9 and 6.1.10).

Benzene is a volatile constituent of crude oil and refined gasoline and motor fuels. Benzene is also a byproduct of the production of coke. It is also used extensively in industry as a raw material or chemical intermediate for the production of other chemicals, such as styrene and phenols and the manufacture of plastics, resins, detergents, pharmaceuticals, pesticides, and dyes (ATSDR 1997).

The short-term effects of ingesting large amounts of benzene include vomiting, stomach irritation, convulsion, increased heart rate, and ultimately death. The oral and dermal reference dose for benzene is 4.0×10^{-3} mg/kg-day (IRIS accessed September 2008) and the inhalation reference concentration is 3.0×10^{-2} mg/m³ (converted to 5.5×10^{-2} mg/kg-day) (IRIS accessed September 2008).

Benzene is classified as a Group A Carcinogen (Known Human Carcinogen). A chemical is classified as Group A if there is sufficient evidence from human observations (epidemiological studies) to support an association between exposure to a chemical agent and cancer in humans. Chronic exposure to benzene produces blood changes, causing several forms of leukemia and harmful effects of the bone marrow resulting in anemia (Sittig 1981; ATSDR 1997). The inhalation unit risk (IUR) for benzene ranges from 2.2×10^{-3} to 7.8×10^{-3} (mg/m³)⁻¹ (IRIS accessed September 2008). The upper end of this range [7.8×10^{-3} (mg/m³)⁻¹] was used as the IUR for this assessment and was converted to an inhalation cancer slope factor of 2.7×10^{-2} (mg/kg-day)⁻¹. An oral slope factor of 5.5×10^{-2} (mg/kg-day)⁻¹ was derived by the USEPA from IUR factor and is used in this assessment (IRIS accessed September 2008).

5.5.2. 2-Methylnaphthalene

2-Methylnaphthalene potentially contributes to Site-wide non-carcinogenic hazard to the child recreator and construction worker exposed to surface and subsurface sediment under the future scenario (Section 6.1.7). This compound is a natural component of crude oil and coal and is found as a pyrolytic byproduct from the combustion of tobacco, wood, petroleum-based fuels and coal. It is also used in the production of pesticides and as a chemical intermediate in the synthesis of vitamin K (Hazardous Substances Data Bank [HSDB], accessed September 2008; IRIS 2003)

No data are available regarding the potential toxicity of 2-methylnaphthalene in exposed humans via the oral route. However, the available animal data indicate that the lung is a sensitive target organ. The critical effect observed in mice following chronic oral exposure to 2-methylnaphthalene (Murata *et al.* 1997) and chronic dermal exposure to methylnaphthalene mixtures (Emi and Konishi 1985; Murata *et al.* 1992) was pulmonary alveolar proteinosis. The oral and dermal reference dose for 2-methylnaphthalene used in this assessment is 4.0×10^{-3} mg/kg/day (IRIS accessed September 2008).

According to IRIS (accessed September 2008), the data regarding the carcinogenicity of 2-methylnaphthalene in mice and the lack of human carcinogenicity data are inadequate to assess human carcinogenic potential.

5.5.3. Dibenzofuran

Dibenzofuran potentially contributes to Site-wide non-carcinogenic hazard to the child recreator and construction worker exposed to surface and subsurface sediment under the future scenario (Section 6.1.7 and 6.1.8). Little information related to the use or toxicity of this compound is available. It is used as an insecticide and in organic synthesis (HSDB accessed September 2008). The oral and dermal reference dose for dibenzofuran used in this assessment was provided by the STSC as a PPRTV report (June 11, 2007) and is 1.0×10^{-3} mg/kg/day.

Dibenzofuran is classified as a Group D agent in IRIS (accessed September 2008) indicating that there is insufficient data available with which to evaluate the carcinogenicity of the constituent.

5.5.4. Naphthalene

Naphthalene potentially contributes to Site-wide non-carcinogenic hazard to the child recreator and construction worker exposed to surface and subsurface sediment under the future scenario (Section 6.1.7 and 6.1.8). Naphthalene is a white substance at room temperature. It has a distinct odor of mothballs or tar. Humidity and sunshine cause evaporation into air within a few hours. When placed in water or soil, bacteria will metabolize naphthalene or will render it airborne within a few hours (ATSDR 1990). The compound is used in the production of dyes, solvents, lubricants, motor fuels (HSDB accessed September 2008) and is a major component of many moth ball preparations.

Adults and children exposed to airborne naphthalene experience vomiting, abdominal pain and anemia (ASTDR 1990). Most of the data is from the inhalation of naphthalene from mothballs. The primary site of toxicity is the erythrocytes resulting in hemolytic anemia. Jaundice is seen upon dermal, inhalation, and oral exposures, as are kidney effects (ATSDR 1990). Several animal studies have demonstrated ocular changes (development of cataracts) following oral naphthalene exposure (Kojima 1992; Murano *et al.* 1993; Yamauchi *et al.* 1986). The oral and dermal reference dose for naphthalene used in this assessment is 2.0×10^{-2} mg/kg/day (IRIS accessed September 2008). The inhalation reference concentration is 3.0×10^{-3} mg/m³ (converted to 8.6×10^{-4} mg/kg/day) (IRIS, accessed September 2008).

Naphthalene is classified as a Group C carcinogen in IRIS (accessed September 2008) indicating that there is limited evidence for carcinogenicity in animals and inadequate evidence for carcinogenicity in humans. Carcinogenic risk from oral or dermal exposure to this constituent was not evaluated in this assessment because no approved oral or absorbed cancer slopes were available. However, the USEPA STSC did suggest that the Inhalation Unit Risk factor [3.4×10^{-2} (mg/m³)⁻¹] and the Inhalation Slope factor [1.2×10^{-1} (mg/kg-day)⁻¹] from the CalEPA be used in this assessment.

5.5.5. Polychlorinated Biphenyls

PCBs potentially contribute to Site-wide carcinogenic risk and non-carcinogenic hazard for trespassing and recreating receptors through the ingestion of fish tissue (Section 6.1.1).

PCBs are mixtures of up to 209 different compounds (congeners) that include a biphenyl and from one to ten chlorine atoms. "Aroclors" were commercial products marketed in the U.S. with differing amounts of the individual congeners. PCBs have been used as a dielectric fluid in electrical equipment such as transformers and capacitors due to their heat resistance and insulating properties. PCBs were also used in the ballasts of fluorescent lights and in hydraulic oils. They can be released to the environment during fires involving electrical equipment containing these compounds. PCBs are

strongly adsorbed on solid surfaces, including glass and metal surfaces in laboratory apparatus, and onto soils, sediment, and particulates in the environment.

1. Non-Cancer Toxicity – The non-cancer effects of PCB include dermatological effects, sore throat, skin rash, gastrointestinal disturbance, eye irritation, and headache, as well as higher serum triglyceride and/or cholesterol levels and high blood pressure at higher blood concentrations of PCBs.

For non-cancer toxicity, the Aroclors have been divided into two groups:

The “Less Chlorinated” Aroclors consist of Aroclors 1016, 1221, 1232, and 1242. This group was characterized in the HHRA by using the oral reference concentration for Aroclor 1016 (7.0×10^{-5} mg/kg-day, IRIS accessed September 2008). The dermal reference dose for the “less chlorinated” group was 7.0×10^{-5} mg/kg/day (IRIS accessed September 2008).

The “Highly Chlorinated” Aroclors consist of Aroclors 1248, 1245, 1260, and 1268. This group was characterized in the HHRA by using the oral reference concentration for Aroclor 1254 (2.0×10^{-5} mg/kg-day, IRIS accessed September 2008). The dermal reference dose for the “highly chlorinated” group was also 2.0×10^{-5} mg/kg/day (IRIS accessed September 2008).

2. Cancer Toxicity – Both groups of PCBs (“less chlorinated” and “highly chlorinated”) are classified as Probable Human Carcinogens (B2) in IRIS (accessed September 2008). A B2 carcinogen is an agent for which there is sufficient evidence for carcinogenicity in animals and inadequate evidence for carcinogenicity in humans. Note that for cancer toxicity, all detected Aroclors were summed as “total PCBs”; this total PCB value was then used to determine the exposure point concentration for cancer toxicity.

The IRIS database has a tiered set of CSFs and this HHRA utilizes the High Risk and Persistence Tier. The criteria used for this tier include food chain exposure, sediment or soil ingestion, dust or aerosol inhalation, any early-life exposure, and the presence of dioxin-like, tumor producing, or persistent congeners. Based on this approach, the CSFs applied for all PCB congeners for oral, dermal, and inhalation exposures were 2.0×10^0 (mg/kg-day) $^{-1}$, 2.0×10^0 (mg/kg-day) $^{-1}$, and 2.0×10^0 (mg/kg-day) $^{-1}$, respectively.

5.5.6. PCDD/PCDFs

PCDD/PCDFs potentially contribute to Site-wide non-carcinogenic hazard for trespassing (older child and adult) and recreating (adult) receptors through the ingestion of fish tissue. Ingestion of this constituent group in fish tissue also contributes to Site-wide carcinogenic risk for the adult trespasser as well as the adult recreator (Section 6.1.1).

PCDD/PCDFs are a group of 210 structurally related chlorinated chemicals that are ubiquitous in the environment. There are a total of 135 PCDFs and 75 different PCDDs. Sources of PCDD/PCDFs include incineration of municipal and certain industrial wastes, chlorination processes used in pulp and paper manufacturing and water treatment systems, and the production and use of certain chlorinated pesticides.

As discussed in Section 4.1.6, 2,3,7,8-TCDD is considered to be the most potent of the PCDD/PCDF compounds isomers with respect to potential toxic effects. 2,3,7,8-TCDD may induce a wide range of toxic effects in laboratory animals including effects on the liver, sex hormone balance, immune system, and *in utero* development (ATSDR 1998). Very few of the toxic effects of 2,3,7,8-TCDD observed in animal species have been reported in exposed human populations (USEPA 1992). The only toxic effect that has been definitively associated with TCDD exposure in human populations is chloracne (an acne-like skin condition) in heavily exposed individuals (USEPA 1992). The oral and dermal reference dose for 2,3,7,8-TCDD used in this assessment was taken from the ATSDR Minimal Risk Levels (MRL) table as suggested by the STSC and is 1.0×10^{-9} mg/kg/day. There was no inhalation reference concentration selected for this constituent group.

PCDD/PCDFs are classified as Probable Human Carcinogens (B2) in IRIS (accessed September 2008). A B2 carcinogen is an agent for which there is sufficient evidence for carcinogenicity in animals, and inadequate evidence for carcinogenicity in humans. 2,3,7,8-TCDD has been shown to induce cancer in laboratory animals at relatively low administered doses (USEPA 1992). In addition, certain studies of occupationally exposed workers in the United States (Fingerhut *et al.* 1991) and Germany (Manz 1991) have reported a possible increase in lung cancer and thyroid cancer rates in occupationally exposed workers. The oral and absorbed cancer slope for this constituent group was taken from USEPA (1997b) as suggested by the USEPA STSC. This value was 1.5×10^{-5} (mg/kg-day)⁻¹.

5.5.7. Polycyclic Aromatic Hydrocarbons

Only three of the 13 major Polycyclic Aromatic Hydrocarbons (PAHs) are discussed in this section: benzo(a)pyrene, benzo(a)anthracene, and phenanthrene. Phenanthrene potentially contributes to Site-wide non-carcinogenic hazard for the child recreator exposed to surface sediment under the future scenario. Benzo(a)anthracene potentially contributes to Site-wide carcinogenic risk for the older child trespasser (current/future) and the child recreator (future) exposed to surface sediment as well as to the future child resident exposed to ground water (modeled as potable water). Benzo(a)pyrene potentially contributes to the Site-wide carcinogenic risk for several scenarios and media (Section 6).

PAHs contain two or more aromatic rings. They are ubiquitous in nature and are both naturally occurring and human-made. PAHs are a component of fossil fuels and are produced from the incomplete combustion of organic compounds. PAHs are found in coal, creosote oils and pitches formed from the distillation of coal tars (ASTDR 1990).

1. Non-Cancer Toxicity – The oral reference doses for phenanthrene as well as other non-carcinogenic PAHs are presented in **Table 5.1** below. For non-carcinogenic PAH's without published reference doses the RfD for pyrene is used. This approach is consistent with the recommendations of the NCEA for PAH surrogates in the Onondaga Lake HHRA.

Table 5.1. Surrogates for Oral Reference Doses for Non-Carcinogenic PAHs.

Non-carcinogenic PAH	Published Oral RfD*	Proposed Surrogate	Oral and Dermal RfD for use in the HHRA
Pyrene	3.0×10^{-2}	NA	3.0×10^{-2} mg/kg/day
Acenaphthylene	NA	Pyrene	3.0×10^{-2} mg/kg/day
Benzo[g,h,i]perylene	NA	Pyrene	3.0×10^{-2} mg/kg/day
Phenanthrene	NA	Pyrene	3.0×10^{-2} mg/kg/day

2. Cancer Toxicity – There are several PAHs that are classified as a Probable Human Carcinogen (B2) in IRIS (accessed September 2008). A B2 carcinogen is an agent for which there is sufficient evidence for carcinogenicity in animals, and inadequate evidence for carcinogenicity in humans.

The USEPA IRIS database (accessed September 2008) has a published CSFs for benzo(a)pyrene of 7.3×10^0 (mg/Kg-day)⁻¹. Using this value and the relative potency approach provided by USEPA in the *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons* (USEPA 1993), the oral CSFs were calculated for the PAHs in **Table 5.2** below.

Table 5.2. Surrogates for Oral and Dermal CSF for Carcinogenic PAHs.			
Carcinogenic PAH	Published Oral CSF*	Relative Potency	Oral and Dermal CSF used in the HHRA
Benzo[a]pyrene	7.3×10^0 (mg/kg-day) ⁻¹	1.0	7.3×10^0 (mg/kg-day) ⁻¹
Benzo[a]anthracene	NA	0.1	7.3×10^{-1} (mg/kg-day) ⁻¹
Benzo[b]fluoranthene	NA	0.1	7.3×10^{-1} (mg/kg-day) ⁻¹
Benzo[k]fluoranthene	NA	0.01	7.3×10^{-2} (mg/kg-day) ⁻¹
Chrysene	NA	0.001	7.3×10^{-3} (mg/kg-day) ⁻¹
Dibenzo[a,h] anthracene	NA	1.0	7.3×10^0 (mg/kg-day) ⁻¹
Indeno[1,2,3-cd]pyrene	NA	0.1	7.3×10^{-1} (mg/kg-day) ⁻¹
NA = not available			
Source: USEPA Integrated Risk Information System (IRIS)			

The oral slope factors for all PAHs were not adjusted for the dermal route of exposure, according to guidance provided in USEPA RAGS, Part E (USEPA 2004b). The STSC suggested that the Inhalation Unit Risk factor [1.1×10^0 (mg/m³)⁻¹] and the Inhalation Slope factor [3.9×10^0 (mg/kg-day)⁻¹] from the CalEPA be used in this assessment for benzo(a)pyrene; however, the relative potency factor approach was not used to adjust the Inhalation Unit Risk values for the other PAHs.

5.5.8. Methylmercury

Estimates of potential risks associated with methylmercury are based on USEPA's current RfD of 1×10^{-4} mg/kg-day (IRIS 2001a). The current RfD, which was verified for use in 1995 and reassessed in 2001 by the USEPA, is based on protection against adverse effects that may occur following prenatal exposure during gestation. The USEPA initially derived the current RfD value from data for Iraqi infants accidentally exposed to alkyl mercury in grain during gestation in 1971 (Marsh *et al.* 1987, as cited in IRIS 2001a). In this population, delayed walking was reported in infants whose mothers had elevated hair methylmercury concentrations. The USEPA subsequently applied analyses of more recent studies as reported by the NRC (NRC 2000). NRC (2000) considered three epidemiological longitudinal developmental studies suitable for quantitative risk assessment: the Seychelles Islands, the Faeroe Islands, and New Zealand studies. The Seychelles study has yielded no evidence of impairment related to methylmercury exposure thus far, while the other two studies have found adverse effects for some neuropsychological endpoints. The Faeroe Islands study is the larger of the latter two studies, and was therefore recommended by NRC for use in derivation of an RfD. The USEPA agreed with the NRC's conclusions, and has proposed the same numeric RfD (0.1 µg/kg-day) based on neuropsychological findings from the Faeroe Islands data. The USEPA used a benchmark dose (BMD) approach to quantify a dose-effect relationship between methylmercury in cord blood and a neurological endpoint. A BMD limit of 58 µg/L cord blood was estimated based on findings from the Boston Naming Test, a neuropsychological evaluation. A methylmercury intake level associated with a blood level of 58 µg/L was calculated to be 1.0 µg/kg-day. A total uncertainty factor

of 10 was then applied, with the resulting RfD (*i.e.*, 0.0001 mg/kg-day), as derived from the Faeroe Islands data, unchanged from the RfD derived from the Iraqi data.

Methylmercury has been classified as Group C – Possible Human Carcinogen based on inadequate data in humans and limited evidence of carcinogenicity in animal studies (IRIS 2001a). No oral CSF has been established by USEPA, and, therefore, methylmercury is not assessed quantitatively for cancer risks in this HHRA.

6. Risk Characterization

Risk characterization is the final step of the risk assessment. It is defined as the combination of the exposure assessment and toxicity assessment to produce an estimate of risk and a characterization of uncertainties in the estimated risk. This section presents the results of the risk assessment for the Site.

6.1. Reasonable Maximum Exposure

Reasonable maximum exposure risks and hazards for Site receptors are presented in RAGS Part D Series Tables 7, 9, and 10. The RAGs Table 7 Series presents the derivation of risks and hazards for Site receptors by exposure medium. The RAGs Table 9 Series summarizes risks and hazards for a given Site receptor across all relevant media. The RAGs Table 10 Series summarizes risks and hazards for a given Site receptor across all relevant media for only those constituents that result in significant risks and/or hazards. The risk characterization discussion below focuses on overall risks and hazards to Site receptors across all relevant media, and identification of constituents that significantly contribute to those risks and hazards (RAGS Part D Table 9 Series).

6.1.1. Current/Future – Adult Trespasser; Older Child Trespasser (Cancer & Non-Cancer)

For the older child trespasser (RAGS Table 9.1 RME; Exposure Unit 1), the estimated total cancer risk is 1×10^{-3} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface sediment (7×10^{-4}), with benzo(a)pyrene and benz(a)anthracene contributing significantly (4×10^{-4} and 2×10^{-4} , respectively). Total cancer risk from exposure to fish tissue (1×10^{-4}), surface soil (2×10^{-4}), and surface water (3×10^{-4}) also exceeds the acceptable regulatory range, with benzo(a)pyrene contributing significantly in the latter two cases (1×10^{-4} for surface soil and 2×10^{-4} for surface water, respectively).

For the older child trespasser, the estimated hazard index of 2×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nervous system, ocular, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to fish tissue (2×10^1). 2,3,7,8-TCDD Equivalent, mercury (as methylmercury), and PCBs contribute significantly (hazard quotient > 1) to hazards from fish tissue.

Risks and hazards for the adult trespasser (RAGS Table 9.2 RME; Exposure Unit 1) are similar to those for the older child trespasser, with the adult trespasser having slightly higher total cancer risk (2×10^{-3}) and hazard index (3×10^1). Unlike the older child trespasser, the primary contribution to the total cancer risk is from exposure to fish tissue (8×10^{-4}), with 2,3,7,8-TCDD Equivalent and PCBs contributing significantly (5×10^{-4} and 3×10^{-4} , respectively). Total cancer risk from exposure to surface sediment (2×10^{-4}), and surface water (5×10^{-4}) also exceeds the acceptable regulatory range, with benzo(a)pyrene contributing significantly (9×10^{-5} and 4×10^{-4} , respectively).

For the adult trespasser, the estimated hazard index of 3×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for nervous system, ocular and other effects exceed 1. Exposure to fish tissue is the pathway contributing most significantly to the total hazard index, with the 2,3,7,8-TCDD Equivalent, mercury (as methylmercury), and PCBs contributing significantly (hazard quotient > 1).

6.1.2. Current/Future – Utility Worker (Cancer & Non-Cancer)

For the utility worker (RAGS Table 9.3 RME; Exposure Unit 1), the estimated total cancer risk is 4×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface water (2×10^{-4}), with benzo(a)pyrene contributing significantly (1×10^{-4}). The estimated hazard index of 8×10^0 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nasal/respiratory system and other effects exceed 1. The primary contribution to the total hazard index is from exposure to subsurface sediment (7×10^0) and subsurface soil (outdoor air) (1×10^1). 2-Methylnaphthalene, dibenzofuran, and naphthalene contribute significantly (hazard quotient > 1) to hazards from subsurface sediment.

For the utility worker in SYW-12 (RAGS Table 9.3a RME; Exposure Unit 9), total cancer risk is 4×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to shallow ground water (4×10^{-4}), with benzo(a)pyrene contributing significantly (3×10^{-4}). The SYW-12 hazard index of 2×10^{-1} is below the acceptable regulatory threshold of 1.

6.1.3. Current/Future – Surveillance Worker (Cancer & Non-Cancer)

For the surveillance worker (RAGS Table 9.5 RME; Exposure Unit 2), the estimated total cancer risk is 7×10^{-6} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The estimated hazard index of 1×10^{-1} is below the acceptable regulatory threshold of 1.

6.1.4. Current/Future – Drainage Ditch Worker (Cancer & Non-Cancer)

For the drainage ditch worker (RAGS Table 9.6 RME; Exposure Unit 3), the estimated total cancer risk (2×10^{-6}) and Site-wide hazard index (4×10^{-2}) are below acceptable regulatory limits.

6.1.5. Current/Future – Railroad Worker (Cancer & Non-Cancer)

For the railroad worker (RAGS Table 9.7 RME; Exposure Unit 4), the estimated total cancer risk is 9×10^{-6} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The estimated hazard index of 8×10^{-2} is below the acceptable regulatory threshold of 1.

For the railroad worker in SYW-12 (RAGS Table 9.7a RME; Exposure Unit 9), the estimated total cancer risk is 4×10^{-5} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The SYW-12 hazard index of 2×10^{-1} is below the acceptable regulatory threshold of 1.

6.1.6. Current/Future – Commercial/Industrial Worker (Cancer & Non-Cancer)

For the current/future commercial/industrial worker (RAGS Table 9.8 RME; Exposure Unit 5), the estimated cancer risk is 3×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface soil (3×10^{-4}), with benzo(a)pyrene contributing significantly (2×10^{-4}). The estimated hazard index of 9×10^{-1} is below the acceptable regulatory threshold of 1.

6.1.7. Current/Future – Adult Recreator; Child Recreator (Cancer & Non-Cancer)

For the child recreator (RAGS Table 9.10 RME; Exposure Unit 6), the estimated total cancer risk is 9×10^{-3} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface sediment (7×10^{-3}), with benzo(a)pyrene and benzo(b)fluoranthene contributing significantly (4×10^{-3} and 1×10^{-3} , respectively). Total cancer risk

from exposure to fish tissue (3×10^{-4}), surface soil (1×10^{-4}), and surface water (1×10^{-3}) also exceeds the acceptable regulatory range, with 2,3,7,8-TCDD Equivalent and PCBs contributing significantly to risk from fish tissue (1×10^{-4} and 1×10^{-4} , respectively), and benzo(a)pyrene contributing significantly to risk from surface water (1×10^{-4}).

For the child recreator, the estimated hazard index of 5×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for nervous system, ocular and other effects exceed 1. The primary contributions to the total hazard index are from exposure to fish tissue and surface sediment (4×10^1 and 4×10^0 , respectively). 2,3,7,8-TCDD Equivalent, mercury (as methylmercury), and PCBs contribute significantly (hazard quotient > 1) to hazards from fish tissue, while chromium contributes to hazard from surface sediment. The total hazard of 3×10^0 from exposure to surface soil also exceeds the regulatory threshold, with 2,3,7,8-TCDD Equivalent contributing significantly.

For the adult recreator (RAGS Table 9.11 RME; Exposure Unit 6), the estimated total cancer risk (2×10^{-3}) and hazard (3×10^1) are lower than that for the child recreator, but still exceed regulatory limits. The primary contribution to the total cancer risk is from exposure to fish tissue (8×10^{-4}), with 2,3,7,8-TCDD Equivalent and PCBs contributing significantly (5×10^{-4} and 3×10^{-4} , respectively). Total cancer risk from exposure to surface sediment (2×10^{-4}) and surface water (5×10^{-4}) also exceeds the acceptable regulatory range, with benzo(a)pyrene contributing significantly to both exposure media (1×10^{-4} and 4×10^{-4} , respectively).

For the adult recreator, the estimated hazard index of 3×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for nervous system, ocular and other effects exceed 1. The primary contribution to the total hazard index is from exposure to fish tissue, with 2,3,7,8-TCDD Equivalent, mercury (as methylmercury), and PCBs contributing significantly (hazard quotient > 1).

For the child recreator in exposure area SYW-12 (RAGS Table 9.10a RME; Exposure Unit 9), the estimated total cancer risk is 4×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface soil (4×10^{-4}), with benzo(a)pyrene contributing significantly (3×10^{-4}). The SYW-12 hazard index of 9×10^{-1} is below the acceptable regulatory threshold of 1.

For the adult recreator in SYW-12 (RAGS Table 9.11a RME; Exposure Unit 9), the total cancer risk (1×10^{-5}) and total hazard index (4×10^{-2}) are within acceptable regulatory limits.

6.1.8. Future – Construction Worker (Cancer & Non-Cancer)

For the construction worker (RAGS Table 9.4 RME; Exposure Unit 1), the estimated total cancer risk is 2×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The greatest contribution to the total cancer risk is from exposure to surface water (8×10^{-5}), with benzo(a)pyrene contributing significantly (6×10^{-5}).

For the construction worker, the estimated hazard index of 3×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nervous system, nasal/respiratory system and other effects exceed 1. The primary contributions to the total hazard index are from exposure to subsurface sediment and subsurface soil (outdoor air) (1×10^1 and 1×10^1 ,

respectively), with 2-methylnaphthalene, dibenzofuran, and naphthalene contributing significantly (hazard quotient > 1) to hazards from sediment and manganese contributing significantly to hazards from soil.

For the construction worker in SYW-12 (RAGS Table 9.4a RME; Exposure Unit 9), the estimated total cancer risk is 2×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to shallow ground water (2×10^{-4}), with benzo(a)pyrene contributing significantly (2×10^{-4}). The SYW-12 hazard index of 2×10^0 for the construction worker exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ other effects exceed 1. The primary contributions to the total hazard index are from exposure to shallow ground water (2×10^0), with chromium contributing significantly (hazard quotient > 1) to hazards from ground water.

6.1.9. Future – Commercial/Industrial Worker (Cancer & Non-Cancer)

For the future commercial/industrial worker (RAGS Table 9.9 RME; Exposure Units 7 & 8), the estimated total cancer risk is 4×10^{-3} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to ground water as potable water (4×10^{-3}), with benzene and benzo(a)pyrene contributing significantly (2×10^{-3} and 1×10^{-3} , respectively). The estimated total cancer risk from exposure to surface soil (2×10^{-4}) also exceeds the acceptable regulatory range, with benzo(a)pyrene contributing significantly (1×10^{-4}).

For the future commercial/industrial worker, the estimated hazard index of 5×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nervous system, lymphocyte, nasal/respiratory system, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to ground water as potable water (6×10^1), with benzene contributing most significantly (3×10^1).

For the future commercial/industrial worker in SYW-12 (RAGS Table 9.9a RME; Exposure Unit 9), the estimated total cancer risk is 6×10^{-5} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The SYW-12 hazard index of 3×10^{-1} for the future commercial/industrial worker is below the acceptable regulatory threshold of 1.

6.1.10. Future – Adult Resident; Child Resident (Cancer & Non-Cancer)

For the child resident (RAGS Table 9.12 RME; Exposure Units 6 & 8), the estimated total cancer risk is 7×10^{-1} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to ground water as potable water (7×10^{-1}), with benzo(a)pyrene and dibenz(a,h)anthracene contributing significantly (5×10^{-1} and 1×10^{-1} , respectively). The estimated total cancer risk from exposure to surface soil (1×10^{-3}) and ground water as shower vapor (9×10^{-3}) also exceeds the acceptable regulatory range, with benzo(a)pyrene contributing significantly to risk from surface soil (7×10^{-4}), and benzene contributing significantly to risk from shower vapor (8×10^{-3}).

For the child resident, the estimated hazard index of 8×10^2 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the liver, kidney, nervous system, lymphocyte, nasal/respiratory system, ocular, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to ground water as shower vapor and potable water (5×10^2 and 2×10^2 , respectively), with benzene contributing most significantly to the

hazards (4×10^2 and 1×10^2 , respectively). The estimated total hazard of 3×10^1 from exposure to surface soil also exceeds the regulatory threshold, with 2,3,7,8-TCDD Equivalent and PCBs contributing significantly (hazard quotient >1).

For the adult resident (RAGS Table 9.13 RME; Exposure Units 6 & 8), the estimated total cancer risk (7×10^{-2}) and hazard (2×10^2) are lower than that for the child resident, but still exceed regulatory limits. The primary contribution to the total cancer risk is from exposure to ground water as potable water (6×10^{-2}), with benzo(a)pyrene contributing significantly (4×10^{-2}). Total cancer risk from exposure to ground water as shower vapor (6×10^{-3}) also exceeds the acceptable regulatory range, with benzene contributing significantly (5×10^{-3}).

For the adult resident, the estimated hazard index of 2×10^2 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the liver, nervous system, lymphocyte, nasal/respiratory, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to ground water as potable water and shower vapor (9×10^1 and 7×10^1 , respectively), with benzene contributing most significantly to both hazards (5×10^1 and 5×10^1 , respectively).

For the child resident in SYW-12 (RAGS Table 9.12a RME; Exposure Unit 9), the estimated total cancer risk is 7×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface soil (7×10^{-4}), with benzo(a)pyrene contributing significantly (5×10^{-4}). The SYW-12 hazard index of 7×10^0 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for ocular and other effects exceed 1. The primary contribution to the total estimated hazard index is from exposure to surface soil (7×10^0), with PCBs contributing most significantly to hazards (4×10^0).

For the adult resident in exposure area SYW-12 (RAGS Table 10.13a RME; Exposure Unit 9), the total cancer risk (5×10^{-5}) and total hazard index (2×10^{-1}) are within acceptable regulatory limits.

6.2. Central Tendency

CT risks and hazards for Site receptors are presented in RAGS Part D Series Tables 7, 9, and 10. The RAGS Table 7 series presents the derivation of risks and hazards for Site receptors by exposure medium. The RAGS Table 9 series summarizes the estimated risks and hazards for a given receptor across all relevant media. The RAGS Table 10 series summarizes the estimated risks and hazards for a given receptor across all relevant media for only those constituents that result in significant risks and/or hazards. In the risk characterization discussion below, the focus is on overall risks and hazards to receptors across all relevant media, and identification of constituents that significantly contribute to those risks and hazards (RAGS Part D Table 9 Series).

6.2.1. Current/Future – Adult Trespasser; Older Child Trespasser (Cancer & Non- Cancer)

For the older child trespasser (RAGS Table 9.1 CT; Exposure Unit 1), the estimated total cancer risk is 2×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface water (2×10^{-4}), with benzo(a)pyrene the largest contributor (1×10^{-4}).

For the older child trespasser, the Site-wide hazard index of 6×10^0 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nervous system, ocular and other effects exceed 1. The primary contribution to the total hazard index is from exposure to fish tissue (5×10^0), with 2,3,7,8-TCDD equivalent, mercury (as methylmercury), and PCBs contribute significantly (hazard quotient > 1).

Risks and hazards for the adult trespasser (RAGS Table 9.2 CT; Exposure Unit 1) are similar to those for the older child trespasser, with the adult trespasser having the same total cancer risk (2×10^{-4}) and slightly higher hazard index (6×10^0). The primary contributions to the total cancer risk are from exposure to surface water (8×10^{-5}) and fish tissue (6×10^{-5}). Benzo(a)pyrene makes the largest contribution (6×10^{-5}) to the surface water risk and 2,3,7,8-TCDD Equivalent and highly chlorinated PCBs contribute significantly (3×10^{-5} and 1×10^{-5} , respectively) to the fish tissue risk.

For the adult trespasser, the estimated hazard index of 6×10^0 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for nervous system, ocular and other effects are greater than 1. Exposure to fish tissue is the primary pathway contributing to the total hazard, with 2,3,7,8-TCDD equivalent, mercury (as methylmercury), and PCBs contribute significantly (hazard quotient > 1).

6.2.2. Current/Future – Utility Worker (Cancer & Non-Cancer)

For the utility worker (RAGS Table 9.3 CT; Exposure Unit 1), the estimated total cancer risk is 2×10^{-5} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The estimated hazard index of 1×10^3 exceeds the acceptable regulatory threshold of 1. The primary contribution to the total hazard index is from exposure to 2,3,7,8-TCDD in surface and subsurface sediments.

For the utility worker in SYW-12 (RAGS Table 9.3a CT; Exposure Unit 9), total cancer risk is 4×10^{-5} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The SYW-12 hazard index of 4×10^2 is below the acceptable regulatory threshold of 1.

6.2.3. Current/Future – Surveillance Worker (Cancer & Non-Cancer)

For the surveillance worker (RAGS Table 9.5 CT; Exposure Unit 2), the estimated total cancer risk is 2×10^{-6} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The estimated hazard index of 1×10^{-1} is below the acceptable regulatory threshold of 1.

6.2.4. Current/Future – Drainage Ditch Worker (Cancer & Non-Cancer)

For the drainage ditch worker (RAGS Table 9.6 CT; Exposure Unit 3), the estimated total cancer risk (2×10^{-7}) and Site-wide hazard index (2×10^{-2}) are within acceptable regulatory limits.

6.2.5. Current/Future – Railroad Worker (Cancer & Non-Cancer)

For the railroad worker (RAGS Table 9.7 CT; Exposure Unit 4), the estimated total cancer risk is 2×10^{-6} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The estimated hazard index of 7×10^2 is below the acceptable regulatory threshold of 1.

For the railroad worker in SYW-12 (RAGS Table 9.7a CT; Exposure Unit 9), the estimated total cancer risk is 9×10^{-6} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The SYW-12 hazard index (1×10^{-1}) is below the acceptable regulatory threshold of 1.

6.2.6. Current/Future – Commercial/Industrial Worker (Cancer & Non-Cancer)

For the current/future commercial/industrial worker (RAGS Table 9.8 CT; Exposure Unit 5), the estimated total cancer risk is 4×10^{-5} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The estimated hazard index of 3×10^{-1} is below the acceptable regulatory threshold of 1.

6.2.7. Current/Future – Adult Recreator; Child Recreator (Cancer & Non-Cancer)

For the child recreator (RAGS Table 9.10 CT; Exposure Unit 6), the estimated total cancer risk is 2×10^{-3} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contributions to the total cancer risk are from exposure to surface water (8×10^{-4}), with the largest contribution from benzo(a)pyrene (6×10^{-4}) and exposure to surface sediment (1×10^{-3}), with benzo(a)pyrene contributing significantly (9×10^{-4}).

For the child recreator, the estimated hazard index of 1×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nervous system, ocular, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to fish tissue (1×10^1), with mercury (as methylmercury) and PCBs contributing significantly (hazard quotient > 1) to hazards from fish tissue.

For the adult recreator (RAGS Table 9.11 CT; Exposure Unit 6), the estimated total cancer risk (2×10^{-4}) and hazard (6×10^0) are lower than that for the child recreator, but still exceed regulatory limits. The primary contribution to the total cancer risk is from exposure to surface water (8×10^{-5}), with benzo(a)pyrene contributing significantly (6×10^{-5}).

For the adult recreator, the estimated hazard index of 6×10^0 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for nervous system, ocular and other effects exceed 1. The primary contribution to the total hazard index is from exposure to fish tissue, with 2,3,7,8-TCDD Equivalent, mercury (as methylmercury), and PCBs contributing significantly (hazard quotient > 1).

For the child recreator in SYW-12 (RAGS Table 9.10a CT; Exposure Unit 9), total cancer risk is 3×10^{-5} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The SYW-12 hazard index (9×10^{-2}) is below the acceptable regulatory threshold of 1.

For the adult recreator in SYW-12 (RAGS Table 9.11a CT; Exposure Unit 9), the total cancer risk (1×10^{-6}), and total hazard index are within acceptable regulatory limits.

6.2.8. Future – Construction Worker (Cancer & Non-Cancer)

For the construction worker (RAGS Table 9.4 CT; Exposure Unit 1), the estimated total cancer risk is 1×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The greatest contribution to the total cancer risk is from exposure to subsurface sediment and surface water (4×10^{-5} and 4×10^{-5} , respectively), with benzo(a)pyrene contributing significantly (2×10^{-5} and 3×10^{-5} , respectively).

For the construction worker, the estimated hazard index of 1×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nervous system, nasal/respiratory, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to subsurface soil in outdoor air and subsurface sediment (3×10^0 and 9×10^0 , respectively), with manganese contributing significantly (hazard quotient > 1) to hazards from soil in

indoor air, and 2-methylnaphthalene, dibenzofuran, naphthalene, contributing significantly to hazards from sediment.

For the construction worker in SYW-12 (RAGS Table 9.4a CT; Exposure Unit 9), total cancer risk is 1×10^{-4} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to shallow ground water (1×10^{-4}), with benzo(a)pyrene the major contributor (8×10^{-5}). The SYW-12 hazard index of 1×10^0 for the construction worker exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ no effects exceed 1. The primary contributions to the total hazard index are from exposure to shallow ground water (8×10^{-1}), with chromium contributing significantly (hazard quotient > 1) to hazards from ground water.

6.2.9. Future – Commercial/Industrial Worker (Cancer & Non-Cancer)

For the future commercial/industrial worker (RAGS Table 9.9 CT; Exposure Units 7 & 8), the estimated total cancer risk is 1×10^{-3} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to ground water as potable water (1×10^{-3}), with benzene and benzo(a)pyrene contributing significantly (7×10^{-4} and 3×10^{-4} , respectively).

For the future commercial/industrial worker, the estimated hazard index of 5×10^1 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the nervous system, lymphocyte, nasal/respiratory system, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to ground water as potable water (5×10^1), with benzene contributing most significantly (2×10^1).

For the future commercial/industrial worker in SYW-12 (RAGS Table 9.9a CT; Exposure Unit 9), the total cancer risk is 8×10^{-6} , which is within the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The SYW-12 hazard index (1×10^{-1}) is below the acceptable regulatory threshold of 1.

6.2.10. Future – Adult Resident; Child Resident (Cancer & Non-Cancer)

For the child resident (RAGS Table 9.12 CT; Exposure Units 6 & 8), the estimated total cancer risk is 1×10^{-1} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to ground water as potable water (1×10^{-1}), with benzo(a)pyrene, benzo(a)anthracene, and dibenzo(a,h)anthracene contributing most significantly (6×10^{-2} , 1×10^{-2} , and 1×10^{-2} , respectively). Total cancer risk from exposure to surface soil (4×10^{-4}) and ground water as shower vapor (3×10^{-3}) also exceeds the acceptable regulatory range, with benzo(a)pyrene contributing significantly to risk from surface soil (3×10^{-4}) and benzene contributing significantly to risk from shower vapor (3×10^{-3}).

For the child resident, the estimated hazard index of 4×10^2 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for the liver, kidney, nervous system, lymphocyte, nasal/respiratory system, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to ground water as shower vapor and potable water (2×10^2 and 2×10^2 , respectively), with benzene contributing most significantly to hazards (1×10^2 and 1×10^2 , respectively).

For the adult resident (RAGS Table 9.13 CT; Exposure Units 6 & 8), the estimated total cancer risk (2×10^{-2}) and hazard (1×10^2) are lower than that for the child resident, but still exceed regulatory limits. The primary contribution to the estimated total cancer risk is from exposure to ground water as potable water (1×10^{-2}), with benzo(a)pyrene contributing most significantly (8×10^{-3}). Total cancer risk from exposure to ground water as shower vapor (2×10^{-3}) also exceeds the acceptable regulatory range, with benzene contributing significantly (2×10^{-3}).

For the adult resident, the estimated hazard index of 1×10^2 exceeds the acceptable regulatory threshold of 1. When segregated by primary target organ, total hazard indices for liver, nervous system, lymphocyte, nasal/respiratory, and other effects exceed 1. The primary contribution to the total hazard index is from exposure to ground water as potable water and shower vapor (9×10^1 and 3×10^1 , respectively), with benzene contributing most significantly to both hazards (4×10^1 and 2×10^1 , respectively).

For the child resident in SYW-12 (RAGS Table 9.12a CT; Exposure Unit 9), total cancer risk is 3×10^{-4} , which exceeds the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} . The primary contribution to the total cancer risk is from exposure to surface soil (3×10^{-4}), with benzo(a)pyrene contributing significantly (2×10^{-4}). The SYW-12 hazard index of 1×10^0 does not exceed the acceptable regulatory threshold of 1.

For the adult resident in exposure area SYW-12 (RAGS Table 9.13a CT; Exposure Unit 9), the total cancer risk (8×10^{-6}) and total hazard index (1×10^{-1}) are within acceptable regulatory limits.

7. Uncertainty Assessment

Estimation of risks to human health that may result from exposure to constituents in the environment is a complex process. Each assumption used in estimating cancer risks and non-cancer hazards, whether regarding the toxicity value for a particular chemical or the value of a parameter in an exposure equation, has a degree of variability and uncertainty associated with it. In each step of the risk assessment process, beginning with the data collection and analysis and continuing through the toxicity assessment, exposure assessment, and risk characterization, conservative assumptions are made that are intended to be protective of human health and to ensure that risks and hazards are not underestimated.

The risk and hazard values generated in this HHRA are not precise, deterministic estimates, but conditional estimates controlled by conservative upper-bound assumptions regarding exposure and toxicity. The calculated risk values provide an upper bound of the potential health risk value, as opposed to a precise, realistic estimate of actual health risks.

Derivation of the risk estimate requires multiplying conservative assumptions, and therefore the numeric effect of the conservatism of the assumptions is multiplied in the process. This is done by convention, consistent with USEPA protocols, with the objective of minimizing the likelihood of underestimating the actual Site risks and hazards. However, this introduces uncertainty into the estimates.

Additional uncertainties can be associated with the major assumptions and scientific judgments made during the evaluation. Assumptions and judgments based on scientific data are necessary in order to define the conceptual boundary of the Site and to facilitate quantitation of receptor pathway scenarios.

The main sources of uncertainty, relative to the assumptions, results, and conclusions of the HHRA are:

- Uncertainty in Site characterization and data quality
- Uncertainty in the selection of the COPCs
- Uncertainty in the Exposure Assessment
- Uncertainty in the Toxicity Assessment
- Uncertainty in the calculation of quantitative risk estimates.

Uncertainties related to these sources and the approaches taken to provide conservative and health protective estimates of Site risks are discussed below.

7.1. Site characterization and Data Quality

Site characterization may be uncertain for a variety of reasons, including:

- Whether sufficient number of samples have been taken to characterize a given area, and whether potential areas of high contamination been sampled
 - For example, in the Lakeshore area, only three surface water samples were collected and maximum detections were all from one sample location (Seep-1; RAGS Table 2.6). Although the sample size is low and only one sample location is representing the

maximum detections, the exposure medium is adequately represented because there are no other surface water locations in the Lakeshore area.

- Whether the data are still relevant – due to either the age of the sample, or changes in site conditions since the samples were collected
 - Analytical data has been collected over significant spatial and temporal scales by multiple investigators. In general, data collected over multiple collection events for the same location have been given equal weight in the HHRA. However, there are cases where more recent sampling events may be preferable to older events. For example, in the Railroad Area, surface sediment data for dioxins was collected for sample location HB-HBSED-04 from depth 0-0.5 ft on May 11, 2001 and June 4, 2003. The 2001 sample contained more non-detect congeners than the 2003 sample, leading to a higher TEQ value (RAGS Table 2.16b) as a result of substitution of non-detects by ½ of the reporting limit. The 2003 data could have been used preferentially over the 2001 sample in order to avoid the bias from non-detect congeners. However, removal of the 2001 data would have resulted in a higher, not lower, overall exposure point concentration, due to a decrease in sample size. Uncertainties for dioxins are further discussed in Section 7.1.1.4.
 - In the case of ground water data from multiple collection events, the multiple data points from the same location aid characterization of the exposure medium by taking into consideration temporal variations.
- Whether the data include results for all contaminants reasonably expected to be present, based both on site history and samples analyzed for a full suite of contaminants
 - Data utilized for this evaluation are the result of the data collection efforts targeted to support the characterization of the Site through the RI/FS process and investigations performed prior to the onset of Site PSA/RI/FS. Section 1.2 describes the development of the site data set.
- The way in which unusual samples/data were addressed.
 - For example, in Harbor Brook sediment, a NAPL sample (HB-T-3-OIL) was collected on February 13, 2001. For dioxin data associated with this sample, many of the congener results were rejected during validation (RAGS Table 2.19b). However, because this sample is not representative of the entire exposure area, the limited dioxin data for this sample location does not prevent adequate characterization of the overall exposure area. Uncertainties for dioxins are further discussed in Section 7.1.1.4.

Data quality can impact the reliability of results and conclusions of human health risk assessments. Most of the available data utilized in this risk assessment were validated and the following actions were taken with respect to assigned validation qualifiers:

- R - The data were determined to be unusable for qualitative and quantitative purposes. Rejected data were not utilized in the risk assessment.
- J - The analyte was positively identified; the associated numerical value was the approximate concentration of the analyte in the sample. The analytical data were not adjusted to compensate for potential high or low bias in the analytical result, due to uncertainty regarding the magnitude of the bias.

B - The analyte was detected in the associated blank, as well as in the sample, at a concentration less than the action limit ($5 \times$ uncommonly-occurring blank contaminant or $10 \times$ commonly-occurring blank contaminant level as appropriate). Consistent with USEPA guidance (USEPA 1989a), it was assumed that "B" qualified data may be attributable to extraneous contamination.

Note on "B" qualifiers: There are two "B" qualifiers associated with the April 1999 HB-Seep-1 sample. This sample was collected prior to the initiation of the PSA and never qualified. The remainder, and majority, of the "B" qualifiers are associated with sediment and surface water samples collected by the NYSDEC in 1996 and 1997. These data were included in the database as provided by the NYSDEC, which has recently indicated these data have been validated and should be treated in a approach similar to "J" qualified data. These data were not utilized in this risk assessment due the lack of depths provided with many of these samples and many of these samples were co-located with more recently collected samples.

Data collected by O'Brien & Gere in 1997 (which comprise a small amount of the overall data) were not validated. As such, there is insufficient documentation to formally assess the accuracy, precision, or representativeness of those data. However, across the various historical investigations no substantial qualitative variations were noted in the analytes and concentrations reported. Therefore, inclusion of historical data results in less uncertainty in the risk estimates as compared with the exclusion of that data.

7.1.1. Chemical Speciation

Several constituents (*e.g.*, mercury and chromium) potentially exist in more than one form at the Site. The quality of the data concerning speciation of these constituents can affect the uncertainty surrounding the results and conclusions presented in the HHRA.

7.1.1.1. Mercury

From a human health perspective, it is the amount of methylmercury, rather than total mercury that is of most interest, since methylmercury is much more readily absorbed into the human bloodstream. The highest concentrations of methyl mercury are found in larger, longer-lived, planktivorous and piscivorous fish species (USEPA 1997c). Larger fish have significant amounts of muscle mass and therefore, an increased storage capacity for methylmercury. Fish muscle tissue stores primarily methylmercury due to its characteristic uptake, its slow depuration or chemical breakdown, and its ability transfer among different tissues (TAMS 2002a). In most cases, size and diet impact methylmercury concentration levels in fish. Speciated data from Onondaga Lake HHRA (NYSDEC 2002) showed a range of methylmercury from 83 to 99 percent with a mean of 97 percent. As a result, it was assumed for the purpose of this health risk assessment that 100% of the "total mercury" in fish tissue was in the methylated form as methylmercury. This assumption provides the best upper bound value and would provide the most conservative estimation of risk.

7.1.1.2. Chromium

As noted in Section 3.3, chromium was not speciated during analysis. Therefore, as a conservative measure, all chromium results were assumed to be hexavalent chromium for both the screening process and in the calculation of risks and hazards. Hexavalent chromium is not the prevalent form of chromium and is expected to be less than 10% of the total chromium reported. This is supported by a study at the nearby Wastebeds 1 through 8 Site where the average $\text{Cr}^{+6}/\text{Cr Total}$ was 0.03 (3%) in surface soils and 0.07 (7%) in subsurface soils in samples where hexavalent chromium was detected.

Hexavalent chromium was detected in only 16 of 57 total samples (28%), 13 of 38 (34%) in surface soil samples and 3 of 13 (23%) subsurface soil samples. Chromium, whether hexavalent or otherwise, was not a risk driver in any scenario (see Section 6). Therefore, conservatively assuming all chromium to be hexavalent did not materially affect the quantitative HHRA.

7.1.1.3. Polychlorinated Biphenyls

As presented in Section 3.1, PCBs were evaluated as “groups” of Aroclors, rather than by individual Aroclors. PCBs were grouped together based on their relative level of chlorination. “Less chlorinated” PCBs (Aroclors 1016, 1221, 1232, and 1242) were combined for analysis and evaluated against the screening and toxicity values for Aroclor 1016. “Highly chlorinated” PCBs (Aroclors 1248, 1254, 1260, and 1268) were combined for analysis and evaluated against the screening values and toxicity values for Aroclor 1254. For screening purposes, “Total PCBs” represented all Aroclors sampled, which were compared to screening values of Aroclor 1254.

7.1.1.4. Dioxin TEQs

At each sample location, PCDD/PCDF congeners were translated into 2,3,7,8-TCDD equivalents using World Health Organization TEFs (Van den Berg *et al.* 2006). Screening and risk evaluations were performed on the derived 2,3,7,8-TCDD Equivalent values. This approach results in higher TEQ values than the approach of using only detected congeners to derive the TEQ

In exposure areas DSA #1, DSA #2, and AOS #1, no subsurface soil (depth > 2 ft) dioxin data were collected. Therefore, evaluation of exposure to dioxins in upper soil (depth 0 to 10 ft) for these exposure areas was based on data from only the surface soil (depth 0 to 2 ft) interval. There is no indication that surface deposition of dioxins would have occurred and subsequently resulted in higher concentrations in this depth interval. Therefore, it is likely that the utilization of data from the 0 to 2 ft depth interval for exposures in the 0 to 10 ft interval has no appreciable impact on the estimated risks and hazards.

Where PCDD/PCDF congeners were non-detect, one-half of the reporting limit was used for deriving TEQ values. In cases where a large proportion of congeners are non-detect and/or where reporting limits for non-detects are elevated, this approach may lead to overestimation of TEQ values. Uncertainties related to reporting limits for dioxin/furan congeners are considered in **Table 7.1** below.

Table 7.1. Derivation of Dioxin TEQs with and without non-detect congeners.

Exposure Area and Medium	Hypothetical Exposure Point Concentration (Detects and Non-Detects)	Units	Hypothetical Exposure Point Concentration (Detects Only)	Units	Hypothetical Exposure Point Concentration Ratio (Detects and Non-Detects / Detects Only)	Method
Lakeshore Area Surface Soil	5.75E-04	mg/kg	1.37E-03	mg/kg	0.4	Use 95% Adjusted Gamma UCL
Lakeshore Area Subsurface Soil	5.75E-04	mg/kg	1.37E-03	mg/kg	0.4	Use 95% Adjusted Gamma UCL
Penn-Can Property Surface Sediment	6.71E-05	mg/kg	6.07E-05	mg/kg	1.1	Use 95% Approximate Gamma UCL

Table 7.1. Derivation of Dioxin TEQs with and without non-detect congeners.

Exposure Area and Medium	Hypothetical Exposure Point Concentration (Detects and Non-Detects)	Units	Hypothetical Exposure Point Concentration (Detects Only)	Units	Hypothetical Exposure Point Concentration Ratio (Detects and Non-Detects / Detects Only)	Method
Railroad Area Surface Sediment	3.40E-05	mg/kg	2.09E-05	mg/kg	1.6	Use 95% Student's-t UCL
Harbor Brook Surface Sediment	4.06E-05	mg/kg	1.96E-05	mg/kg	2.1	Use 95% Chebyshev (Mean, Sd) UCL
Harbor Brook Subsurface Sediment	1.31E-04	mg/kg	2.73E-05	mg/kg	4.8	Use 95% Chebyshev (Mean, Sd) UCL
East Flume Surface Sediment	4.59E-04	mg/kg	2.11E-04	mg/kg	2.2	Use 95% Chebyshev (Mean, Sd) UCL
I-690 Drainage Ditch Surface Sediment	1.98E-05	mg/kg	1.01E-05	mg/kg	2.0	Use 95% Chebyshev (Mean, Sd) UCL
DSA #2 Surface Soil	2.71E-06	mg/kg	1.89E-07	mg/kg	14.3	Mean 1/2 RL and Detects
DSA #2 Subsurface Soil	2.71E-06	mg/kg	1.89E-07	mg/kg	14.3	Mean 1/2 RL and Detects
AOS #1 Surface Soil	1.83E-05	mg/kg	2.10E-05	mg/kg	0.9	Use 95% Approximate Gamma UCL
AOS #1 Subsurface Soil	1.83E-05	mg/kg	2.10E-05	mg/kg	0.9	Use 95% Approximate Gamma UCL
AOS #2 Surface Sediment	6.09E-07	mg/kg	3.84E-07	mg/kg	1.6	Mean 1/2 RL and Detects
SYW-12 Surface Soil	1.62E-04	mg/kg	3.16E-05	mg/kg	5.1	Use 95% Approximate Gamma UCL
SYW-12 Subsurface Soil	1.62E-04	mg/kg	3.16E-05	mg/kg	5.1	Use 95% Approximate Gamma UCL

For each exposure area and medium in which dioxins were detected, TEQs were derived with detected congeners only and with both detected and non-detected congeners using one-half of the reporting limit for non-detected congeners. The 95% upper confidence limit on the mean of these TEQs were then derived using USEPA ProUCL Version 4) to provide hypothetical exposure point concentrations for each exposure area and medium.

With the exception of DSA #2, on average, the ratio of hypothetical exposure point concentrations using both detected and non-detected congeners versus using only detected congeners was approximately 2. In other words, there is approximately a factor of 2 uncertainty associated with congener detection limits. This uncertainty would be propagated into risk estimates for dioxins in this HHRA.

In the case of DSA #2, the larger ratio is due to a relatively large proportion of non-detect congeners in the soil samples combined with a low sample number that constrains which statistics may be used to derive hypothetical exposure point concentrations.

7.1.2. Sample Depth Labeling

An apparent discrepancy between the listed depth and those of other collocated samples was first pointed out by NYSDEC (2008). A subsequent QC check revealed that one ground water sample from location HB-WA-03S taken on 3/8/2007 was incorrectly labeled in the original RI database with start and end depths of 20 ft and 30 ft, respectively, whereas the correct start and end depths were 3 ft and 13 ft, respectively. Incidentally, concentrations of 1,2-dichlorobenzene (7560 ug/l) and 1,4-dichlorobenzene (8700 ug/l) in this sample would represent the maximum detected concentration in the Lakeshore Area. Inclusion of these samples into the shallow ground water data subset yielded revised Exposure Unit 1 exposure point concentrations for 1,2-dichlorobenzene and 1,4-dichlorobenzene of 1451 ug/l and 1291 ug/l, respectively. This is compared to the previously calculated 1156 ug/l and 847.2 ug/l 1,2-dichlorobenzene and 1,4-dichlorobenzene exposure point concentrations.

However, a review of RAGS Table 7.4 (construction worker; the only receptor exposed to shallow ground water in this HHRA) indicates the following:

Compound	Cancer risk	Noncancer hazard
1,2-dichlorobenzene	Not Available	1E-04
1,4-dichlorobenzene	7E-10	1E-04

Based on the above, it is clear that the previously calculated cancer risks and noncancer hazards for these two compounds are 3 and 4 orders of magnitude lower than their respective thresholds (1E-06 and 1, respectively). Therefore, the changes to absolute EPC values for these two compounds do not result in any material change in the risk or hazard profile for the pathways associated with shallow ground water. Updating the RAGS table series to account for this contextually insignificant error would add little value to this HHRA; instead, the above discussion is offered as a substitute.

7.2. Selection of COPCs

Uncertainty in the selection of COPCs may result from the selection of analytical parameters used to evaluate environmental media and the screening of analytes for inclusion in the quantitative evaluation of risk.

7.2.1. Selection of Analytical Parameters

Consistent with guidance for investigations conducted under CERCLA, the selection of analytical parameters were based on Site history and historical operations. Although there is detailed knowledge of historical operations at the Site, full knowledge of constituents that may have been included in the Solvay Waste is unlikely. Most sampling programs, however, utilized broad-spectrum analyses (*e.g.*, Target Compound List or Target Analyte List) to evaluate environmental media. Therefore, the uncertainty in the selection of the appropriate analytical parameters is low.

7.2.2. COPC Screening Process

In this document, a conservative screening process consistent with USEPA guidance (USEPA 1989a) was applied. In that process, the maximum detected concentrations of the detected constituents in

surface soil, combined surface and subsurface soil, surface water, surface sediment, shallow ground water, and all ground water were compared to conservative screening values for the protection of human health.

The screening values utilized were the lowest of the USEPA Region PRGs (USEPA 2004a) or the USEPA Region 3 RBCs (USEPA 2007a). RBC and PRGs for tap water were applied to screen surface water and ground water detected concentrations. RBCs and PRGs for residential soils were applied to screen the soil and sediment detected concentrations. RBCs and PRGs utilized in the screening process corresponded to a cancer risk of 10^{-6} or a hazard quotient of 0.1. Constituents detected in media that did not have established RBC or PRGs were carried forward for further evaluation in the risk assessment. In addition, all detected Group A carcinogens (*e.g.*, arsenic, benzene, chromium) were retained as COPCs even if their maximum detected concentration did not exceed their respective screening criteria. As noted above, unspeciated chromium was evaluated as hexavalent chromium. Because of the extremely conservative approach taken in the screening process, uncertainty related to the development of the COPCs list is relatively low and the likelihood that a constituent that may pose an unacceptable risk to human health has not been evaluated is extremely low.

In addition to the COPC selection process described above, naturally occurring inorganic compounds were eliminated from the COPC list if they were essential nutrients. Based on this consideration, calcium, magnesium, potassium, and sodium were not carried forward as COPCs for the risk assessment. Wet chemistry analytes and geochemical parameters were not included in the risk assessment (*e.g.*, chloride, nitrogen, and total organic carbon). Their constituents are not expected to pose an unacceptable hazard at concentrations measured at the Wastebed B/Harbor Brook site.

Other considerations leading to potential uncertainty in the screening process include screening analytical results for several constituents against benchmarks for surrogate compounds. These include:

- 3&4-Methylphenol was screened against the criteria for 4-methylphenol
- All chlordane constituents were summed and screened against the chlordane RBC and technical chlordane PRG criteria.
- “Less chlorinated” PCBs (Aroclor 1016, 1221, 1232, and 1242) were combined for analysis and evaluated against the screening and toxicity values for Aroclor 1242.
- “Highly chlorinated” PCBs (Aroclor 1248, 1254, 1260, and 1268) were combined for analysis, evaluated against the screening values and toxicity values for Aroclor 1254.

7.3. Uncertainty in the Exposure Assessment

The selected receptors and exposure scenarios presented in this HHRA are based on current and historical observations of activities at the Site and likely potential future uses of the Site. The specific exposure assumptions for a given scenario tend to represent conservative estimates that were approved and agreed upon by USEPA and NYSDEC.

The primary areas of uncertainty affecting the Exposure Assessment for these involve the assumptions affecting exposure pathways, the estimation of exposure point concentrations, and the parameters used to estimate chemical doses. The uncertainties associated with these various sources are discussed below.

7.3.1. Central Tendency and Reasonable Maximum Exposure Scenarios

This risk assessment contains many layers of conservative assumptions. For example, in the RME scenario, the values selected for exposure point concentrations in each equation used to calculate risks to the RME individual are upper-bound estimates. Therefore, the estimated risks for the RME scenarios are likely to be greater than the UCL of all potential risks. If the risk assessment was able to capture the uncertainty and variability associated with each parameter, it is likely that the actual potential risk to the RME individual would be less than the risks estimated in this assessment.

In this HHRA, both CT and RME scenarios were evaluated. As a result, some uncertainty in the evaluation of potential exposures was eliminated. Where published CT or RME parameters were not available, best professional judgment was used, thereby potentially increasing the uncertainty. In some cases, the USEPA recommended RME default values for exposure parameters were used conservatively for CT estimates, adding increased uncertainty. The default or selected exposure coefficients or parameters used in this assessment likely resulted in a moderate overestimation of risk, even in the cases of the reasonably maximally exposed individual.

7.3.2. Drinking Water Exposure Scenario

In accordance with the NYSDEC's request, the hypothetical drinking water scenario was evaluated in the risk assessment. However, Site-related ground water is not used as a drinking or industrial water supply and is highly unlikely to be used as a drinking or industrial supply in the future, since the area is supplied by municipal water from the Village of Solvay. Furthermore, the yield of the overburden ground water unit is inadequate for water supply wells and the high salinity of the deep aquifer (3,000 mg/l chloride) precludes its use as drinking water.

7.3.3. Calculation of Exposure Point Concentration

Uncertainties associated with the development of EPCs are typically related to the quality and quantity of the data available and the protocols used to generate the EPC.

The methodology used to develop the EPCs used in this risk assessment is discussed in detail in Section 4. Statistical and procedural methods were applied to the data in order to develop an estimate of the EPC for COPCs selected for each Exposure Unit on a medium-specific basis. The general approach used the following criteria:

- Where a given data set contained less than three sample points or only one unique detected sample, the maximum value for each analyte in that data set was used as the EPC.
- For data sets with four or more data points, and at least two unique detected samples, statistical methods were applied.

In the latter case, the ProUCL Version 4.0 statistical software package (USEPA 2007b) was used to examine the data distribution and develop an upper confidence level on the arithmetic mean (UCL). ProUCL was run using Regression on Order Statistics (ROS), which is a method for accounting for non-detect samples in the data set. ROS infers values for non-detect samples based on the distribution of detected data, and thus reduces the sensitivity to different reporting limits. ProUCL recommends the most appropriate UCL to use given the distribution type. The UCL recommended by ProUCL was subsequently applied as the EPC.

As noted in Section 4.1.1, in some cases the 95% UCL is less than the reported average concentration. In instances where the detection frequency is low and non-detect samples largely outnumber detected

samples, the 95% UCL recommended by ProUCL Version 4.0 can be smaller than the mean detected concentration, since it reflects the large number of non-detect samples. In these cases, the maximum detected concentration is used as the EPC, citing “Insufficient Data” as the rationale.

7.3.4. Derivation of 95% UCLs – Regression on Order Statistics Versus ½ Detection Limit Substitution

Although ProUCL Version 4.0 includes Regression on Order Statistics (ROS), for those constituents that were found to be associated with the most significant contributions to unacceptable levels of risk, EPA Region 2 recommended also developing EPCs using the approach of substituting non-detects with ½ of the detection limit (DL). For these compounds, a comparison of EPCs calculated using ProUCL Version 4.0 with ROS statistics versus EPCs calculated using simple substitution of non-detect samples by half the reporting limit was conducted. This analysis is presented below by deriving EPCs for benzo(a)pyrene, benzene, and dibenz(a,h)anthracene using both methods.

For several compounds that contribute heavily to total risk, we calculated upper confidence limits on the mean after substituting non-detects with ½ of the detection limit. We compare the resulting EPC with those determined by ProUCL using ROS method. Although some non-detect samples have extremely high detection limits, which bias the UCL, we have not excluded non-detect samples with high detection limits so that the two statistical methods can be compared with the same exact data. Note that sensitivity to high detection limit samples is specific to the ½ DL calculation exercise. The ROS method, used in the quantitative HHRA, is not sensitive to high detection limits and thus high reporting limits were not excluded in the quantitative risk assessment.

Results of the two methods are summarized in **Table 7.2** below. For the four cases shown below the ½ DL substitution yields higher exposure point concentrations than the ROS method. It should be noted; however, the ½ DL substitution method alters the distribution of the data, which causes ProUCL to choose a different statistical test than was chosen in the ROS case.

Exposure Unit	Medium	Constituent	Detection Frequency	ROS EPC	ROS Statistic	½ DL EPC	½ DL Statistic	Ratio of ½ DL EPC to ROS EPC
6	Sediment	benzo(a) pyrene (mg/kg)	38/40	106.3	97.5% KM (Chebyshev) UCL	155.2	99% Chebyshev (Mean, SD) UCL	1.5
8	Ground Water	Benzene (µg/L)	93/178	5831	97.5% KM (Chebyshev) UCL	8487	99% Chebyshev (Mean, SD) UCL	1.5
8	Ground Water	benzo(a) pyrene (µg/L)	17/167	19.97	95% KM (Chebyshev) UCL	87.6	97.5% Chebyshev (Mean, SD) UCL	4.4
8	Ground Water	dibenz(a,h) anthracene (µg/L)	4/167	2.841	95% KM (t) UCL	88.58	95% Student's-t UCL	31.2

Based on these example cases, the EPC derived from the ½ DL substitution method is about 50% higher than the ROS EPC when the detection frequency is at least 50%. For data with lower detection

frequency, the EPC derived from the $\frac{1}{2}$ DL substitution method can be over an order of magnitude higher than the ROS EPC. This difference would be propagated into the risks derived in this HHRA if the $\frac{1}{2}$ DL substitution method were used.

7.3.5. Fish Tissue Concentrations

To evaluate the Recreator exposure scenario, a quantitative assessment was conducted by utilizing fish tissue exposure point concentrations derived in the *Onondaga Lake Human Health Risk Assessment* (NYSDEC 2002). Although the types and levels of constituents do not necessarily reflect impacts directly from this Site, because most fish do move throughout the lake, they do represent the possible constituents and levels that a fishing recreator may encounter. The uncertainties related to the concentrations found in fish fillets can be found in human health risk assessment for Onondaga Lake (NYSDEC 2002, page 7-2). However, the use of Onondaga Lake data is considered to result in less uncertainty than the use of modeled fish tissue constituent concentrations derived from Harbor Brook sediment data.

7.3.6. Particulate Emissions and Volatilization Estimates

The inhalation of air particulates and volatile compounds generated from Site soils (and I-690 drainage ditch sediment for volatile emissions) was evaluated in the Wastebed B/Harbor Brook HHRA. Because the USEPA Region 9 PRG (USEPA 2004a) criteria utilized in the screening process are protective of multi-pathway exposure to soil, uncertainty surrounding the potential effects related to those constituents that were not carried forward is greatly reduced.

The calculation of the Particulate Emission Factor (PEF) and the Volatilization Factor (VF) are discussed in Section 4.1.5. Of those soil constituents that were retained, volatile organic compounds were evaluated using the soil-to-air volatilization factor (see **Appendix E**). Other types of constituents (metals, PCBs, pesticides, SVOCs, dioxins, and others) were evaluated as particulate emissions (see **Appendix F**). Because the calculation of estimated air concentrations may be affected by a variety of factors including temperature, wind speed, vegetative cover, *etc.*, the concentrations used in this HHRA do not represent precise estimates.

7.3.7. Uncertainties in the Soil/Sediment Dermal Exposure Pathway Assumptions

Soil/Sediment-to-Skin Adherence Factors (AF) and Dermal Absorption Factors (ABS) recommended by the NYSDEC were applied in the exposure assessment. In addition, route-to-route extrapolation factors were applied in the estimation of absorbed dose for each receptor.

7.3.7.1. Soil/Sediment-to-Skin Adherence Factors

The soil/sediment to skin adherence factors (AF) represent the average mass of soil that adheres to the skin over each exposure event. The AF depends on the specific activity being conducted and is typically higher for body parts with greater exposure to the soils or sediments. The specific RME and CT AFs used in this HHRA were obtained from USEPA Risk Assessment Guidance (RAGS Part E, USEPA 2004b, Exhibit 3-3) and the rationale for the various AFs used in this document are discussed in Section 4.3.3. Although this guidance provides recommended AFs for various activities and receptor categories, there is a wide range of AFs that can be found in other guidance documents and published literature. As such, the actual AFs for any given activity for a receptor at the Site cannot be determined precisely. The AFs chosen in this document, however, tend to represent conservative values chosen in this document tend to represent conservative values that will likely overestimate the amount of soil or sediment that adheres to the skin of a receptors. Consequently, risks and hazards associated with dermal exposure for soil will likely be overestimated.

7.3.7.2. Dermal Absorption Factors

The dermal absorption factor (ABS) represents the fraction of the soil constituent that may be absorbed through the skin over each exposure event. In general, metals are poorly absorbed through the skin whereas organic constituents may be absorbed more easily. As discussed in Section 4.3.2, constituent-specific values were obtained from USEPA Risk Assessment Guidance (RAGS Part E, USEPA 2004b, Exhibit 3-4). If chemical specific data for dermal absorption was not available, 100% dermal absorption was assumed. In the latter case, it is highly likely that dermal exposure to COPCs is overestimated.

7.3.7.3. Route-to-Route Extrapolation

Most toxicity values are based on studies related to either exposure via inhalation or, usually, ingestion rather than on dermal studies. Consequently, an extrapolation from one of these exposure routes to the absorbed dermal dose must be used to determine the appropriate reference dose for dermal exposure. In this HHRA, oral absorption efficiencies used in the route-to-route extrapolations were from obtained from Exhibit 4-1 of USEPA (2004b) RAGS Part E. The process for selection of the oral absorption efficiencies is as follows:

- For oral absorption efficiency for dermal greater than 50%, no adjustments were made for the dermal route.
- For constituents with a range of oral absorption efficiencies for dermal in Exhibit 4-1, the highest value is reported
- For constituents not listed in Exhibit 4-1, an absorption efficiency of 1 (100%) was assumed.

Inherent in this process is the introduction of uncertainty surrounding the absorbed RfD for dermal and absorbed cancer slope factor for dermal presented in Tables 5.1 and 6.1; however, the impact of the uncertainty is difficult to estimate.

7.3.7.4. Skin Surface Area Available for Dermal Contact

Skin surface area for dermal contact and absorption (SA) from water and soil were derived from a variety of sources and, in some cases, were made using on best professional judgment based on Site-specific knowledge. In most cases, the RME and CT values for SA were identical, however, in one case, the SA for the surveillance worker was reduced from and RME value of 2480 cm² to a CT value of 1930 cm²/day. This was based on the Site-specific knowledge that only the head and hands are exposed because a worker typically wears long sleeves for much of the year. For other scenarios evaluated, the chosen values are generally equal to or greater than those recommended in USEPA RAGS Part E (2004).

7.3.8. Uncertainties Associated with the Ingestion Pathway

Uncertainties associated with the ingestion pathway for soil, sediment and surface water are evaluated below.

7.3.8.1. Incidental Soil and Sediment Ingestion Rates

Ingestion rates for soil and sediment used in this HHRA represent the amount of these media that are ingested as a result of activities associated with each receptor. Typically, receptors with greater contact with soil or sediment (*e.g.*, construction worker) have a greater rate of incidental ingestion than those whose activities result in less contact with soil or sediment (*e.g.*, an office or factory worker).

A soil incidental ingestion rate of 330 mg/day (RME) was applied for the construction, utility worker, and drainage ditch worker; however, this value may overestimate potential soil exposures for these receptors. Other assessments have indicated that default incidental ingestion rates in the range of 100 to 200 mg/day are appropriate for high soil contact activities. Draft NYSDEC guidance for the evaluation of petroleum release sites (NYSDEC 1997) apply a default construction worker soil incidental ingestion rate of 82 mg/day, whereas the USEPA default rates for evaluation of agricultural scenarios is 100 mg/day (reviewed in USEPA 1997a). Sheppard (1995) (in USEPA 1997a, Table 4-15) estimated an incidental ingestion rate of 20 mg/hr for gardening activities. Based on this estimate, the total soil ingested over five to eight hours would be 100 to 160 mg/day. The CT evaluation (using 100 mg/day for the utility worker; and 330 mg/day for the construction & drainage ditch workers) provides an indication of the impact that the uncertainty surrounding this value has on the estimated risks and hazards for these receptors.

An incidental soil ingestion RME value of 200 mg/day for a younger child recreator or resident is applied in the RME scenario following USEPA and NYSDEC's recommendation. This value is acknowledged as being a conservative estimate of the mean (*Exposure Factors Handbook*, USEPA 1997a, Table 4.23) and likely overestimates ingestion for these receptors. A value of 100 mg/day is typically used in the RME for this type of scenario. Consequently, the rate of incidental soil ingestion for this receptor likely leads to an overestimation of increased cancer risk and hazards.

7.3.8.2. Water Ingestion Rates

Incidental ingestion of surface water was not evaluated in this HHRA because such ingestion by the chosen receptors is considered *de minimis*. This assumption may lead to an underestimate of risks and hazards, however, it is not expected to have an appreciable impact on the results.

Although Site-wide ground water is not considered potable water, a hypothetical drinking water scenario was evaluated in the risk assessment. For this scenario, water intake is assumed to be 2 L/day for adult residents and 1 L/day for younger child residents, consistent with USEPA guidance (RAGS Part A, USEPA 1989a, Exhibit 6-11). The adult water ingestion rate is based on lognormal distribution with an arithmetic mean of 1.26 and a standard deviation of 0.66 L/day.

7.3.9. Uncertainties in the Exposure Frequencies

Although the exposure frequencies used in evaluating human exposure in this risk assessment are generally conservative, it is possible that some receptors could be exposed at a greater frequency than that evaluated here. For instance, a trespasser was evaluated based on an exposure frequency of 42 days/year. It is possible that a homeless trespasser may be resident on-site more than 42 days/year, particularly due to time spent on-site during the warmer months of the year. As such, the risk and hazard estimates in this HHRA may underestimate this exposure.

7.4. Uncertainties in Toxicity Values

Toxicity information for many chemicals is often limited. Consequently, there are varying degrees of uncertainty associated with toxicity values (*i.e.*, cancer slope factors, reference doses). For example, uncertainties can arise from the following sources:

- *Extrapolation from Animal Studies to Humans* - Toxicity results are often derived from studies in animals, and there are substantial uncertainties in the inter-species extrapolation of animal results to humans due to differences in toxicokinetics and toxicodynamics. In general, EPA deals with this uncertainty by application of an uncertainty factor of 10. That is, in cases where humans are either equally sensitive or less sensitive than animals, the toxicity factors will substantially overestimate risk.
- *Extrapolation from High Dose to Low Dose* - Most animal studies are performed using relatively high exposure levels, and there is often uncertainty in the best way to extrapolate the dose-response curve to the lower exposure levels typically experienced by humans at a Superfund site. In general, EPA deals with this issue by assuming a conservative dose response model, and by using a conservative estimate of the LOAEL and NOAEL.
- *Extrapolation from Continuous Exposure to Intermittent Exposure* - Most animal studies are performed using a relatively constant exposure design, while most human exposures occur intermittently (especially for recreational visitors). Current risk assessment methods assume that risk is proportional to average dose rather than dose rate, and this could result in either an overestimate or an underestimate of true risk.
- *Lack of Adequate Test Results* - In some cases, only a few studies are available to characterize the toxicity of a chemical, and uncertainties exist not only in the dose response curve, but also in the nature and severity of the adverse effects which the chemical may cause. The USEPA typically deals with this uncertainty by applying an uncertainty factor of 10 to 100 to account for limitations in the database. Thus, in cases where available data do identify the most sensitive endpoint of toxicity, risk estimates will substantially overestimate true hazard.
- *Potentially Sensitive Human Subpopulations* - In general, it is assumed that some humans may be more sensitive than others to the adverse effects of a chemical, but data are usually not available to determine if this is true. EPA typically deals with this uncertainty by applying an uncertainty factor of 10. Thus, most people are expected to have a risk 10 times lower than calculated, and even if some people do tend to be most sensitive, the calculated risks may still be larger than actual.

In general, uncertainty in toxicity factors is one of the largest sources of uncertainty in the development of estimates of risks and hazards at a site. Because of the conservative methods that are used in dealing with the uncertainties, it is much more likely that the uncertainty will result in an overestimation rather than an underestimation of risk. Uncertainty in toxicity factors also arises from lack of knowledge on the potential interactive effects of different chemicals. Most RfD and slope factor values are derived from studies of the adverse effects of pure chemicals. However, human exposure scenarios usually involve multiple chemicals, raising the possibility that synergistic or antagonistic interactions might occur. However, data are not adequate to permit any quantitative adjustment in toxicity values or risk calculations based on inter-chemical interactions. This uncertainty may result in overestimates or underestimates of risk.

- *Lack of Quantitative Toxicity Values for Detected Chemicals* - For constituents of potential concern without quantitative toxicity values, risks/hazards could not be estimated, resulting in the potential under-estimation of risks and hazards.

- **TCE Cancer Slope Factor** - An inhalation cancer slope factor for trichloroethene (TCE) of 0.4 mg/kg-day (USEPA 2001) was utilized in the risk assessment. This is a conservative draft provisional toxicity value adopted by USEPA. For reference, the prior inhalation cancer slope factor for TCE from other sources (USEPA 1995; CalEPA) range from 7.0×10^{-3} to 4.0×10^{-1} (mg/kg-d)⁻¹. Therefore, cancer risks from inhalation of TCE may be overestimated.

7.5. Spatial Hot Spots

As with many affected sites, the spatial distribution of constituents in environmental media can be significantly heterogeneous with localized areas of elevated concentrations. To determine whether a particular area is a spatial hot spot, the table below presents the percentage of constituents screened in as COPC for each exposure area and the mean of all exposure areas for a given exposure medium and chemical type. For this study, an exposure area can be considered a hot spot if the COPC percentage of constituents is more than one standard deviation greater than the mean of all exposure areas for a particular exposure medium. Percentages that exceed these criteria are shown in bold in **Table 7.3** below. Thus for shallow ground water, SYW-12 is a hot spot for metals, the Lakeshore Area is a hotspot for SVOCs and VOCs, and DSA #2 is a hotspot for VOCs. For sub-surface sediment, Harbor Brook is a hot spot for VOCs, and the Penn-Can Property is a hotspot for SVOCs. East Flume is a hot spots for pesticides in surface sediment, and Harbor Brook is also a hot spot for surface sediment metals and VOCs. In surface soil, Lakeshore Area, Railroad Area, AOS #2, and DSA #1 are hotspots for metals, pesticides, SVOCs, and VOCs, respectively. The I-690 drainage ditch is a hot spot for both metals and VOCs in surface water, and the Lakeshore Area is a hot spot for SVOCs in surface water.

Table 7.3. Spatial Distribution Summary.

		Percentage of Constituents Screened in as COPC by Exposure Area					
Exposure Area	Exposure Medium	Dioxins %	Metals %	PCBs %	Pesticides %	SVOC %	VOC %
AOS #1	Shallow Ground Water	-	38	-	0	40	15
DSA #2	Shallow Ground Water	-	28	-	0	38	65
Lakeshore Area	Shallow Ground Water	-	52	100	100	87	67
Penn-Can Property	Shallow Ground Water	-	48	100	-	67	25
Railroad Area	Shallow Ground Water	-	44	-	-	50	20
SYW-12	Shallow Ground Water	-	67	-	0	67	19
<i>Exposure medium mean</i>		-	<i>46</i>	<i>100</i>	<i>25</i>	<i>58</i>	<i>35</i>
<i>Exposure medium mean + standard deviation</i>		-	<i>59</i>	<i>100</i>	<i>75</i>	<i>77</i>	<i>59</i>
AOS #1	Sub-Surface Soil	100	38	100	17	56	20
DSA #1	Sub-Surface Soil	100	35	100	-	50	53
DSA #2	Sub-Surface Soil	0	33	100	0	70	50
Harbor Brook	Sub-Surface Sediment	100	42	67	13	77	65
Lakeshore Area	Sub-Surface Soil	100	52	100	14	57	32
Penn-Can Property	Sub-Surface Soil	-	38	100	0	88	14
Railroad Area	Sub-Surface Soil	-	39	67	20	35	14
SYW-12	Sub-Surface Soil	100	42	67	0	41	5
<i>Exposure medium mean</i>		<i>83</i>	<i>40</i>	<i>88</i>	<i>9</i>	<i>59</i>	<i>32</i>
<i>Exposure medium mean + standard deviation</i>		<i>124</i>	<i>46</i>	<i>105</i>	<i>18</i>	<i>77</i>	<i>54</i>

Table 7.3. Spatial Distribution Summary.

		Percentage of Constituents Screened in as COPC by Exposure Area					
Exposure Area	Exposure Medium	Dioxins	Metals	PCBs	Pesticides	SVOC	VOC
		%	%	%	%	%	%
AOS #2	Surface Sediment	0	31	-	-	47	25
East Flume	Surface Sediment	100	29	100	33	58	28
Harbor Brook	Surface Sediment	100	33	67	7	54	50
I-690 Ditch	Surface Sediment	100	27	100	0	42	14
Penn-Can Property	Surface Sediment	100	32	100	0	45	17
Railroad Area	Surface Sediment	100	26	100	0	41	0
<i>Exposure medium mean</i>		83	30	93	8	48	22
<i>Exposure medium mean + standard deviation</i>		124	32	108	22	55	39
AOS #1	Surface Soil	100	43	100	17	55	19
AOS #2	Surface Soil	-	31	-	-	57	0
DSA #1	Surface Soil	100	32	100	-	50	38
DSA #2	Surface Soil	0	30	100	0	47	27
Lakeshore Area	Surface Soil	100	52	100	17	38	24
Penn-Can Property	Surface Soil	-	38	100	0	54	14
Railroad Area	Surface Soil	-	41	67	25	39	22
SYW-12	Surface Soil	100	38	100	0	38	6
<i>Exposure medium mean</i>		80	38	95	10	47	19
<i>Exposure medium mean + standard deviation</i>		125	45	108	21	55	31
East Flume	Surface Water	-	0	-	-	-	0
Harbor Brook	Surface Water	-	21	-	-	47	11
I-690 Ditch	Surface Water	-	39	-	0	57	56
Lakeshore Area	Surface Water	-	17	-	-	79	44
Penn-Can Property	Surface Water	-	21	-	-	50	25
Railroad Area	Surface Water	-	28	-	-	75	25
<i>Exposure medium mean</i>		-	21	-	0	62	27
<i>Exposure medium mean + standard deviation</i>		-	34	-	-	76	47
AOS #1	Vapor Intrusion	-	-	-	-	73	31
DSA #2	Vapor Intrusion	-	-	-	-	69	52
SYW-12	Vapor Intrusion	-	-	-	-	73	29
<i>Exposure medium mean</i>		-	-	-	-	72	37
<i>Exposure medium mean + standard deviation</i>		-	-	-	-	74	50

In addition to exposure areas representing hot spots, there is the potential for hot spots to exist within a specific exposure area. For example, in Harbor Brook sediment, a NAPL sample (HB-T-3-OIL) was collected on February 13, 2001. Because this sample is not representative of the entire exposure area, there is a potential for underestimation of risks from the primary components of the NAPLs (e.g., BTEX, chlorinated benzenes, naphthalene) for this localized area.

7.6. Risk-Based Hot Spots

7.6.1. Site-Wide Cancer Risk

As shown in **Table 7.4** below, Site-wide cancer risks for the majority of Site receptors are driven by exposure to benzo(a)pyrene in Site exposure media (surface sediment, surface water, surface soil, ground water as potable water). In addition, Site-wide cancer risks for the adult trespasser and recreator are driven by exposure to dioxins (evaluated as 2,3,7,8-TCDD Equivalent) and PCBs in fish tissue. In sum, with regard to a risk-based definition of hot spots, benzo(a)pyrene in the above Site media and dioxins and PCBs in fish tissue represent hot spots. Addressing these constituents would reduce the bulk of Site cancer risks towards acceptable regulatory ranges.

With regard to constituents in fish tissue, because fish tissue exposure point concentrations were obtained from the Onondaga Lake HHRA and not from Harbor Brook itself, there is some uncertainty in the risk estimates for exposure to fish tissue, particularly with respect to risks strictly posed by Site-related constituents.

Table 7.4. Summary of Risk Drivers for Site-Wide Cancer Risk.			
Timeframe	Receptor	Primary Exposure Medium	Primary Constituents
Current/Future	Older child Trespasser	Surface Sediment	Benzo(a)pyrene, Benzo(a)anthracene
Current/Future	Adult Trespasser	Fish Tissue	2,3,7,8-TCDD Equivalent and PCBs
Current/Future	Utility Worker	Surface Water	Benzo(a)pyrene
Current/Future	Surveillance Worker	N/Ap	N/Ap
Current/Future	Drainage Ditch Worker	N/Ap	N/Ap
Current/Future	Railroad Worker	N/Ap	N/Ap
Current/Future	Commercial/Industrial Worker	Surface Soil	Benzo(a)pyrene
Future	Child Recreator	Surface Sediment	Benzo(a)pyrene, Benzo(b)fluoranthene
Future	Adult Recreator	Fish Tissue	2,3,7,8-TCDD Equivalent and PCBs
Future	Construction Worker	Surface Water	Benzo(a)pyrene
Future	Commercial/Industrial Worker	Ground Water as Potable Water	Benzo(a)pyrene, Benzene
Future	Child Resident	Ground Water as Potable Water	Benzo(a)pyrene, Dibenzo(a,h)anthracene
Future	Adult Resident	Ground Water as Potable Water	Benzo(a)pyrene
Notes: Site-Wide – Includes all Site exposure areas except SYW-12. N/Ap – Not applicable (acceptable risk or hazard). Primary Exposure Medium – Exposure medium responsible for majority of receptor risk or hazard. Primary Constituents – Constituents responsible for majority of receptor risk or hazard.			

7.6.2. Site-Wide Non-Cancer Hazards

Although total Site-wide non-cancer hazards for a number of Site receptors are within the acceptable regulatory threshold of 1 (**Table 7.5** below), Site-wide hazards for the remaining receptors are driven by:

- 2,3,7,8-TCDD Equivalent, thallium, and PCBs in fish tissue
- 2-Methylnaphthalene, dibenzofuran, naphthalene, and phenanthrene in sediment
- benzene in ground water

Because these constituents are responsible for the majority of Site non-cancer hazards, they represent risk-based hot spots. Addressing these constituents would considerably reduce Site non-cancer hazards towards acceptable regulatory limits.

However, as with the discussion of cancer risks above, because fish tissue exposure point concentrations were obtained from the Onondaga Lake HHRA and not from Harbor Brook itself, there is some uncertainty in the risk estimates for exposure to fish tissue. In addition, the future use of ground water as potable water and for showering represents a highly unlikely exposure scenario, given that municipal water supply is available and in use by current Site workers, and any future workers or residents would likely use municipal water.

Table 7.5. Summary of Drivers for Site-Wide Non-Cancer Hazards.			
Timeframe	Receptor	Primary Exposure Medium	Primary Constituents
Current/ Future	Older child Trespasser	Fish tissue	2,3,7,8-TCDD Equivalent, Mercury (as methyl mercury), and PCBs
Current/ Future	Adult Trespasser	Fish Tissue	2,3,7,8-TCDD Equivalent, Mercury (as methyl mercury), and PCBs
Current/ Future	Utility Worker	Surface and Subsurface Sediment	2,3,7,8-TCDD Equivalent
Current/ Future	Surveillance Worker	N/Ap	N/Ap
Current/ Future	Drainage Ditch Worker	N/Ap	N/Ap
Current/ Future	Railroad Worker	N/Ap	N/Ap
Current/ Future	Commercial/Industrial Worker	N/Ap	N/Ap
Future	Child Recreator	Fish Tissue	2,3,7,8-TCDD Equivalent, Mercury (as methyl mercury), and PCBs
Future	Adult Recreator	Fish Tissue	2,3,7,8-TCDD Equivalent, Mercury (as methyl mercury), and PCBs
Future	Construction Worker	Surface and Subsurface Sediment	2-Methylnaphthalene, Dibenzofuran, and Naphthalene
Future	Commercial/Industrial Worker	Ground Water as Potable Water	Benzene
Future	Child Resident	Ground Water as Shower Vapor	Benzene
Future	Adult Resident	Ground Water as Potable Water	Benzene
Notes: <u>Site-Wide</u> – Includes all Site exposure areas except SYW-12. <u>N/Ap</u> – Not applicable (acceptable risk or hazard). <u>Primary Exposure Medium</u> – Exposure medium responsible for majority of receptor risk or hazard. <u>Primary Constituents</u> – Constituents responsible for majority of receptor risk or hazard.			

7.6.3. SYW-12 Cancer Risks and Non-Cancer Hazards

With regard to receptor risks and hazards in exposure area SYW-12, cancer risks that exceed the acceptable regulatory range of 1×10^{-4} to 1×10^{-6} are driven by the following:

- Benzo(a)pyrene in shallow ground water for the utility worker
- Benzo(a)pyrene in surface soil for the child recreator
- Benzo(a)pyrene in shallow ground water for the construction worker
- Benzo(a)pyrene in surface soil for the child resident

In sum, benzo(a)pyrene in the above exposure media represent risk-based hot spots. Addressing benzo(a)pyrene in these media would substantially reduce receptor cancer risks in the SYW-12 exposure area.

With regard to receptor non-cancer hazards in exposure area SYW-12, only hazards for the child resident exceed the regulatory threshold of 1. For this receptor, hazards are driven by exposure to PCBs in surface soil, which represent a risk-based hot spot. Addressing PCBs in surface soil would substantially reduce non-cancer hazards for the child resident in the SYW-12 exposure area.

7.7. Central Tendency Risks and Hazards

There are several receptors and exposure scenarios that indicate unacceptable RME cancer risks but acceptable CT cancer risks. These receptors are the Wastebed B/Harbor Brook current/future utility worker, current/future utility worker at SYW-12, the current/future commercial/industrial worker, the child recreator at SYW-12, and the Wastebed B/Harbor Brook future construction worker and the future construction worker at SYW-12. The RME cancer risks for these receptors are 4×10^{-4} , 4×10^{-4} , 3×10^{-4} , 4×10^{-4} , 2×10^{-4} , and 2×10^{-4} , respectively. These values are all within an order of magnitude of the acceptable range, and drop to within the acceptable range in the central tendency scenario. In contrast, receptors whose RME risk greatly exceeds the acceptable range also have CT risks above the acceptable range. Similarly, the child resident in SYW-12 has an unacceptable RME hazard quotient (7) that drops into the acceptable range in the CT scenario. **Table 7.6** provides an overview of the risks and hazards for both the RME and CT scenarios.

Table 7.6. Summary of Risks and Hazards for RME and CT Scenarios.

Timeframe	Receptor	Acceptable RME Cancer Risk?	Acceptable CT Cancer Risk?	Acceptable RME Non-Cancer Hazard?	Acceptable CT Non-Cancer Hazard?
Current/Future	Older child Trespasser	no	no	no	no
Current/Future	Adult Trespasser	no	no	no	no
Current/Future	Utility Worker	no	yes	no	no
Current/Future	Utility worker SYW-12	no	yes	yes	yes
Current/Future	Surveillance Worker	yes	yes	yes	yes
Current/Future	Drainage Ditch Worker	yes	yes	yes	yes
Current/Future	Railroad Worker	yes	yes	yes	yes
Current/Future	Railroad Worker SYW-12	yes	yes	yes	yes
Current/Future	Commercial/Industrial Worker	no	yes	yes	yes
Future	Child Recreator	no	no	no	no
Future	Child Recreator SYW-12	no	yes	yes	yes

Table 7.6. Summary of Risks and Hazards for RME and CT Scenarios.

Timeframe	Receptor	Acceptable RME Cancer Risk?	Acceptable CT Cancer Risk?	Acceptable RME Non-Cancer Hazard?	Acceptable CT Non-Cancer Hazard?
Future	Adult Recreator	no	no	no	no
Future	Adult Recreator SYW-12	yes	yes	yes	yes
Future	Construction Worker	no	yes	no	no
Future	Construction Worker SYW-12	no	yes	no	yes
Future	Commercial/Industrial Worker	no	no	no	no
Future	Commercial/Industrial Worker SYW-12	yes	yes	yes	yes
Future	Child Resident	no	no	no	no
Future	Child Resident SYW-12	no	no	no	yes
Future	Adult Resident	no	no	no	no
Future	Adult Resident SYW-12	yes	yes	yes	yes

The following paragraphs provide a discussion of factors affecting cancer risks and hazards deemed unacceptable under the RME scenario but acceptable under the CT scenario.

7.7.1. Current/Future Utility Worker at Wastebed B/Harbor Brook and SYW-12

Cancer risk between the RME and CT scenario differed by two orders of magnitude. Although most exposure parameters remained constant between the RME and CT scenarios, some did vary significantly resulting in different risk estimates. Ingestion rates of soil differed between the two scenarios with 330 mg/day used for the RME and 100 mg/day used for the CT. The sediment to skin adherence factor was 0.9 for RME and 0.2 for CT. Exposure frequency and duration also differed between the RME and CT scenarios with 20 days over 25 years for the RME and 5 days over 9 years for the CT for exposure frequency and duration, respectively.

7.7.2. Current/Future Commercial/Industrial Worker

Risk estimates for the commercial industrial worker were 3×10^{-4} under the RME scenario and 4×10^{-5} under the CT scenario. Differences in exposure parameters occurred primarily in the ingestion and dermal pathways. The RME soil ingestion rate was 100 mg/day whereas the CT was 50 mg/day. The RME soil to skin adherence factor was 0.3 mg/cm^3 and the CT was 0.1 mg/cm^3 . The exposure frequency and duration also differed between the RME (250 days per year for 25 years) and CT scenarios (219 days per year for 9 years).

7.7.3. Future Child Recreator at SYW-12

The RME and CT scenarios for cancer risk for the child recreator at SYW-12 differed by less than one order of magnitude (4×10^{-4} and 9×10^{-3} , respectively). However, this difference resulted in unacceptable cancer risk in the RME scenario, but acceptable risk in the CT scenario. The largest difference in risk resulted from exposure to SYW-12 soils. This is a result of differences in exposure parameters related to soil ingestion and dermal contact. The ingestion rate of soils for the RME scenario was 200 mg/day, whereas it was 100 mg/day the CT scenario. Also, the fraction ingested from soil was altered from 1.0 for the RME scenario to 0.5 for the CT scenario. The largest difference

in exposure parameters, however, was the soil to skin adherence factor (3 mg/cm^3 for the RME versus 0.2 mg/cm^3 for the CT). Other parameters that differed were the exposure frequency, exposure time, and event duration. All of these parameters contributed to the differing cancer risk estimates for the two scenarios.

7.7.4. Future Construction Worker at Wastebed B/Harbor Brook and SYW-12

The estimated cancer risk to the construction worker at the Wastebed B/Harbor Brook Site was found to be unacceptable under the RME scenario but not the CT scenario. Likewise, the construction worker at SYW-12 demonstrated unacceptable cancer risks and non-cancer hazards under the RME scenario, but not under the CT scenario. Exposure parameters that differed between the RME and CT scenario for the construction worker included the inhalation rate (RME $3.2 \text{ m}^3/\text{hr}$ versus CT $1.6 \text{ m}^3/\text{hr}$), the soil to skin adherence factor (RME 0.3 mg/cm^3 versus CT 0.1 mg/cm^3), and the exposure frequency (RME 250 days/year versus CT 125 days/year). These differences were significant enough to result in an unacceptable risk in the RME scenario and acceptable risk in the CT scenario.

7.7.5. Future Child Resident at SYW-12

The non-cancer hazard estimates for child residents at SYW-12 were greater than unity under the RME scenario and were acceptable (equal to or less than unity) under the CT scenario. Differences in exposure parameters included soil ingestion rate (200 mg/day in the RME scenario and 100 mg/day in the CT scenario); soil to skin adherence factor (0.2 mg/cm^3 in the RME scenario and 0.04 mg/cm^3 in the CT scenario); and the event duration (1 hr/event in the RME scenario and 0.33 hr/event in the CT scenario). These differences resulted in the non-cancer hazard dropping from 7.0 under the RME scenario to 1.0 under the CT scenario.

7.8. Future Exposure Scenarios

Although the HHRA accounts for potential future exposure scenarios, there may be some potential future exposure scenarios that are not complete, but may become relevant. For example, there is a bike trail proposed for an adjacent Site. If the trail were to be extended to the Wastebed B/Harbor Brook Site, it would introduce a new future recreational exposure scenario. The HHRA also includes future child and adult residents for Exposure Units 6 and 8, even though residential use is not anticipated because the Site is zoned as industrial.

7.9. Uncertainty Due to Combination of Conservative Assumptions and Estimates

A consequence of adding risk estimates across chemicals and across pathways is that any conservatism that is contained in individual estimates tends to be compounded, and the final risk estimate may be especially conservative. For example, risk from each chemical in each medium is based on a conservative estimate of the concentration, and a conservative estimate of the toxicity. Thus, each individual estimate itself tends to be doubly conservative. When risks are summed across all chemicals, this conservatism is compounded, resulting in an estimate of the total risk that is very conservative. Likewise, when RME risks are summed across multiple exposure pathways, this is equivalent to assuming that the same individual is simultaneously exposed at the high end of the exposure distribution for each pathway (this is also unlikely). Thus, risk estimates based on the combination of risks across chemicals and pathways are more uncertain, and are likely more conservative, than risk estimates for individual chemicals and pathways.

7.10. Summary of Uncertainties

Because of the uncertainties summarized above, none of the exposure and risk calculations presented above should be interpreted as precise measures of the true risk. Rather, all values should be interpreted as uncertain estimates. Because many (but not all) of the approaches for dealing with uncertainty are intended to be conservative (*i.e.*, are more likely to overestimate than underestimate), the risk values above should generally be thought of as high-end estimates of the true risk, and actual risks are probably somewhat lower than the calculated values.

8. Conclusion

This HHRA considered exposure pathways for a variety of human receptors under both current conditions and future scenarios. The following receptors were considered:

- current/future adult and older child trespassers
- current/future utility, drainage ditch, surveillance, and railroad workers
- current/future commercial/industrial workers
- current/future adult and child recreational visitors
- future construction workers
- future commercial/industrial workers, and
- future child and adult residents.

Within each exposure unit, the HHRA identified potential exposure pathways for receptors and constituents. A complete exposure pathway exists if there is a constituent source; a mechanism for release, retention, or transport of the contaminant; human contact with the medium; and an exposure route at the contact point.

Constituents of potential concern were determined for each exposure area. For each medium, the maximum detected concentration of the constituent was compared to a conservative screening value for the protection of human health. In general, constituents that exceed the screening value or do not have screening values available were retained as COPCs for further evaluation, while those below the screening value were excluded.

Cancer risks and non-cancer hazards were quantified for the reasonable maximum exposure and central tendency scenarios. The regulatory range for acceptable cancer risk is 10^{-6} to 10^{-4} , whereas non-cancer hazards are considered acceptable if they are below 1. This study presents the total risk and hazard for each receptor summed over all media, pathways, and constituents, and identifies the exposure media and constituents that contribute most significantly to the total risks and hazards.

The HHRA indicated that cancer risks and non-cancer hazards were within acceptable limits for the Surveillance Worker, Drainage Ditch Worker, and Railroad Worker. Cancer risks and/or non-cancer hazards exceeded the acceptable regulatory thresholds for the adult and child trespassers, utility workers, commercial/industrial workers, adult and recreators, construction workers, and potential future adult and child residents under the reasonable maximum exposure (RME) scenarios. The table below summarizes the risks and hazards for each receptor. Risks and hazards are presented for each exposure medium and summed across all media.

Table 8.1. Site Risks and Hazards Summary.

			Cancer Risk		Non-Cancer Hazards	
Timeframe	Receptor	Exposure Medium	RME	CT	RME	CT
Current/ Future	Older Child Trespasser	Fish Tissue	1×10^{-4}	3×10^{-5}	2×10^1	5×10^0
		Surface Sediment	7×10^{-4}	4×10^{-5}	7×10^{-1}	6×10^{-1}
		Surface Soil	2×10^{-4}	9×10^{-6}	1×10^0	1×10^{-1}
		Outdoor Air	9×10^{-8}	3×10^{-8}	7×10^{-4}	3×10^{-4}
		Surface Water	3×10^{-4}	2×10^{-4}	2×10^{-1}	8×10^{-2}
		All Media	1×10^{-3}	2×10^{-4}	2×10^1	6×10^0

Table 8.1. Site Risks and Hazards Summary.

Timeframe	Receptor	Exposure Medium	Cancer Risk		Non-Cancer Hazards	
			RME	CT	RME	CT
Current/ Future	Adult Trespasser	Fish Tissue	8×10^{-4}	6×10^{-5}	3×10^1	6×10^0
		Surface Sediment	2×10^{-4}	2×10^{-5}	1×10^{-1}	5×10^{-2}
		Surface Soil	4×10^{-5}	4×10^{-6}	1×10^{-1}	6×10^{-2}
		Outdoor Air	5×10^{-7}	3×10^{-8}	8×10^{-4}	2×10^{-4}
		Surface Water	5×10^{-4}	8×10^{-5}	1×10^{-1}	7×10^{-2}
		All Media	2×10^{-3}	2×10^{-4}	3×10^1	6×10^0
Current/ Future	Utility Worker	Surface/Subsurface Sed.	1×10^{-4}	3×10^{-6}	7×10^0	$1 \times 10^{+3}$
		Surface/Subsurface Soil	1×10^{-4}	4×10^{-6}	4×10^{-1}	3×10^{-2}
		Outdoor Air	2×10^{-5}	2×10^{-6}	1×10^{-0}	1×10^{-1}
		Surface Water	2×10^{-4}	1×10^{-5}	7×10^{-2}	2×10^{-2}
		Shallow Ground Water	4×10^{-6}	3×10^{-7}	3×10^{-2}	7×10^{-3}
		All Media	4×10^{-4}	2×10^{-5}	8×10^0	$1 \times 10^{+3}$
Current/ Future	Utility Worker SYW-12	Surface/Subsurface Soil	1×10^{-5}	4×10^{-7}	6×10^{-2}	5×10^{-3}
		Outdoor Air	9×10^{-9}	8×10^{-10}	1×10^{-4}	3×10^{-5}
		Shallow Ground Water	4×10^{-4}	4×10^{-5}	1×10^{-1}	3×10^{-2}
		All Media	4×10^{-4}	4×10^{-5}	2×10^{-1}	4×10^{-2}
Current/ Future	Surveillance Worker	Surface Soil	6×10^{-6}	2×10^{-6}	1×10^{-1}	1×10^{-1}
		Outdoor Air	7×10^{-7}	3×10^{-8}	1×10^{-3}	2×10^{-4}
		All Media	7×10^{-6}	2×10^{-6}	1×10^{-1}	1×10^{-1}
Current/ Future	Drainage Ditch Worker	Surface Sediment	2×10^{-6}	2×10^{-7}	2×10^{-2}	8×10^{-3}
		Outdoor Air	1×10^{-8}	2×10^{-9}	1×10^{-4}	6×10^{-5}
		Surface Water	2×10^{-7}	4×10^{-8}	3×10^{-2}	1×10^{-2}
		All Media	2×10^{-6}	2×10^{-7}	4×10^{-2}	2×10^{-2}
Current/ Future	Railroad Worker	Surface Soil	9×10^{-6}	2×10^{-6}	8×10^{-2}	7×10^{-2}
		Outdoor Air	2×10^{-8}	3×10^{-9}	2×10^{-3}	9×10^{-4}
		All Media	9×10^{-6}	2×10^{-6}	8×10^{-2}	7×10^{-2}
Current/ Future	Railroad Worker SYW-12	Surface Soil	4×10^{-5}	9×10^{-6}	2×10^{-1}	1×10^{-1}
		Outdoor Air	3×10^{-8}	6×10^{-9}	5×10^{-4}	3×10^{-4}
		All Media	4×10^{-5}	9×10^{-6}	2×10^{-1}	1×10^{-1}
Current/ Future	Commercial/Industrial Worker	Surface Soil	3×10^{-4}	4×10^{-5}	9×10^{-1}	3×10^{-1}
		Outdoor Air	1×10^{-7}	4×10^{-8}	6×10^{-3}	5×10^{-3}
		All Media	3×10^{-4}	4×10^{-5}	9×10^{-1}	3×10^{-1}
Current/Future	Child Recreator SYW-12	Surface Soil	4×10^{-4}	3×10^{-5}	9×10^{-1}	9×10^{-2}
		Outdoor Air	7×10^{-9}	2×10^{-9}	5×10^{-4}	2×10^{-4}
		All Media	4×10^{-4}	3×10^{-5}	9×10^{-1}	9×10^{-2}
Current/Future	Adult Recreator SYW-12	Surface Soil	1×10^{-5}	2×10^{-6}	4×10^{-2}	2×10^{-2}
		Outdoor Air	1×10^{-8}	8×10^{-10}	1×10^{-4}	3×10^{-5}
		All Media	1×10^{-5}	2×10^{-6}	4×10^{-2}	2×10^{-2}
Future	Child Recreator	Fish Tissue	3×10^{-4}	6×10^{-5}	4×10^1	1×10^1
		Surface Sediment	7×10^{-3}	1×10^{-3}	4×10^0	6×10^{-1}
		Surface Soil	1×10^{-4}	4×10^{-5}	3×10^0	3×10^{-1}
		Outdoor Air	5×10^{-7}	2×10^{-7}	3×10^{-3}	9×10^{-4}
		Surface Water	1×10^{-3}	8×10^{-4}	5×10^{-1}	2×10^{-1}
		All Media	9×10^{-3}	2×10^{-3}	5×10^1	1×10^1

Table 8.1. Site Risks and Hazards Summary.

Timeframe	Receptor	Exposure Medium	Cancer Risk		Non-Cancer Hazards	
			RME	CT	RME	CT
Future	Adult Recreator	Fish Tissue	8×10^{-4}	6×10^{-5}	3×10^1	6×10^0
		Surface Sediment	2×10^{-4}	3×10^{-5}	2×10^{-1}	8×10^{-2}
		Surface Soil	3×10^{-5}	3×10^{-6}	2×10^{-1}	6×10^{-2}
		Outdoor Air	7×10^{-7}	5×10^{-8}	8×10^{-4}	2×10^{-4}
		Surface Water	5×10^{-4}	8×10^{-5}	2×10^{-1}	1×10^{-1}
		All Media	2×10^{-3}	2×10^{-4}	3×10^1	6×10^0
Future	Construction Worker	Surface/Subsurface Sed.	6×10^{-5}	4×10^{-5}	1×10^1	9×10^0
		Surface/Subsurface Soil	5×10^{-5}	2×10^{-5}	4×10^0	2×10^0
		Outdoor Air	2×10^{-5}	5×10^{-6}	1×10^1	3×10^0
		Surface Water	8×10^{-5}	4×10^{-5}	9×10^{-1}	4×10^{-1}
		Shallow Ground Water	2×10^{-6}	9×10^{-7}	3×10^{-1}	2×10^{-1}
		All Media	2×10^{-4}	1×10^{-4}	3×10^1	1×10^1
Future	Construction Worker SYW-12	Surface/Subsurface Soil	5×10^{-6}	2×10^{-6}	7×10^{-1}	3×10^{-1}
		Outdoor Air	1×10^{-8}	3×10^{-9}	4×10^{-3}	9×10^{-4}
		Shallow Ground Water	2×10^{-4}	1×10^{-4}	2×10^0	8×10^{-1}
		All Media	2×10^{-4}	1×10^{-4}	2×10^0	1×10^0
Future	Commercial/Industrial Worker	Surface Soil	2×10^{-4}	3×10^{-5}	1×10^0	4×10^{-1}
		Outdoor Air	5×10^{-6}	2×10^{-6}	9×10^{-3}	8×10^{-3}
		Potable Water	4×10^{-3}	1×10^{-3}	6×10^1	5×10^1
		All Media	4×10^{-3}	1×10^{-3}	6×10^1	5×10^1
Future	Commercial/Industrial Worker SYW-12	Surface Soil	6×10^{-5}	8×10^{-6}	3×10^{-1}	1×10^{-1}
		Outdoor Air	1×10^{-7}	3×10^{-8}	2×10^{-3}	2×10^{-3}
		All Media	6×10^{-5}	8×10^{-6}	3×10^{-1}	1×10^{-1}
Future	Child Resident	Surface Soil	1×10^{-3}	4×10^{-4}	3×10^1	5×10^0
		Outdoor Air	8×10^{-6}	8×10^{-6}	5×10^{-2}	5×10^{-2}
		Potable Water	7×10^{-1}	1×10^{-1}	2×10^2	2×10^2
		Shower Vapor	9×10^{-3}	3×10^{-3}	5×10^2	2×10^2
		All Media	7×10^{-1}	1×10^{-1}	8×10^2	4×10^2
Future	Adult Resident	Surface Soil	9×10^{-5}	2×10^{-5}	7×10^{-1}	6×10^{-1}
		Outdoor Air	1×10^{-5}	3×10^{-6}	1×10^{-2}	1×10^{-2}
		Potable Water	6×10^{-2}	1×10^{-2}	9×10^1	9×10^1
		Shower Vapor	6×10^{-3}	2×10^{-3}	7×10^1	3×10^1
		All Media	7×10^{-2}	2×10^{-2}	2×10^2	1×10^2
Future	Child Resident SYW-12*	Surface Soil	7×10^{-4}	3×10^{-4}	7×10^0	1×10^0
		Outdoor Air	1×10^{-7}	1×10^{-7}	9×10^{-3}	9×10^{-3}
		All Media	7×10^{-4}	3×10^{-4}	7×10^0	1×10^0
Future	Adult Resident SYW-12*	Surface Soil	5×10^{-5}	8×10^{-6}	2×10^{-1}	1×10^{-1}
		Outdoor Air	2×10^{-7}	5×10^{-8}	2×10^{-3}	2×10^{-3}
		All Media	5×10^{-5}	8×10^{-6}	2×10^{-1}	1×10^{-1}

* The exposure units listed in this HHRA do not include SYW-12, with the exception of EU8, which does include SYW-12. Therefore, although not specifically listed in Table 8.1 above, the potable water and shower vapor scenarios have already been included for the child and adult residents of SYW-12.

The greatest cancer risk posed to current receptors is 2×10^{-3} for the adult trespasser and the greatest non-cancer hazard is 30 for the same receptor. The greatest cancer risk and non-cancer hazard posed

to a potential future receptor is for the future child resident. The cancer risk of 7×10^{-1} is driven primarily by exposure to ground water as a drinking water source and to surface soil. The non-cancer hazard of 8×10^2 is also driven primarily by exposure to ground water as a drinking water source and to surface soil. As noted previously, the use of ground water at the Site for potable applications is considered hypothetical and is extremely unlikely for several reasons: 1) the area is supplied by municipal water from OCWA; 2) the yield of the overburden ground water unit is inadequate for water supply wells; and 3) the high salinity of the deep aquifer (3,000 mg/l chloride) precludes its use as drinking water.

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TABLE 1
HISTORICAL DATA SOURCES
HONEYWELL WASTEBED B/HARBOR BROOK - GEDDES AND SYRACUSE, NEW YORK

Report/Investigation Title	Sampled Area/Date	Chemical Analyses for Data Used in Human Health Risk Assessment	Data Used in Human Health Risk Assessment
Supplemental Sediment Sampling at Onondaga Lake – East Flume (PTI, 1994)	Lower East Flume/1993	VOCs, SVOCs, pesticides, PCBs, metals, mercury, grain size, TOC, chloride, and calcium carbonate	Sediment samples collected by PTI for the Onondaga Lake RI
Harbor Brook surface water and sediment sampling (O'Brien & Gere)	Harbor Brook/Nov. 1996	VOCs, SVOCs, PCBs, pesticides, and inorganics	Twelve sediment samples from the 0 to 12-inch depth interval and eight sediment samples from 12 inches to refusal (26.5 inches max.)
Harbor Brook Sediment IRM Investigation Report (BBL, 2001)	Harbor Brook to Hiawatha Boulevard/ Jan. and Feb. 2001	VOCs, SVOCs, pesticides, PCBs, metals, total mercury, cyanide, and TOC; some samples analyzed for PCDD/PCDFs	Sediment probing data, Harbor Brook sediments, and wetlands soil borings
Onondaga Lake Human Health Risk Assessment (NYSDEC, 2002)	Onondaga Lake	---	Fish tissue exposure point concentrations
Willis Avenue Chlorobenzene Site Remedial Investigation (O'Brien & Gere, 2002)	Lakeshore Area/Apr. 1992, Oct. 1992, and Jan. 1995	VOCs, SVOCs, metals, and mercury	Ground water samples from wells located on the Wastebed B/Harbor Brook Site
	Bank of Harbor Brook north of I-690/ Apr. 1999	VOCs, SVOCs, PCBs, pesticides, and metals	One seep sampled
	Dredge Spoils Area #2/Sept. and Oct. 1998	TCLP VOCs, TCLP SVOCs, TCLP herbicides, TCLP metals, and TCLP mercury	Soil boring subsurface soil samples
	Dredge Spoils Areas #1 and #2/Oct. 1997	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide	Test pit subsurface soil samples
	East Flume/Oct. 1997	VOCs, PCBs/pesticides, mercury, PCDD/PCDFs, and TOC	Sediment samples
Revised Remedial Investigation Report. Wastebed B/Harbor Brook Site; Preliminary Site Assessment (O'Brien & Gere, 2007)	Lakeshore Area, Penn-Can Property, and Railroad Area/Sept. 2000 and May 2001	VOCs, SVOCs, pesticides, PCBs, metals, and mercury	Ground water samples
	Lakeshore Area, Penn-Can Property, and Railroad Area/July 2000 and Feb. and Mar. 2001	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide	Geoprobe boring surface and subsurface soil samples
	Lakeshore Area, Penn-Can Property, and Railroad Area/July 2000 and Feb. and Mar. 2001	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide	Soil boring surface and subsurface samples
	Lakeshore Area, Penn-Can Property, and Railroad Area/July 2000	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide; some samples also analyzed for TCLP analyses, reactivity, and ignitability	Test pit subsurface soil samples
	Harbor Brook, Penn-Can Property, Railroad Area, I-690 drainage ditch on Lakeshore Area/May 2001	VOCs, SVOCs, pesticides, PCBs, metals, and mercury; field parameters: temperature, specific conductivity, dissolved oxygen, and pH	Surface water samples
	Penn-Can Property, Railroad Area, I-690 drainage ditch on Lakeshore Area/May 2001	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and PCDD/PCDFs	Sediment samples
	Lakeshore Area/Aug. 2000	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide	Surface and subsurface soil samples collected from wetland soil borings
CSX supplemental sediment sampling (O'Brien & Gere, 2007)	Harbor Brook/Nov. 2002	VOCs, SVOCs, pesticides, PCBs, metals, mercury, cyanide, and PCDD/PCDFs	Sediment samples

TABLE 1
HISTORICAL DATA SOURCES
HONEYWELL WASTEBED B/HARBOR BROOK - GEDDES AND SYRACUSE, NEW YORK

Report/Investigation Title	Sampled Area/Date	Chemical Analyses for Data Used in Human Health Risk Assessment	Data Used in Human Health Risk Assessment
Revised Remedial Investigation Report. Wastebed B/Harbor Brook Site; Remedial Investigation (O'Brien & Gere, 2007)	Lakeshore Area, Penn-Can Property, Railroad Area, AOS #1, and AOS #2/May 2003 and Aug. 2003	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and major cations and anions	Ground water samples
	Harbor Brook, Penn-Can Property, Railroad Area, and I-690 drainage ditch on Lakeshore Area/June 2003 and Sep. 2003	VOCs, SVOCs, pesticides, PCBs (including Aroclor 1268), metals, mercury, cyanide, pH, and hardness	Surface water samples
	Harbor Brook, Penn-Can Property, Railroad Area, I-690 drainage ditch on Lakeshore Area, and drainage ditch associated with AOS #2/June 2003	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide	Sediment samples
	Onondaga Lake shore and Harbor Brook banks/Dec. 2002 and June 2003	VOCs, SVOCs, pesticides, PCBs (including Aroclor 1268), metals, mercury, cyanide, and major cations and anions	Seep water samples
	I-690 storm drain system/June 2003 and Sep. 2003	VOCs, SVOCs, pesticides, PCBs, metals, mercury, cyanide, and PCDD/PCDFs	Storm water and sediment sampled from I-690 catch basins
	Lakeshore Area, Penn-Can Property, Railroad Area, AOS #1, and AOS #2/Dec. 2001 to Mar. 2002	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide	Soil boring subsurface soil samples
	Lakeshore Area, Penn-Can Property, Railroad Area, AOS #1, and AOS #2/Dec. 2002	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide. Some wetland samples also analyzed for methylmercury and polychlorinated dioxins and furans	Surface soil samples
	Penn-Can Property drum area/May 2003	VOCs, SVOCs, pesticides, PCBs, metals, mercury, and cyanide	Surface soil samples
Revised Remedial Investigation Report. Wastebed B/Harbor Brook Site; Supplemental RI (O'Brien & Gere, 2007)	SYW-12/Dec. 2006 to Jan. 2007	VOCs, SVOCs (including PXE & PTE), pesticides, PCBs, metals, mercury, and cyanide	Soil borings subsurface soils
	SYW-12/Mar. 2007 and July and Aug. 2007	VOCs, SVOCs, pesticides, PCBs, metals, mercury, hardness, alkalinity, ammonia, TKN, CBOD, and major cations and anions	Ground water samples
	SYW-12/Dec. 2006 to Jan. 2007	VOCs, SVOCs, pesticides, PCBs, metals, mercury, cyanide, total petroleum hydrocarbons/petroleum fingerprint, methyl mercury, TOC, grain size, and PCDD/PCDFs	Surface soil (wetland sediment) samples
	Dredge Spoils Areas #1 and #2/Nov. 2006	VOCs, SVOCs (including PXE and PTE), pesticides, PCBs, metals, mercury, and cyanide	Test pit subsurface soil samples
	Harbor Brook/Oct. 2006	VOCs, SVOCs (including PXE & PTE), pesticides, PCBs, metals, mercury, and cyanide	Sediment samples
	Wastebed B/Harbor Brook/Oct. to Nov. 2006	VOCs, SVOCs (including PXE & PTE), pesticides, PCBs, metals, mercury, and cyanide	Soil borings subsurface soils

TABLE 1
HISTORICAL DATA SOURCES
HONEYWELL WASTEBED B/HARBOR BROOK - GEDDES AND SYRACUSE, NEW YORK

Report/Investigation Title		Sampled Area/Date	Chemical Analyses for Data Used in Human Health Risk Assessment	Data Used in Human Health Risk Assessment
Revised Remedial Investigation Report. Wastebed B/Harbor Brook Site;		Soil boring HB-SB-65/Nov. 2006	VOCs, SVOCs, pesticides, PCBs (including Aroclor 1268), metals, mercury, and cyanide	One surface soil sample
Supplemental RI (cont'd.) (O'Brien & Gere, 2007)		Lakeshore Area and Penn-Can Property/Nov. 2006	Method TO-15 VOCs	Twenty four soil vapor samples and four sub-slab samples
Notes:				
VOC = Volatile Organic Compound		PCDD/PCDF = Polychlorinated Dibenzo-p-Dioxin/Polychlorinated Dibenzofuran		
SVOC = Semivolatile Organic Compound		TCLP = Toxicity Characteristic Leaching Potential		
PCB = Polychlorinated Biphenyl		AOS = Area of Study		
TOC = Total Organic Carbon		PXE = 1-phenyl-1-[2,4-dimethylphenyl]-ethane		
BBL = Blasland, Bouck & Lee		PTE = 1-phenyl-1-[4-methylphenyl]-ethane		
NYSDEC = New York State Department of Environmental Conservation				

TABLE 2
CHEMICAL-SPECIFIC EXPOSURE PARAMETERS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Chemical	FA	t*	tao	B	GI	Source	ABS	Source	Source	PC	Source
1,1,2,2-TETRACHLOROETHANE	1	2.24	0.93	0	1	1	-	-	-	0.0069	4
1,1,2-TRICHLOROETHANE	1	1.43	0.6	0	1	1	-	-	-	0.0064	4
1,1-BIPHENYL	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.06547025	5
1,2,3-TRICHLOROENZENE	-	-	-	-	1	1	-	-	-	0.04919036	5
1,2,4-TRICHLOROENZENE	1	2.66	1.11	0.3	1	1	-	-	-	0.066	4
1,2,4-TRIMETHYLBENZENE	-	-	-	-	1	1	-	-	-	0.105118705	5
1,2-DICHLOROENZENE	1	1.71	0.71	0.2	1	1	-	-	-	0.041	4
1,2-DICHLOROETHANE	1	0.92	0.38	0	1	1	-	-	-	0.0042	4
1,2-DICHLOROPROPANE	1	1.1	0.46	0	1	1	-	-	-	0.0078	4
1,3,5-TRIMETHYLBENZENE	-	-	-	-	1	1	-	-	-	0.084316778	5
1,3-DICHLOROENZENE	1	1.71	0.71	0.3	1	1	-	-	-	0.058	4
1,4-DICHLOROENZENE	1	1.71	0.71	0.2	1	1	-	-	-	0.042	4
1-Methylnaphthalene	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.130701328	5
2,2-OXYBIS(1-CHLOROPROPANE)	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.008814547	5
2,3,7,8-TCDD Equivalent	0.5	30.09	6.82	5.6	1	1	0.03	USEPA 2004, Exhibit 3-4	3	0.81	4
2,4,6-TRICHLOROPHENOL	1	3.27	1.36	0.2	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.035	4
2,4-DICHLOROPHENOL	1	2.1	0.87	0.1	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.021	4
2,4-DIMETHYLPHENOL	1	1.24	0.52	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.011	4
2,4-DINITROPHENOL	1	2.76	1.15	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0015	4
2,4-DINITROTOLUENE	1	2.69	1.12	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0031	4
2,6-DINITROTOLUENE	1	2.69	1.12	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0021	4
2-CHLOROPHENOL	1	1.34	0.56	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.008	4
2-HEXANONE	-	-	-	-	1	1	-	-	-	0.00354735	5
2-METHYLNAPHTHALENE	-	-	-	-	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.130701328	5
2-METHYLPHENOL	1	1.03	0.43	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0077	4
2-NITROANILINE	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.004054413	5
2-NITROPHENOL	1	1.54	0.64	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.004	4
3&4-METHYLPHENOL	1	1.03	0.43	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.007381761	5
3,3-DICHLOROBENZIDINE	1	6.72	2.8	0.1	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.012562382	5
3-NITROANILINE	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.002141549	5
4,4-DDD	0.8	25.99	6.65	1.2	1	1	-	-	-	0.18	4
4,4-DDT	0.7	42.51	10.45	1.9	1	1	0.03	USEPA 2004, Exhibit 3-4	3	0.27	4
4,6-DINITRO-2-METHYLPHENOL	1	3.3	1.38	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0031	4
4-BROMOPHENYL PHENYL ETHER	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.183393526	5
4-CHLORO-3-METHYLPHENOL	1	1.61	0.67	0.1	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.029	4
4-CHLOROPHENYL PHENYL ETHER	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.055808969	5
4-METHYLPHENOL	1	1.03	0.43	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0077	4
4-NITROANILINE	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.002207639	5
4-NITROPHENOL	1	1.54	0.64	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0048	4
ACENAPHTHENE	-	-	-	-	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.084100775	5
ACENAPHTHYLENE	-	-	-	-	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.061725412	5
ACETONE	-	-	-	-	1	1	-	-	-	0.000520955	5
ALDRIN	1	28.54	11.89	0	1	1	-	-	-	0.0014	4
ALPHA CHLORDANE	0.7	51.05	21.27	0.3	1	1	-	-	-	0.034	4
ALPHA-BHC	-	-	-	-	1	1	-	-	-	0.024151939	5
ALUMINUM	-	-	-	-	1	1	-	-	-	0.001	6
ANTHRACENE	-	-	-	-	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.158180156	5
ANTIMONY	-	-	-	-	0.15	2	-	-	-	0.001	7
ARSENIC	-	-	-	-	1	1	0.03	USEPA 2004, Exhibit 3-4	3	0.001	7
ATRAZINE	-	-	-	-	1	1	-	-	-	0.007106995	5
BARIUM	-	-	-	-	0.07	2	-	-	-	0.001	7
BENZO(A)ANTHRACENE	1	8.53	2.03	2.8	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.47	4
BENZENE	1	0.7	0.29	0.1	1	1	-	-	-	0.015	4
BENZO(A)PYRENE	1	11.67	2.69	4.3	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.7	4
BENZO(B)FLUORANTHENE	1	12.03	2.77	4.3	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.7	4
BENZO(G,H,I)PERYLENE	-	-	-	-	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.893305484	5
BENZO(K)FLUORANTHENE	-	-	-	-	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.611797925	5
BERYLLIUM	-	-	-	-	0.007	2	-	-	-	0.001	7
BIS(2-CHLOROETHOXY)METHANE	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.000864649	5
BIS(2-CHLOROETHYL)ETHER	1	1.62	0.68	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0018	4
BIS(2-ETHYLHEXYL)PHTHALATE	0.8	39.93	16.64	0.2	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.025	4
BROMODICHLOROMETHANE	1	2.12	0.88	0	1	1	-	-	-	0.0046	4
BROMOMETHANE	1	0.87	0.36	0	1	1	-	-	-	0.0028	4
CADMIUM	-	-	-	-	0.025	2	0.001	USEPA 2004, Exhibit 3-4	3	0.001	7
CARBAZOLE	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.027649036	5
CARBON DISULFIDE	1	0.72	0.3	0.1	1	1	-	-	-	0.017	4
CARBON TETRACHLORIDE	1	1.86	0.78	0.1	1	1	-	-	-	0.016	4
CHLORDANE	0.7	50.91	21.21	0.3	1	1	0.04	USEPA 2004, Exhibit 3-4	3	0.038	4
CHLOROENZENE	1	1.09	0.46	0.1	1	1	-	-	-	0.028	4
CHLORODIBROMOMETHANE	1	3.77	1.57	0	1	1	-	-	-	0.0032	4
CHLOROETHANE	1	0.59	0.24	0	1	1	-	-	-	0.0061	4
CHLOROFORM	1	1.19	0.5	0	1	1	-	-	-	0.0068	4

TABLE 2
CHEMICAL-SPECIFIC EXPOSURE PARAMETERS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

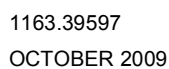
Chemical	FA	t*	tao	B	GI	Source	ABS	Source	Source	PC	Source
CHROMIUM	-	-	-	-	0.025	2	-	-	-	0.002	7
CHRYSENE	1	8.53	2.03	2.8	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.47	4
CIS-1,3-DICHLOROPROPENE	-	-	-	-	1	1	-	-	-	0.007917134	5
COBALT	-	-	-	-	1	1	-	-	-	0.0004	7
COPPER	-	-	-	-	1	1	-	-	-	0.001	7
CYANIDE	-	-	-	-	1	1	-	-	-	0.001	7
DELTA-BHC	-	-	-	-	1	1	-	-	-	0.01893878	5
DIBENZO(A,H)ANTHRACENE	0.6	17.57	3.88	9.7	1	1	0.13	USEPA 2004, Exhibit 3-4	3	1.5	4
DIBENZOFURAN	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.102659702	5
DICHLOROBENZENES	-	-	-	-	1	1	-	-	-	0.034801663	5
DIELDRIN	0.8	35.09	14.62	0.1	1	1	-	-	-	0.012	4
DODECANE	-	-	-	-	1	1	-	-	-	2.279796066	5
ENDOSULFAN I	-	-	-	-	1	1	-	-	-	0.001836877	5
ENDOSULFAN II	-	-	-	-	1	1	-	-	-	0.002043055	5
ENDOSULFAN SULFATE	-	-	-	-	1	1	-	-	-	0.001714147	5
ENDRIN ALDEHYDE	-	-	-	-	1	1	-	-	-	0.017175287	5
ENDRIN KETONE	-	-	-	-	1	1	-	-	-	0.022924718	5
ETHYLBENZENE	1	1.01	0.42	0.2	1	1	-	-	-	0.049	4
FLUORANTHENE	1	5.68	1.45	1.2	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.22	4
FLUORENE	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.106844458	5
HEPTACHLOR EPOXIDE (as Heptaclor)	-	-	-	-	1	1	-	-	-	0.0086	4
HEXACHLOROBENZENE	0.9	16.21	4.22	0.9	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.13	4
HEXACHLOROBUTADIENE	0.9	7.42	3.09	0.5	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.081	4
HEXACHLOROETHANE	1	5.44	2.27	0.2	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.03	4
HIGHLY CHLORINATED PCBs	-	-	-	-	1	1	0.14	USEPA 2004, Exhibit 3-4	3	0.87821328	5
INDENO(1,2,3-CD)PYRENE	0.6	16.83	3.78	6.7	1	1	0.13	USEPA 2004, Exhibit 3-4	3	1	4
IRON	-	-	-	-	1	1	-	-	-	0.001	6
ISOPROPYLBENZENE	-	-	-	-	1	1	-	-	-	0.087606433	5
LEAD	-	-	-	-	1	1	-	-	-	0.0001	7
LESS CHLORINATED PCBs	-	-	-	-	1	1	0.14	USEPA 2004, Exhibit 3-4	3	0.433071895	5
MANGANESE	-	-	-	-	0.04	2	-	-	-	0.001	7
MERCURY	-	-	-	-	1	1	-	-	-	0.001	7
METHYLENE CHLORIDE	1	0.76	0.32	0	1	1	-	-	-	0.0035	4
NAPHTHALENE	1	1.34	0.56	0.2	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.047	4
NICKEL	-	-	-	-	0.04	2	-	-	-	0.0002	7
NITROBENZENE	-	-	-	-	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.005228583	5
N-NITROSO-DI-N-PROPYLAMINE	1	1.37	0.57	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0023	4
PENTACHLOROPHENOL	0.9	13.82	3.33	2.5	1	1	0.25	USEPA 2004, Exhibit 3-4	3	0.39	4
PHENANTHRENE	1	4.11	1.06	0.7	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.14	4
PHENOL	1	0.86	0.36	0	1	1	0.1	USEPA 2004, Exhibit 3-4	3	0.0043	4
P-ISOPROPYLTOLUENE	-	-	-	-	1	1	-	-	-	0.122565432	5
PYRENE	-	-	-	-	1	1	0.13	USEPA 2004, Exhibit 3-4	3	0.307009631	5
SEC-BUTYLBENZENE	-	-	-	-	1	1	-	-	-	0.111901008	5
SELENIUM	-	-	-	-	1	1	-	-	-	0.001	7
SILVER	-	-	-	-	0.04	2	-	-	-	0.0006	7
STYRENE	1	0.98	0.41	0.1	1	1	-	-	-	0.037	4
TETRACHLOROETHENE	1	2.18	0.91	0.2	1	1	-	-	-	0.033	4
THALLIUM	-	-	-	-	1	1	-	-	-	0.001	7
TOLUENE	1	0.84	0.35	0.1	1	1	-	-	-	0.031	4
TOXAPHENE	0.8	53.75	22.4	0.1	1	1	-	-	-	0.012	4
TRANS-1,3-DICHLOROPROPENE	-	-	-	-	1	1	-	-	-	0.007917134	5
TRICHLOROETHENE	1	1.39	0.58	0.1	1	1	-	-	-	0.012	4
VANADIUM	-	-	-	-	0.026	2	-	-	-	0.001	7
VINYL CHLORIDE	1	0.57	0.24	0	1	1	-	-	-	0.0056	4
XYLENES, TOTAL	-	-	-	-	1	1	-	-	-	0.052173263	5
ZINC	-	-	-	-	1	1	-	-	-	0.0006	7

Notes:

- 1: USEPA 2004, Section 4.2
- 2: USEPA 2004, Exhibit 4-1
- 3: USEPA 2004, Exhibit 3-4
- 4: USEPA 2004, Exhibit B-3
- 5: USEPA 2004, Calculated per Equation 3.8
- 6: USEPA 2004, Exhibit 3-1
- 7: USEPA 2004, Exhibit B-4



QUADRANGLE LOCATION



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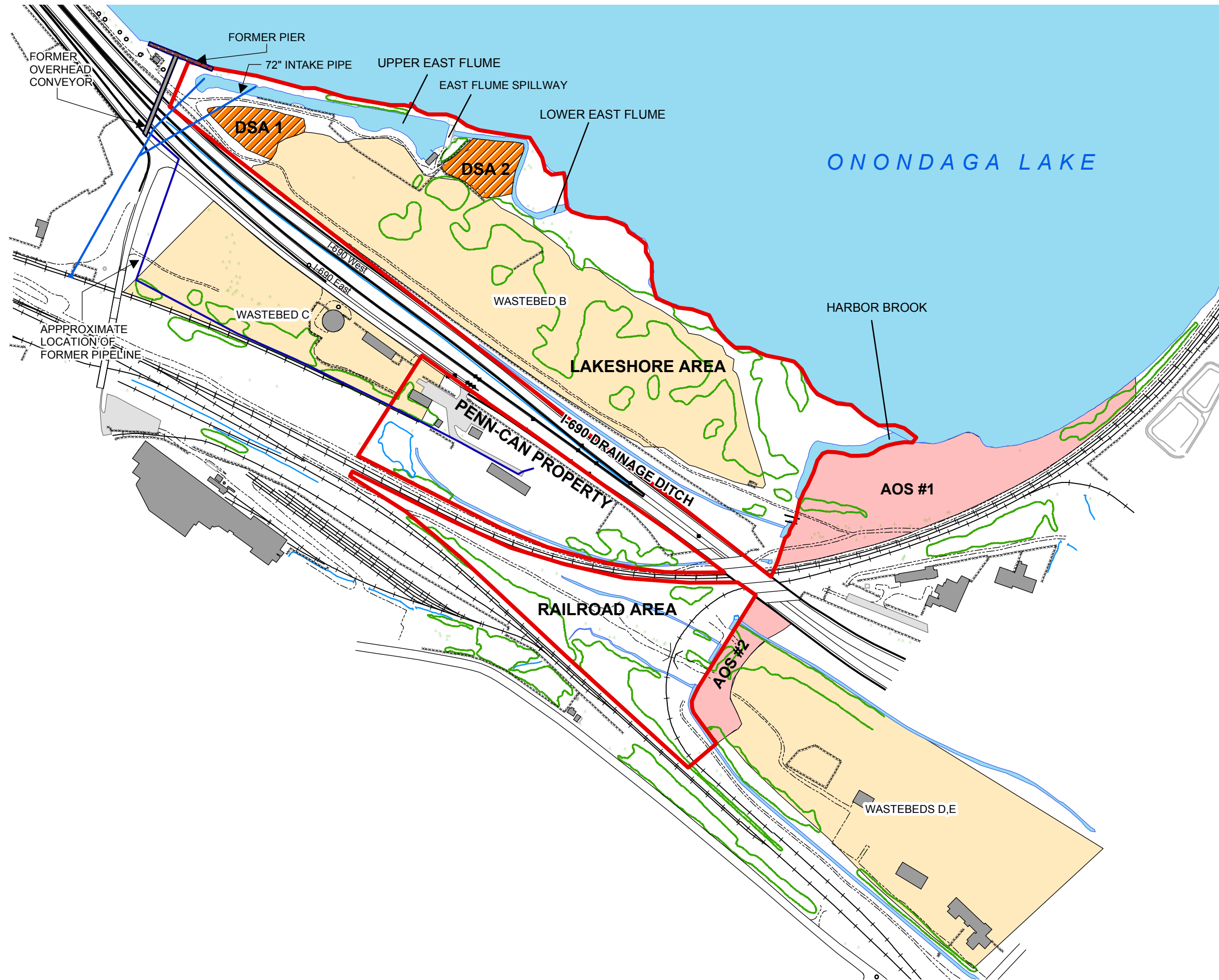


FIGURE 2



LEGEND

- EXPOSURE AREAS**
- HARBOR BROOK SITE
 - DREDGE SPOIL AREA
 - ADDITIONAL AREA OF STUDY
- SITE FEATURES**
- TREE LINE
 - DRAINAGE
 - FENCELINE
 - UNPAVED ACCESS ROAD
 - PAVED ROAD
 - HIGHWAY
 - RAILROAD
 - PAVED PARKING/DRIVEWAY
 - EXISTING BUILDING
 - WASTEBEDS
 - OPEN WATER

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

SITE PLAN

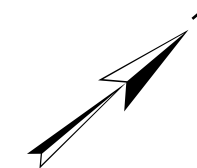


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FIGURE 3



LEGEND

 SYW-12 AREA

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

SYW-12
SITE PLAN



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FIGURE 4

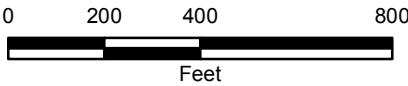


LEGEND

- HARBOR BROOK SITE
- ADDITIONAL AREA OF STUDY
- CONTOUR LINES**
 - 2 ft INTERVAL
 - 10 ft INTERVAL

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

TOPOGRAPHIC MAP



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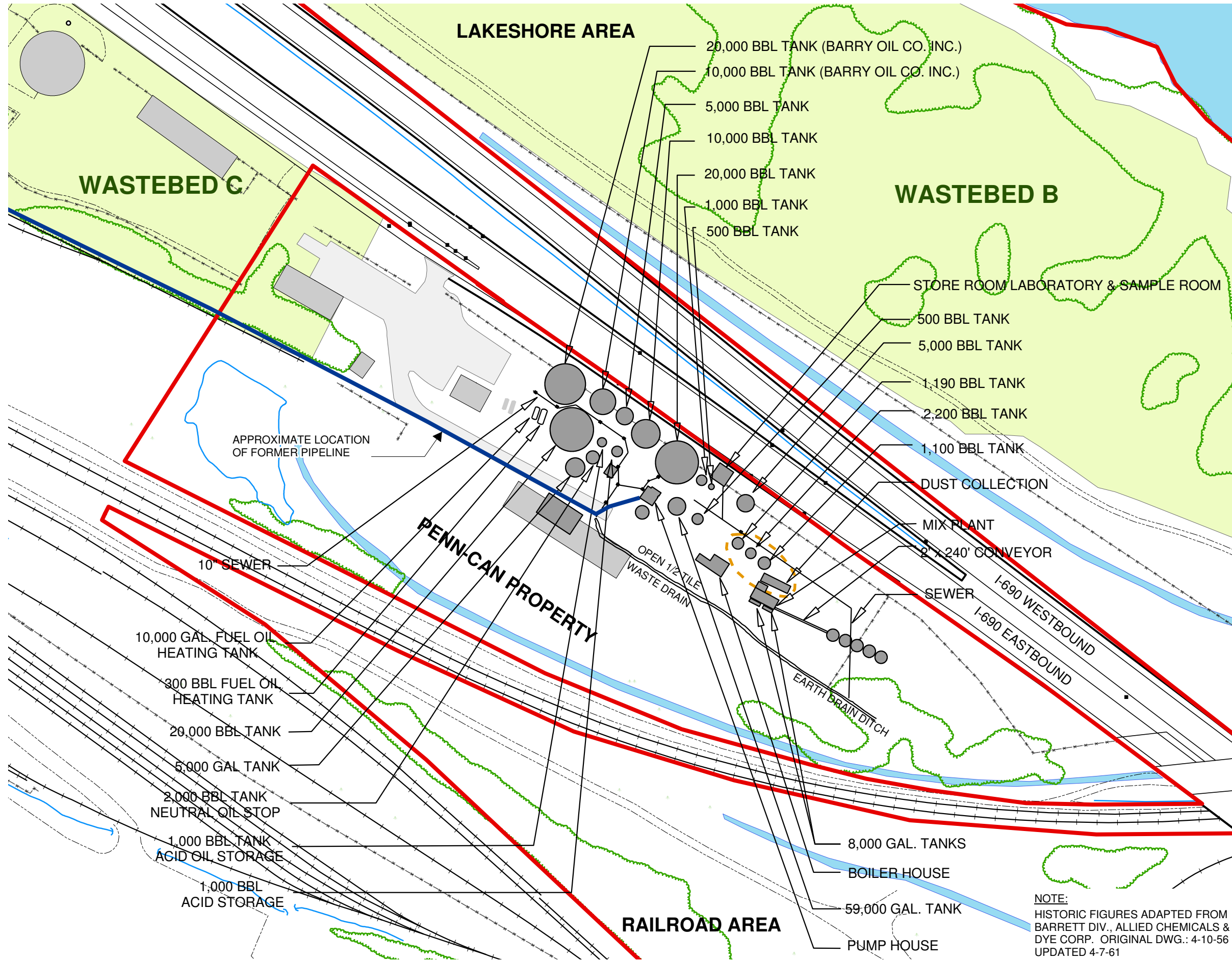


FIGURE 5



LEGEND

- HARBOR BROOK SITE
- APPROXIMATE LOCATION OF ASPHALT TANK BOTTOM PIT
- HISTORIC STRUCTURE LOCATION
- EXISTING BUILDING
- PAVED PARKING/DRIVEWAY
- WASTEBEDS
- FENCELINE
- UNPAVED ACCESS ROAD
- PAVED ROAD
- HIGHWAY
- RAILROAD
- DRAINAGE
- TREE LINE

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

LOCATION OF FORMER
BARRETT PAVING
FACILITY TANKS AND
STRUCTURES



NOTE:
HISTORIC FIGURES ADAPTED FROM
BARRETT DIV., ALLIED CHEMICALS &
DYE CORP. ORIGINAL DWG.: 4-10-56
UPDATED 4-7-61

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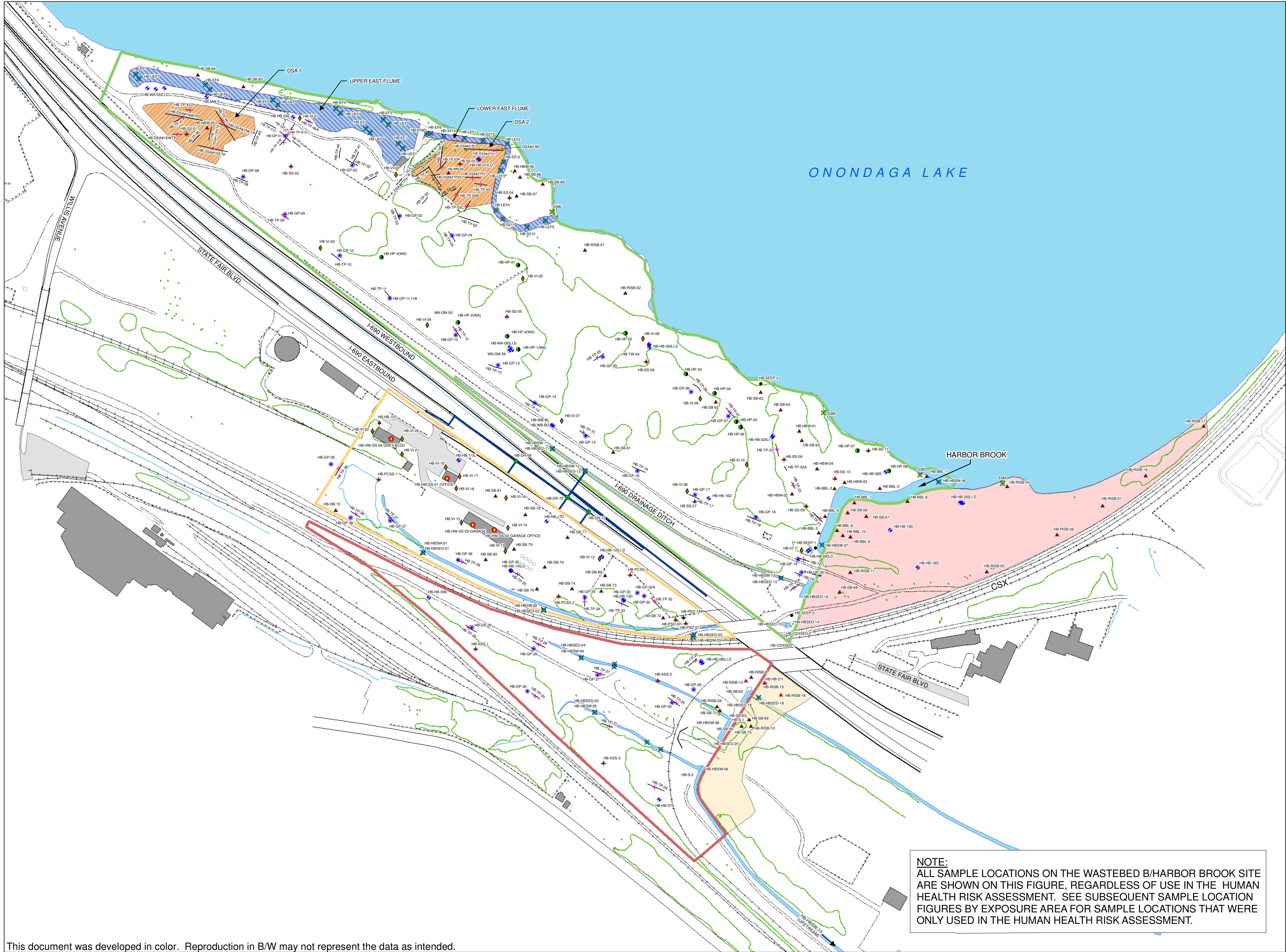


FIGURE 6



LEGEND

LOCATION TYPE

- CATCH BASIN
- GEOPROBE
- HYDROPUNCH
- MONITORING WELL
- SEDIMENT BORING
- SEEP
- SOIL BORING
- SOIL BORING (NOT SAMPLED/VISUAL ONLY)
- SOIL VAPOR
- SUB SLAB
- SURFACE SOIL
- SURFACE WATER/SEDIMENT
- TAR
- TEST PIT SAMPLE
- WETLAND SEDIMENT
- TEST PIT

EXPOSURE AREA

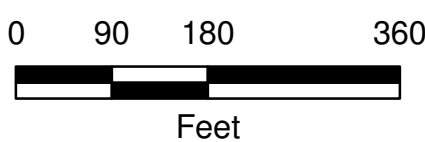
- RAILROAD AREA
- LAKESHORE AREA
- PENN-CAN PROPERTY
- ADDITIONAL AREA OF STUDY #1
- ADDITIONAL AREA OF STUDY #2
- DREDGE SPOIL AREA
- EAST FLUME
- I-190 DRAINAGE DITCH

SITE FEATURES

- TREE LINE
- STORM SEWER
- UNPAVED ACCESS ROAD
- PAVED ROAD
- HIGHWAY
- FENCELINE
- RAILROAD
- EXISTING BUILDING
- PAVED PARKING/DRIVEWAY

HONEYWELL
WASTEBED B/ HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

SAMPLE LOCATIONS



NOTE:
ALL SAMPLE LOCATIONS ON THE WASTEBED B/HARBOR BROOK SITE
ARE SHOWN ON THIS FIGURE, REGARDLESS OF USE IN THE HUMAN
HEALTH RISK ASSESSMENT. SEE SUBSEQUENT SAMPLE LOCATION
FIGURES BY EXPOSURE AREA FOR SAMPLE LOCATIONS THAT WERE
ONLY USED IN THE HUMAN HEALTH RISK ASSESSMENT.

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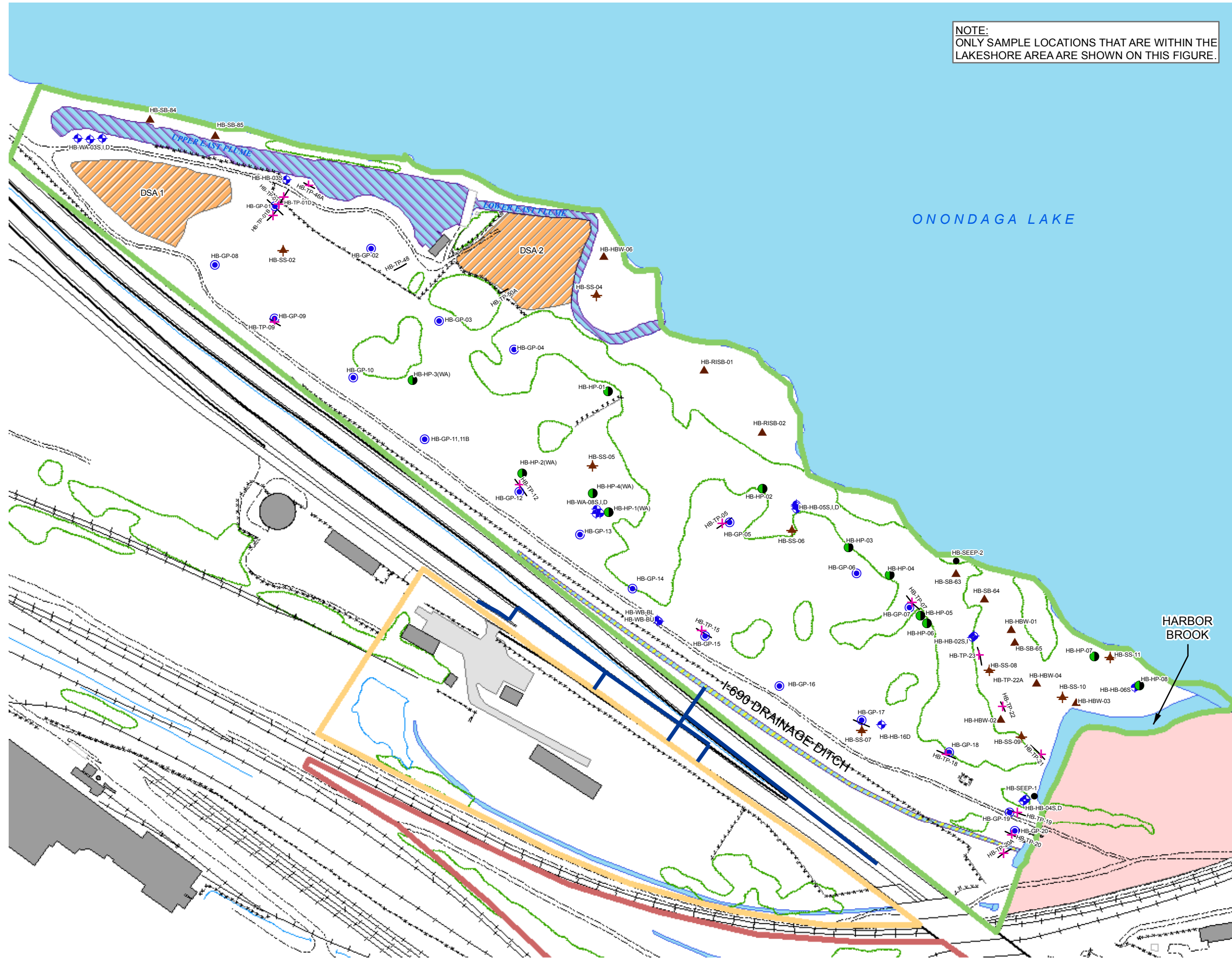


FIGURE 6A



LEGEND

LOCATION TYPE

- GEOPROBE
- HYDRO PUNCH
- MONITORING WELL
- SEEP
- ▲ SOIL BORING
- ▲ SURFACE SOIL
- ✦ TEST PIT SAMPLE
- TEST PIT

SITE NAME

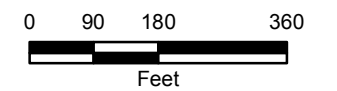
- RAILROAD AREA
- LAKESHORE AREA
- PENN-CAN PROPERTY
- EAST FLUME
- I-690 DRAINAGE DITCH
- DREDGE SPOIL AREA
- ADDITIONAL AREA OF STUDY #1

SITE FEATURES

- TREE LINE
- UNPAVED ACCESS ROAD
- PAVED ROAD
- HIGHWAY
- RAILROAD
- EXISTING BUILDING
- PAVED PARKING/DRIVEWAY

HONEYWELL
WASTEBED B/ HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

LAKESHORE AREA
SAMPLE LOCATIONS



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4

LEGEND

LOCATION TYPE

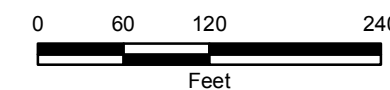
-  GEOPROBE
-  MONITORING WELL
-  SOIL BORING
-  SUB SLAB
-  SURFACE SOIL
-  SURFACE WATER/SEDIMENT
-  TAR
-  TEST PIT SAMPLE
-  TEST PIT

EXPOSURE AREAS

-  I-690 DRAINAGE DITCH
 EAST FLUME
 RAILROAD AREA
 LAKESHORE AREA
 PENN-CAN PROPERTY

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

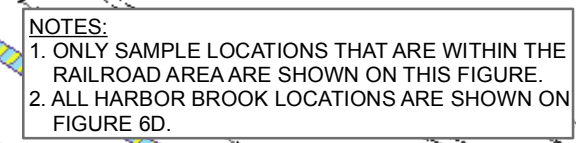
PENN-CAN PROPERTY SAMPLE LOCATIONS



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4

LOCATION TYPE

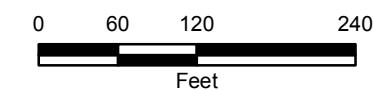
-  GEOPROBE
 MONITORING WELL
 SOIL BORING
 SURFACE SOIL
 SURFACE WATER/SEDIMENT
 TEST PIT SAMPLE
 TEST PIT

EXPOSURE AREAS

-  I-690 DRAINAGE DITCH
 EAST FLUME
 RAILROAD AREA
 LAKESHORE AREA
 PENN-CAN PROPERTY

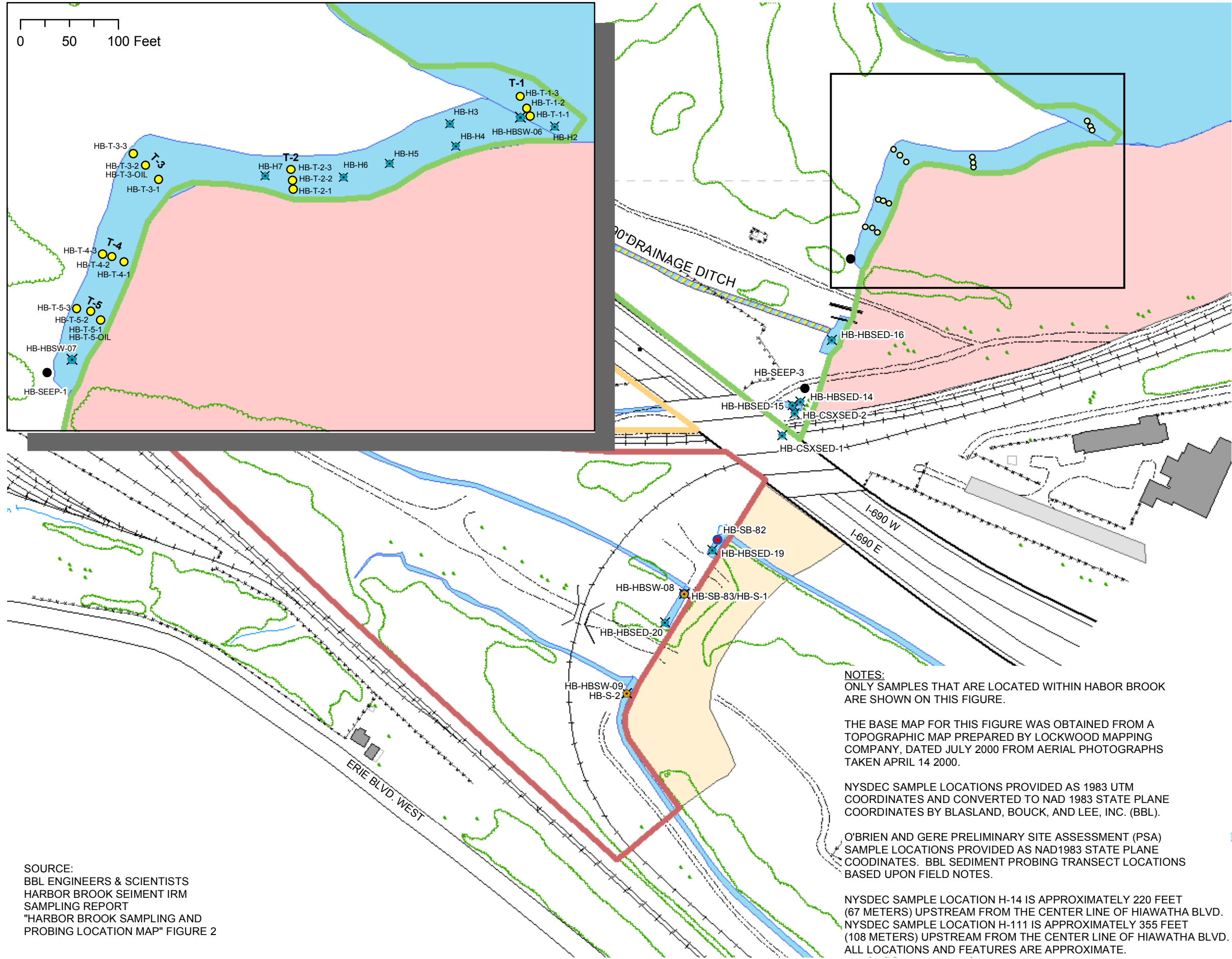
HONEYWELL
WASTEBED B/ HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

RAILROAD AREA SAMPLE LOCATIONS



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SOURCE:
BBL ENGINEERS & SCIENTISTS
HARBOR BROOK SEIMENT IRM
SAMPLING REPORT
"HARBOR BROOK SAMPLING AND
PROBING LOCATION MAP" FIGURE 2

NOTES:
ONLY SAMPLES THAT ARE LOCATED WITHIN HARBOR BROOK
ARE SHOWN ON THIS FIGURE.

THE BASE MAP FOR THIS FIGURE WAS OBTAINED FROM A
TOPOGRAPHIC MAP PREPARED BY LOCKWOOD MAPPING
COMPANY, DATED JULY 2000 FROM AERIAL PHOTOGRAPHS
TAKEN APRIL 14 2000.

NYSDEC SAMPLE LOCATIONS PROVIDED AS 1983 UTM
COORDINATES AND CONVERTED TO NAD 1983 STATE PLANE
COORDINATES BY BLASLAND, BOUCK, AND LEE, INC. (BBL).

O'BRIEN AND GERE PRELIMINARY SITE ASSESSMENT (PSA)
SAMPLE LOCATIONS PROVIDED AS NAD1983 STATE PLANE
COORDINATES. BBL SEDIMENT PROBING TRANSECT LOCATIONS
BASED UPON FIELD NOTES.

NYSDEC SAMPLE LOCATION H-14 IS APPROXIMATELY 220 FEET
(67 METERS) UPSTREAM FROM THE CENTER LINE OF HIAWATHA BLVD.
NYSDEC SAMPLE LOCATION H-111 IS APPROXIMATELY 355 FEET
(108 METERS) UPSTREAM FROM THE CENTER LINE OF HIAWATHA BLVD.
ALL LOCATIONS AND FEATURES ARE APPROXIMATE.

FIGURE 6D



LEGEND

HISTORIC SAMPLE TYPES

- BBL SEDIMENT CORE SAMPLE
- ✕ BBL SEDIMENT SAMPLING LOCATION FOR IRM

SAMPLE TYPE

- SEDIMENT BORING
- ✕ SURFACE WATER/SEDIMENT
- SEEP

EXPOSURE AREA

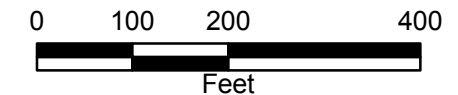
- Railroad Area
- Lakeshore Area
- Penn-Can Property
- ADDITIONAL AREA OF STUDY #1
- ADDITIONAL AREA OF STUDY #2
- I-690 DRAINAGE DITCH

SITE FEATURES

- TREE LINE
- DRAINAGE
- FENCELINE
- UNPAVED ACCESS ROAD
- PAVED ROAD
- HIGHWAY
- RAILROAD
- EXISTING BUILDING
- PAVED PARKING/DRIVEWAY

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT

HARBOR BROOK
SAMPLING LOCATIONS



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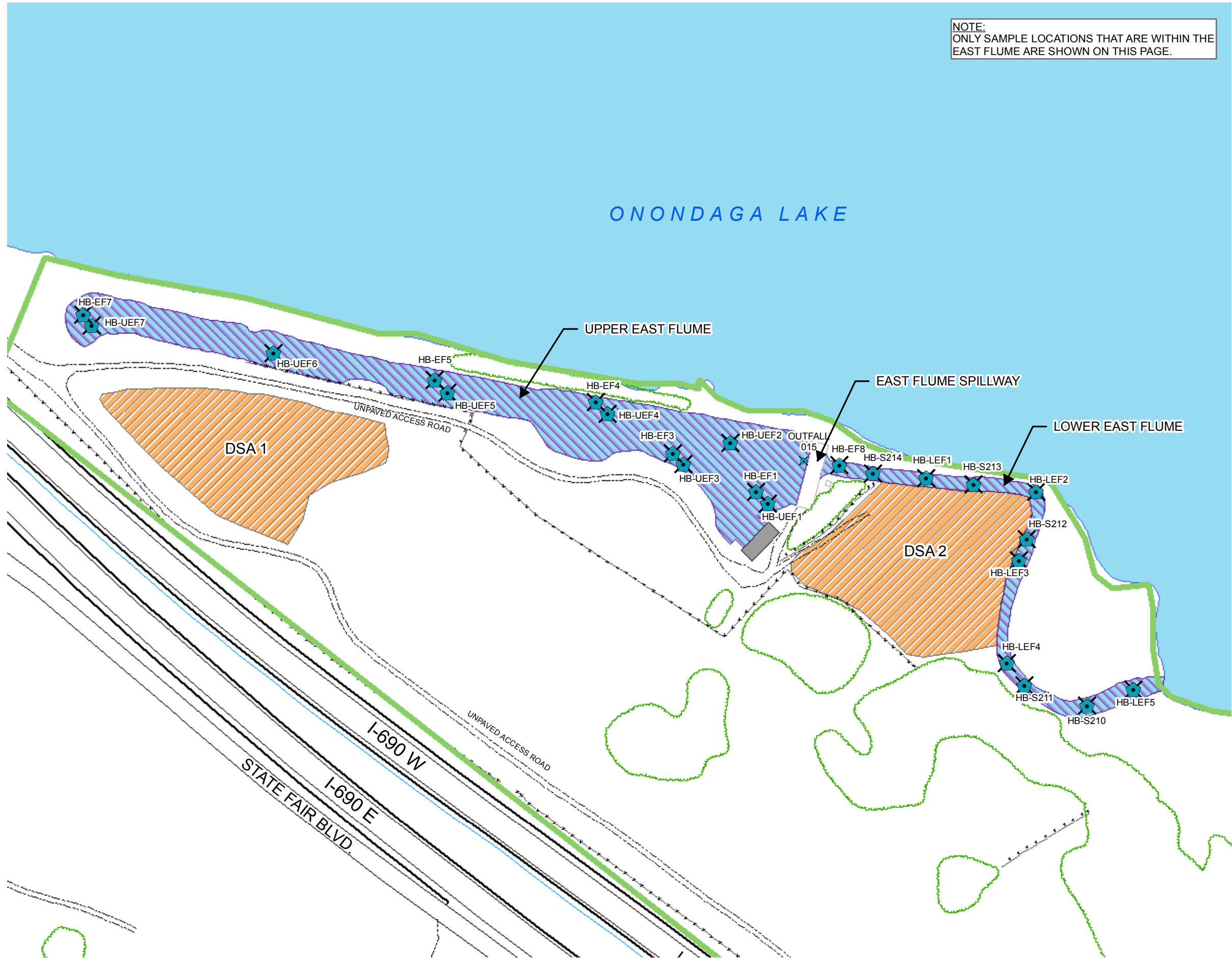


FIGURE 6E



LEGEND

SURFACE WATER/SEDIMENT

EXPOSURE AREA

LAKESHORE AREA

EAST FLUME

DREDGE SPOIL AREA

SITE FEATURES

TREE LINE

UNPAVED ACCESS ROAD

PAVED ROAD

HIGHWAY

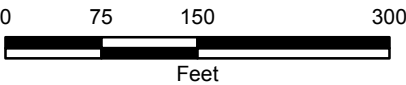
FENCELINE

RAILROAD

EXISTING BUILDING

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH
RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

EAST FLUME
SAMPLE LOCATIONS



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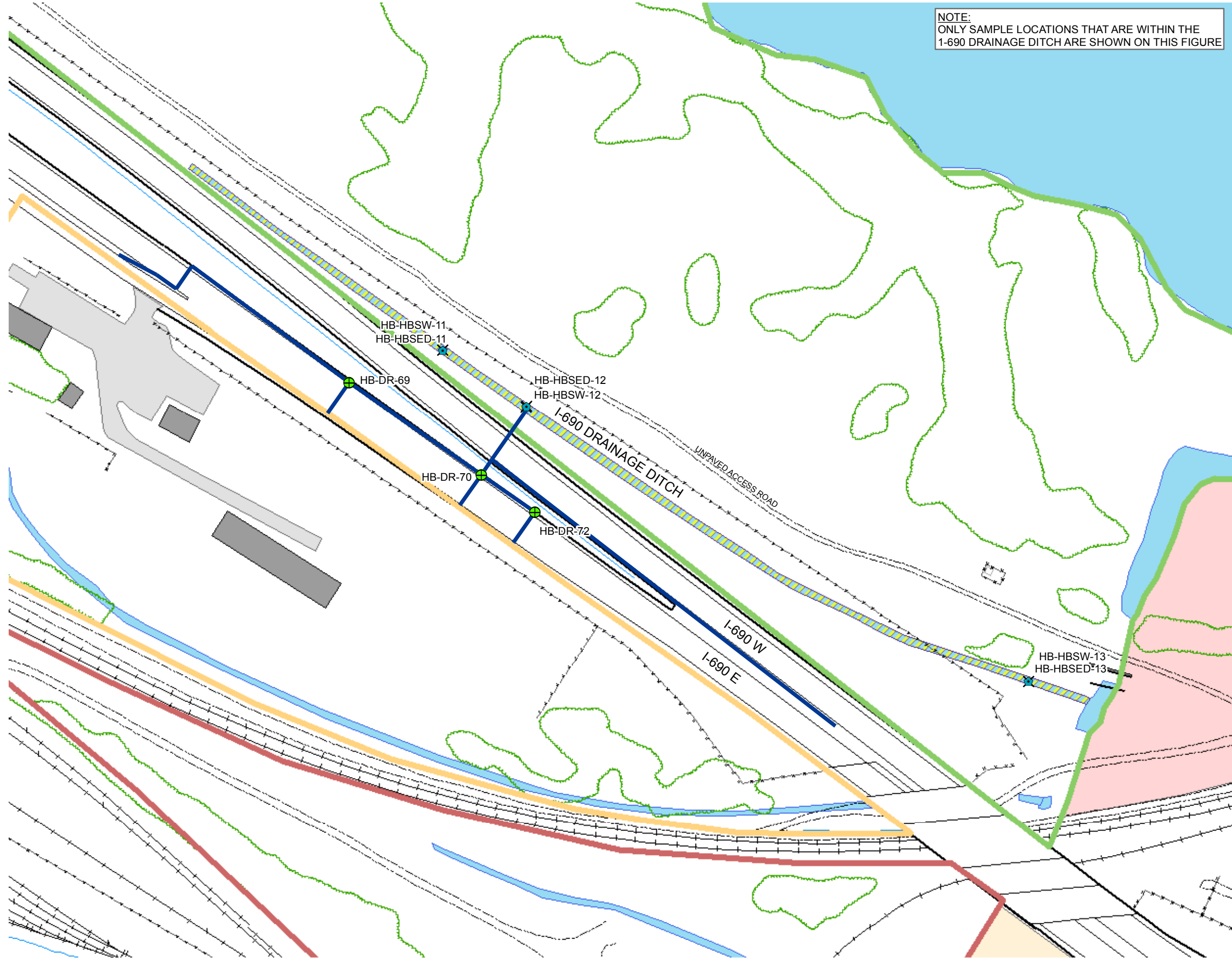


FIGURE 6F



LEGEND

LOCATION TYPE

- CATCH BASIN
- SURFACE WATER/SEDIMENT

EXPOSURE AREA

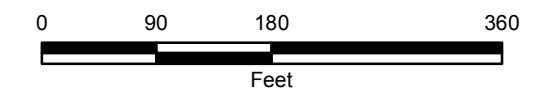
- RAILROAD AREA
- LAKESHORE AREA
- PENN-CAN PROPERTY
- EAST FLUME
- I-690 DRAINAGE DITCH
- DREDGE SPOIL AREA
- ADDITIONAL AREA OF STUDY #1
- ADDITIONAL AREA OF STUDY #2

SITE FEATURES

- TREE LINE
- UNPAVED ACCESS ROAD
- PAVED ROAD
- HIGHWAY
- RAILROAD
- EXISTING BUILDING
- PAVED PARKING/DRIVEWAY

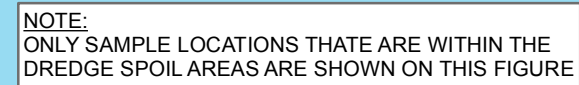
HONEYWELL
WASTEBED B/ HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

I-690 DRAINAGE DITCH
SAMPLE LOCATIONS



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4

LEGEND








LOCATION TYPE

-  MONITORING WELL
 SURFACE SOIL
 TEST PIT SAMPLE
 TEST PIT

EXPOSURE AREA

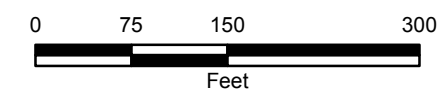
-  LAKESHORE AREA
 EAST FLUME
 DREDGE SPOIL AREA

SITE FEATURES

-  TREE LINE
 UNPAVED ACCESS ROAD
 PAVED ROAD
 HIGHWAY
 FENCELINE
 RAILROAD
 EXISTING BUILDING

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH
RISK ASSESSMENT
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DREDGE SPOIL AREA 1 AND 2 SAMPLE LOCATIONS



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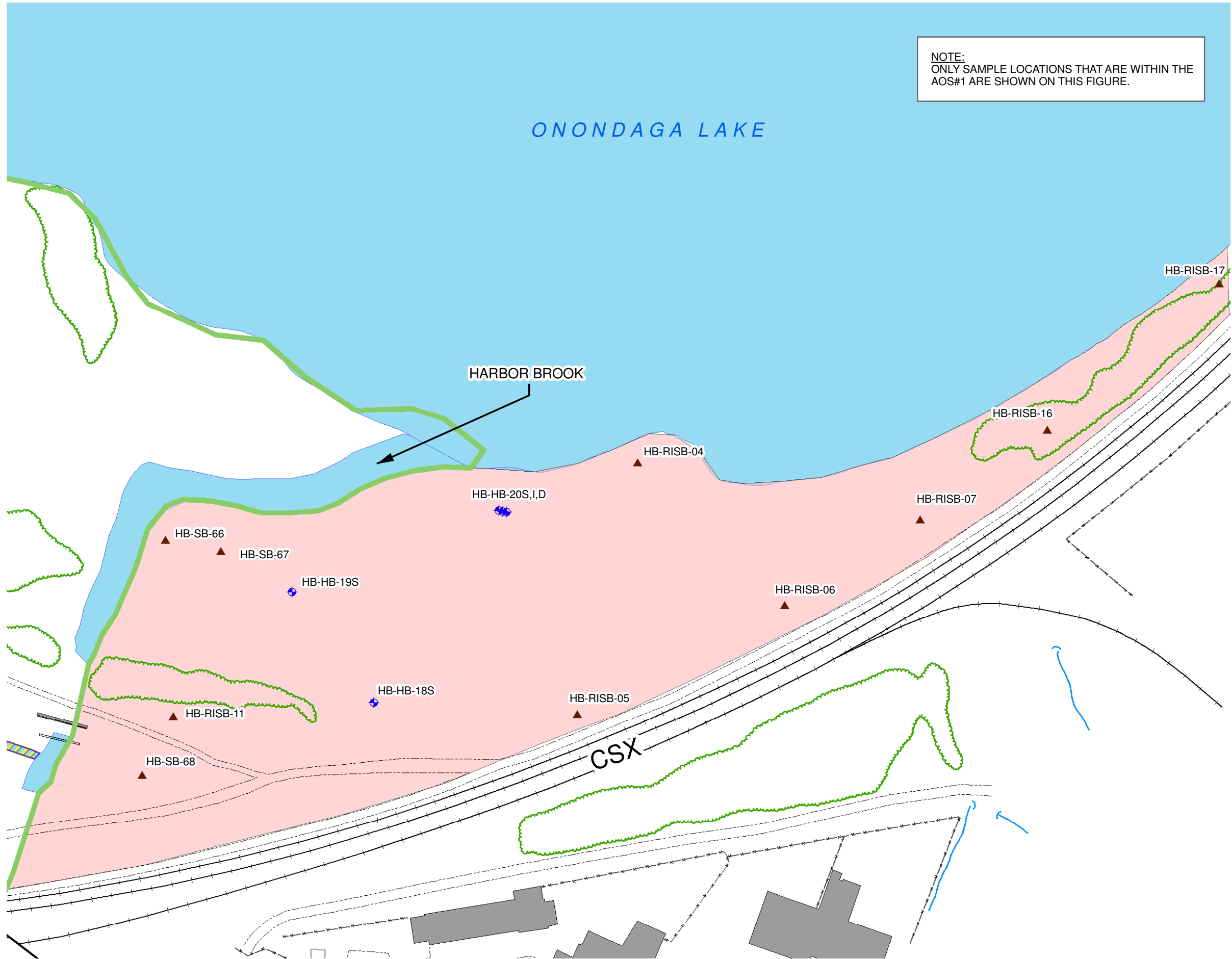


FIGURE 6H

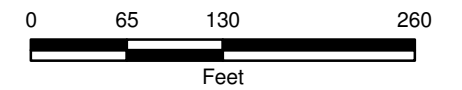


LEGEND

- LOCATION TYPE**
- MONITORING WELL
 - SOIL BORING
- EXPOSURE AREA**
- LAKESHORE AREA
 - ADDITIONAL AREA OF STUDY #1
 - I-690 DRAINAGE DITCH
- SITE FEATURES**
- TREE LINE
 - UNPAVED ACCESS ROAD
 - PAVED ROAD
 - HIGHWAY
 - FENCELINE
 - RAILROAD
 - EXISTING BUILDING

HONEYWELL
WATEBEDS B/ HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

ADDITION AREA
OF STUDY #1
SAMPLE LOCATIONS



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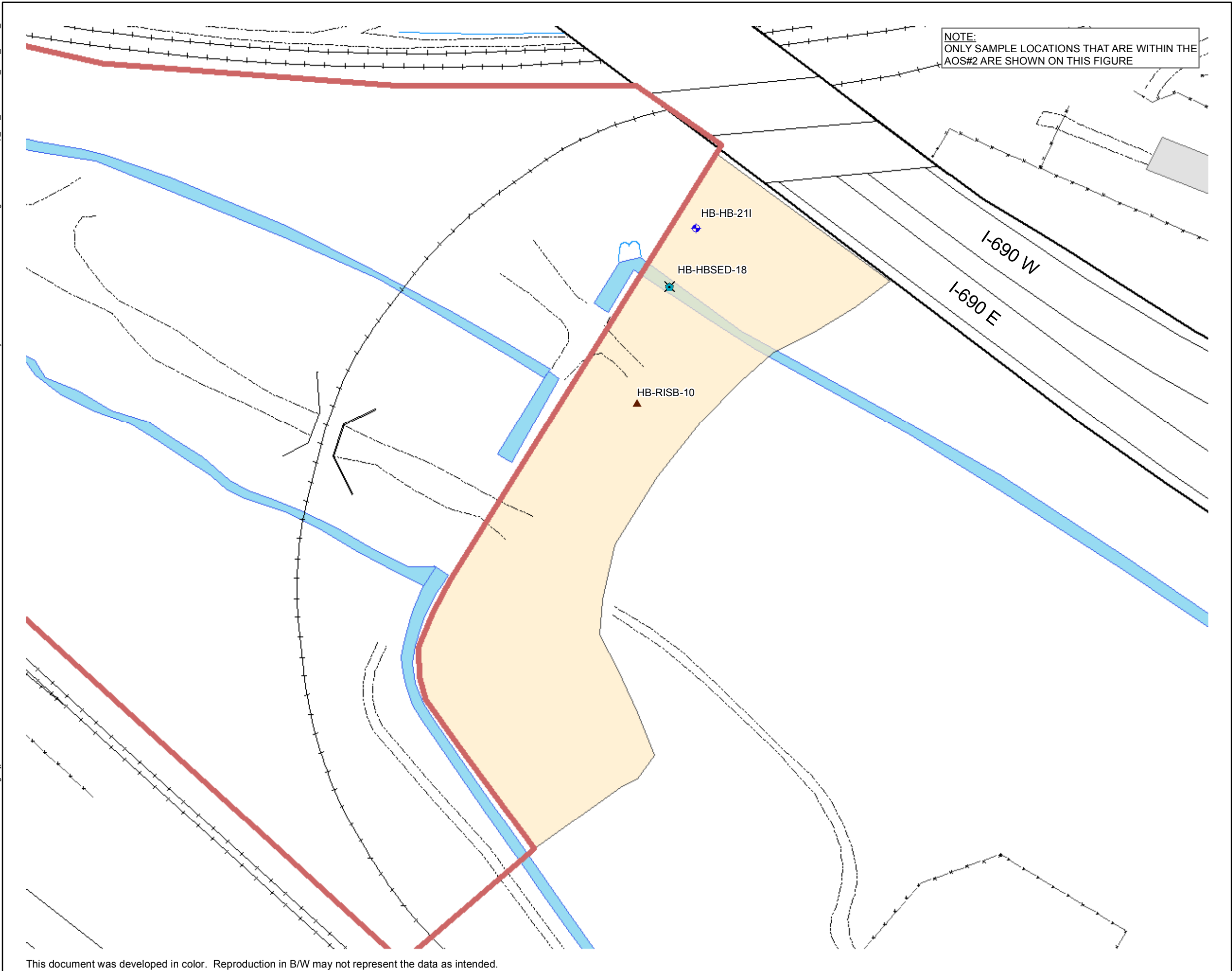


FIGURE 6I



LEGEND

LOCATION TYPE

- MONITORING WELL
- SOIL BORING
- SURFACE WATER/SEDIMENT

EXPOSURE AREA

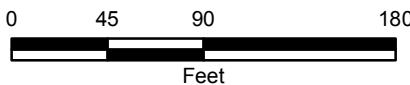
- RAILROAD AREA
- ADDITIONAL AREA OF STUDY #2

SITE FEATURES

- TREE LINE
- UNPAVED ACCESS ROAD
- PAVED ROAD
- HIGHWAY
- PAVED PARKING/DRIVEWAY
- RAILROAD

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

ADDITIONAL AREA
OF STUDY #2
SAMPLE LOCATIONS

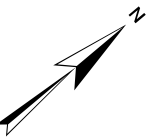


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FIGURE 7



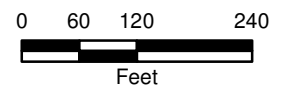
LEGEND

SAMPLED LOCATIONS

- WETLAND SOIL BORING/GW SCREENING
- MONITORING WELL
- WETLAND SOIL BORING
- WETLAND SEDIMENT
- SYW-12 SITE BORDER

HONEYWELL
WASTEBED B/HARBOR BROOK
HUMAN HEALTH RISK ASSESSMENT
GEDDES AND SYRACUSE, NY

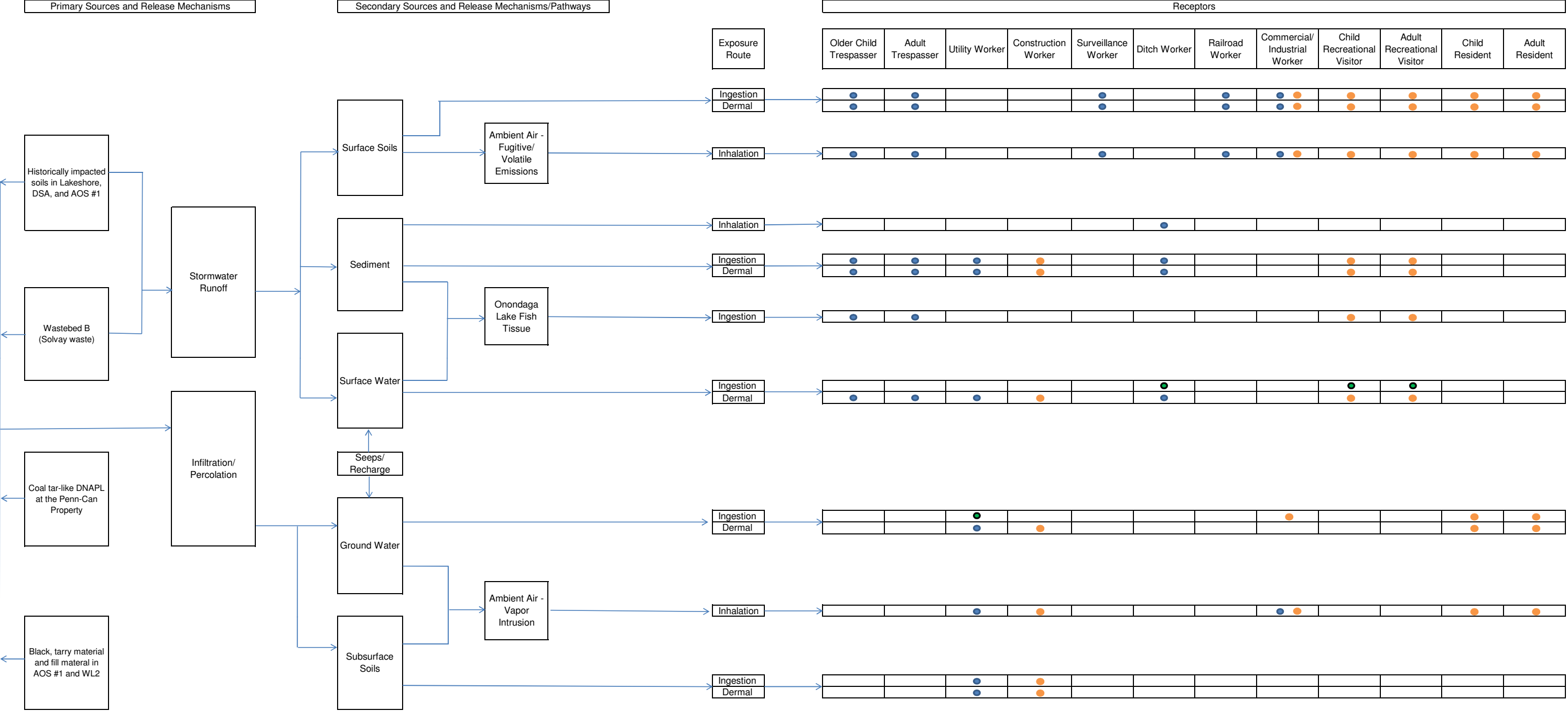
SYW-12
SAMPLE LOCATIONS



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FIGURE 8
CONCEPTUAL SITE MODEL
SITE WIDE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK



Notes:

- Receptor pathway present in future scenarios.
- Receptor pathway present in current/future scenarios.
- Potentially complete pathway but not evaluated in the HHRA because exposure is expected to be *de minimis*.
- Blank Incomplete pathway.

**APPENDICES A through C and H
(Electronic –CD Included)**

Appendix A – Site Data Set

**Appendix B – Sample Locations
Summary**

**Appendix C – Pro-UCL Version 4.0
Output**

**Appendix H – EPA Toxicity Value
Requests Correspondence**



TOXICOLOGICAL REVIEW

OF

2-HEXANONE

(CAS No. 591-78-6)

**In Support of Summary Information on the
Integrated Risk Information System (IRIS)**

February 2008

NOTICE

This document is an **External Review draft**. This information is distributed solely for the purpose of pre-dissemination peer review under applicable information quality guidelines. It has not been formally disseminated by EPA. It does not represent and should not be construed to represent any Agency determination or policy. It is being circulated for review of its technical accuracy and science policy implications.

U.S. Environmental Protection Agency
Washington, DC

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LIST OF ACRONYMS AND ABBREVIATIONS

AIC	Akaike Information Criterion
AVEP	average visual evoked potential
BMC	benchmark dose concentration
BMCL	benchmark concentration, lower 95% confidence limit
BMD	benchmark dose
BMDL	benchmark dose, lower 95% confidence limit
BMDS	benchmark dose software
BMR	benchmark response
CASRN	Chemical Abstracts Service Registry Number
CNS	central nervous system
CYP	cytochrome
CYP450	cytochrome P450
EEG	electroencephalogram
EPA	Environmental Protection Agency
EPN	O-ethyl O-4-nitrophenyl phenylphosphonothioate
EROD	ethoxyresorufin O-deethylase
GD	gestational day
HEC	human equivalent concentration
HSDB	Hazardous Substances Data Bank
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
LD₅₀	median lethal dose
LOAEL	lowest-observed-adverse-effect level
MAP	muscle action potential
MCV	motor (nerve) conduction velocity
MEK	methyl ethyl ketone
MiBK	methyl isobutyl ketone
NLM	National Library of Medicine
NOAEL	no-observed-adverse-effect level
NRC	National Research Council
PBTK	physiologically based toxicokinetic
PNS	peripheral nervous system
POD	point of departure
PROD	pentoxyresorufin O-depentylase
RfC	reference concentration
RfD	reference dose
s.c.	subcutaneous
TLV	threshold limit value
TSO	toluene side-chain oxidase
UF	uncertainty factor
w/w	weight/weight

FOREWORD

The purpose of this Toxicological Review is to provide scientific support and rationale for the hazard and dose-response assessment in IRIS pertaining to chronic exposure to 2-hexanone. It is not intended to be a comprehensive treatise on the chemical or toxicological nature of 2-hexanone.

The intent of Section 6, *Major Conclusions in the Characterization of Hazard and Dose Response*, is to present the major conclusions reached in the derivation of the reference dose, reference concentration, and cancer assessment, where applicable, and to characterize the overall confidence in the quantitative and qualitative aspects of hazard and dose response by addressing the quality of data and related uncertainties. The discussion is intended to convey the limitations of the assessment and to aid and guide the risk assessor in the ensuing steps of the risk assessment process.

For other general information about this assessment or other questions relating to IRIS, the reader is referred to EPA's IRIS Hotline at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

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1. INTRODUCTION

This document presents background information and justification for the Integrated Risk Information System (IRIS) Summary of the hazard and dose-response assessment of 2-hexanone. IRIS Summaries may include oral reference dose (RfD) and inhalation reference concentration (RfC) values for chronic and less-than-lifetime exposure durations, and a carcinogenicity assessment.

The RfD and RfC, if derived, provide quantitative information for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action. The RfD (expressed in units of mg/kg-day) is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The inhalation RfC (expressed in units of mg/m³) is analogous to the oral RfD, but provides a continuous inhalation exposure estimate. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrapulmonary or systemic effects). Reference values are generally derived for chronic exposures (up to a lifetime), but may also be derived for acute (#24 hours), short-term (greater than 24 hours to 30 days), and subchronic (greater than 30 days to 10% of average lifetime) exposure durations, all of which are derived based on an assumption of continuous exposure throughout the duration specified. Unless specified otherwise, the RfD and RfC are derived for chronic exposure duration.

The carcinogenicity assessment provides information on the carcinogenic hazard potential of the substance in question and quantitative estimates of risk from oral and inhalation exposure may be derived. The information includes a weight-of-evidence judgment of the likelihood that the agent is a human carcinogen and the conditions under which the carcinogenic effects may be expressed. Quantitative risk estimates may be derived from the application of a low-dose extrapolation procedure. If derived, the oral slope factor is an upper bound on the estimate of risk per mg/kg-day of oral exposure. Similarly, a unit risk is an upper bound on the estimate of risk per µg/m³ air breathed.

Development of these hazard identification and dose-response assessments for 2-hexanone has followed the general guidelines for risk assessment as set forth by the National Research Council (1983). EPA guidelines and technical reports that may have been used in the development of this assessment include the following: *Guidelines for Mutagenicity Risk Assessment* (U.S. EPA, 1986), *Guidelines for Developmental Toxicity Risk Assessment* (U.S. EPA, 1991), *Guidelines for Reproductive Toxicity Risk Assessment* (U.S. EPA, 1996), *Guidelines for Neurotoxicity Risk Assessment* (U.S. EPA, 1998a), *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), *Supplemental Guidance for Assessing Susceptibility from Early-*

Life Exposure to Carcinogens (U.S. EPA, 2005b), *Recommendations for and Documentation of Biological Values for Use in Risk Assessment* (U.S. EPA, 1988), *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994), *Use of the Benchmark Dose Approach in Health Risk Assessment* (U.S. EPA, 1995a), *Science Policy Council Handbook: Peer Review* (U.S. EPA, 2006, 2000a, 1998b), *Science Policy Council Handbook: Risk Characterization* (U.S. EPA, 2000b), and *Benchmark Dose Technical Guidance Document* (U.S. EPA, 2000c).

The literature search strategy employed for this compound was based on the Chemical Abstracts Registry Service Number (CASRN) and at least one common name. Any pertinent scientific information submitted by the public to the IRIS Submission Desk was also considered in the development of this document. The relevant literature was reviewed through March 2007.

2. CHEMICAL AND PHYSICAL INFORMATION

Structurally, 2-hexanone consists of a keto group flanked by a methyl group and an n-butyl group (Figure 2-1). The compound is a colorless liquid with a characteristic acetone-like odor but more pungent (NLM, 2005). Synonyms for 2-hexanone include the following: methyl butyl ketone, methyl n-butyl ketone, butyl methyl ketone, MnBK, and propylacetone.

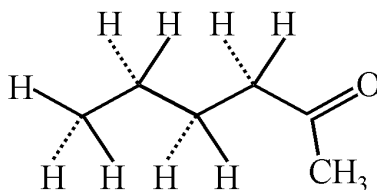


Figure 2-1. Chemical structure of 2-hexanone.

Pertinent physical and chemical properties of 2-hexanone are listed below (NLM, 2005).

Chemical formula	C ₆ H ₁₂ O
Molecular weight	100.16
Melting point	-55.5°C
Boiling point	127.6°C
Flash point	23°C
Density	0.8113 at 20°C
Water solubility	1.64 × 10 ⁴ mg/L at 20°C
Log K _{ow}	1.38
Vapor pressure	11.6 mm Hg at 25°C
Conversion factor	1 ppm = 4.1 mg/m ³ ; 1 mg/m ³ = 0.244 ppm

2-Hexanone is produced commercially by the catalyzed reaction of acetic acid and ethylene under pressure followed by distillation to purify the material (NLM, 2005). The compound has been used as a solvent for lacquers, ink thinners, nitrocellulose, resins, oils, fats, and waxes. It is a medium evaporating solvent for nitrocellulose acrylates, vinyl, and alkyd coatings (polyester coating derived from an alcohol and an acid or acid anhydride).

In 1977, the combined production and import of 2-hexanone in the U.S. was between 453 and 4500 metric tons (NLM, 2005); no breakdown of these figures was provided. The only U.S. producer of 2-hexanone, the Tennessee Eastman Company division of Eastman Kodak, discontinued production of 2-hexanone in 1979 and sold its remaining reserves by 1981 (NLM,

2005). 2-Hexanone is not produced or used in the U.S., and no information on importation is available (ATSDR, 1992).

3. TOXICOKINETICS

3.1. ABSORPTION

3.1.1. Pulmonary Absorption Studies

The available data indicate that 2-hexanone is well absorbed after administration via the inhalation route. DiVincenzo et al. (1978) exposed three healthy male volunteers (ages 22 to 53 years) to 2-hexanone (>97% pure, containing methyl isobutyl ketone [MiBK] and traces of 2-hexanol) at 10 or 50 ppm for 7.5 hours or 100 ppm for 4 hours. The 7.5-hour exposures were interrupted after 4 hours for a 0.5-hour lunch period. The volunteers were sedentary during the exposure. Expired air and venous blood samples were collected before, during, and after exposure. Exposures to 10 and 50 ppm for 7.5 hours produced mean 2-hexanone breath concentrations of 1.4 and 9.3 ppm, respectively. Fifteen minutes after exposure to 10 or 50 ppm, the expired air concentrations of 2-hexanone were 0.1 and 0.5 ppm, respectively. Exposure to 100 ppm for 4 hours produced an average 2-hexanone breath concentration of 22 ppm. These results indicated that between 75 and 92% of the inhaled 2-hexanone vapor was absorbed by the lungs and respiratory tract (DiVincenzo et al., 1978). 2-Hexanone was not detected in the expired air 3 hours after cessation of exposure to 50 or 100 ppm 2-hexanone.

DiVincenzo et al. (1978) exposed four young male beagles to 2-hexanone (>97% pure, containing MiBK and traces of 2-hexanol) for 6 hours at concentrations of 50 or 100 ppm. Over the first 4 hours of the exposure period, the hexanone in exhaled air had time-weighted average concentrations of 16 and 35 ppm for the low and high exposure groups, respectively. Thirty minutes after cessation of exposure to 50 ppm 2-hexanone, the breath concentration of 2-hexanone decreased to 0.7 ppm. 2-Hexanone was below the limit of detection by 3 to 5 hours after the exposure. It was determined that about 65–68% of the inhaled vapor was absorbed by the lungs.

3.1.2. Gastrointestinal Tract Absorption Studies

2-Hexanone appears to be well absorbed after oral administration. DiVincenzo et al. (1978) administered 2 μ Ci of 1- 14 C]-hexanone dissolved in corn oil via a gelatin capsule to human volunteers; the total dose was 0.1 mg/kg. Most of the 2-hexanone-derived radioactivity was exhaled as 14 CO₂, reaching a peak within 4 hours of dosing and then decreasing slowly over the next 3 to 5 days. The major portion of radioactivity excretion in urine occurred during the first 48 hours but continued at measurable levels until 8 days after dosing. The cumulative 8-day elimination of radioactivity in breath and urine averaged 39.5 and 26.3%, respectively. The overall recovery of 14 C was 65.8%. The authors presumed that the remainder of the radioactivity was retained in tissue or fat deposits.

Administration of 1-[¹⁴C]-2-hexanone at 20 or 200 mg/kg by gavage to rats resulted in excretion of about 1.1% of the administered radioactivity in the feces, about 44% in the breath, and 38% in urine, with about 15% remaining in the carcass after 48 hours and 8% remaining after 6 days (DiVincenzo et al., 1977). The results were similar at either dose level. These findings suggest that about 98% of the administered dose was absorbed via the gastrointestinal tract.

3.1.3. Dermal Absorption Studies

2-Hexanone is also absorbed after dermal application. DiVincenzo et al. (1978) exposed six human volunteers (ages 30–53 years) to radiolabeled 1-[¹⁴C]-2-hexanone (>97% purity, contaminants not stated). The labeled compound was applied to the ventral surface of the forearm that had been shaved 24 hours prior to testing. 1-[¹⁴C]-2-hexanone was held in contact with the skin for 60 minutes, and precautions were taken to ensure that inhalation exposure did not occur. The surface area of the skin subjected to solvent was 55.6 cm². Calculated skin absorption rates were 4.8 and 8.0 µg/cm²-minute. The quantities of 2-hexanone absorbed by two volunteers were 15.96 and 26.81 mg, respectively. The major respiratory excretion metabolite of 1-[¹⁴C]-hexanone was ¹⁴CO₂. A substantial portion of the dose was also excreted in urine; however, the chemical nature of urinary radioactivity was not characterized further.

In a similar set of experiments, DiVincenzo et al. (1978) applied 1-[¹⁴C]-2-hexanone (>97% purity, impurities not stated) to the clipped thorax (55.6 cm²) of beagles. Exposures were carried out for 5 minutes to 1 hour. By 5 minutes, 11 mg of 2-hexanone had penetrated the skin, and there was no apparent change in the absorption of 2-hexanone during the next 15 minutes. However, after 20 minutes the absorption increased markedly so that, by 60 minutes, 77 mg of 2-hexanone had penetrated the skin. The 8-hour cumulative excretion of radioactivity in two dogs dosed with 1-[¹⁴C]-2-hexanone was 0.5% of the dose as unchanged 2-hexanone and 9.7% as ¹⁴CO₂ in the breath; urinary radioactivity amounted to 6.5% of the dose. The 8-hour excretion of radioactivity averaged 16.8% of the dose. The fraction of the applied 2-hexanone dose that was absorbed was not calculated.

O'Donoghue and Krasavage (1981) exposed two male beagles (one of which was pretreated with 2-hexanone) to 2-hexanone by tail dipping. Both dogs were exposed to 2-hexanone on an area of 22 cm² on the first day of exposure, and then the exposure area was doubled on the second day (44.1 cm²). It was found that by 8–12 minutes, both dogs had comparable serum levels of 2-hexanone. Doubling the exposed area increased serum levels of 2-hexanone 6 to 20 times. None of the blood samples contained detectable levels of the 2-hexanone metabolites 5-hydroxy-2-hexanone, 2,5-hexanedione, or 2,5-hexanediol. Similar exposures were repeated with three different dogs for 16 minutes followed by two post exposure samples 9 and 19 minutes later (25 and 35 minute samples, respectively). One animal had detectable levels of 2-hexanone in blood within 4 minutes, but the time to detectable levels was

highly variable among the animals. The highest level observed was 3.2 µg/mL. Nineteen minutes post exposure serum levels of 2-hexanone were still detectable. Twenty-four hours later, no 2-hexanone was detected (O'Donoghue and Krasavage, 1981).

To examine the effects of multiple exposures, O'Donoghue and Krasavage (1981) exposed three male dogs as above to 2-hexanone on two occasions 4 hours apart. Samples obtained after the second treatment were not significantly different from the morning samples, indicating the absence of accumulation of detectable 2-hexanone and 2,5-hexanedione levels in the serum.

O'Donoghue and Krasavage (1981) performed comparison studies on percutaneous absorption of 2-hexanone between dog and rabbit skin. Significantly more 2-hexanone was absorbed through rabbit skin compared with dog skin and probably, as a consequence, the metabolite 5-hydroxy-2-hexanone was detected in the serum of rabbits. Overall, the skin studies indicated that 2-hexanone was readily absorbed through the skin; detectable serum levels were present after approximately 10 minutes of exposure to less than 1% of body skin surface; detectable serum levels persisted for approximately 20 minutes post exposure; and, in rabbits, a metabolite (5-hydroxy-2-hexanone) was rapidly formed and detectable in the serum.

3.2. DISTRIBUTION

Duguay and Plaa (1995) treated male Sprague-Dawley rats by gavage with 2-hexanone (>99%, spectrophotometric grade) at 0.5, 1, or 2 mmol/kg (50, 100, or 200 mg/kg) in corn oil (dose volume 10 mL/kg), once daily for 3 days. The animals were sacrificed 1 hour after the last gavage. Dose-dependent increases in plasma and lung 2-hexanone levels were observed, whereas the concentration in the liver increased only with the highest dose (Table 3-1). Calculations for statistically significant differences among dose groups were not performed (Duguay and Plaa 1995).

Table 3-1. Concentrations of 2-hexanone in plasma, liver, and lung of male rats following oral exposure for 3 days

Tissue concentration	Dose		
	0.5 mmol/kg	1 mmol/kg	2 mmol/kg
Plasma (µg/mL)	2.4 ± 1.2	4.7 ± 1.1	8.5 ± 2.0
Liver (µg/g)	1.7 ± 0.5	1.6 ± 0.3	3.8 ± 1.2
Lung (µg/g)	1.1 ± 0.7	4.9 ± 1.1	13.9 ± 4.9

Source: Duguay and Plaa (1995).

In a parallel series of experiments from the same study, Duguay and Plaa (1995) exposed male Sprague-Dawley rats to a total body exposure of 2-hexanone at concentrations of 75, 150, or 300 ppm (307.5, 615, or 1230 mg/m³). Animals were exposed on 3 consecutive days for 4 hours per day. Animals were sacrificed immediately after the last exposure on the third day.

The concentration of 2-hexanone in plasma, liver, and lung increased in a dose-dependent manner (Table 3-2). It should be noted, however, that because whole body exposures were performed, some oral and dermal absorption may have taken place.

Table 3-2. Concentrations of 2-hexanone in plasma, liver, and lung of male rats following inhalation exposure for 3 days

Tissue concentration	Dose		
	75 ppm	150 ppm	300 ppm
Plasma (µg/mL)	1.2 ± 0.3	2.6 ± 0.7	9.7 ± 0.7
Liver (µg/g)	0.7 ± 0.5	1.2 ± 0.8	2.2 ± 0.4
Lung (µg/g)	0.7 ± 0.2	2.8 ± 0.5	9.3 ± 1.2

Source: Duguay and Plaa (1995).

In male CD/COBS rats administered a single gavage dose of [¹⁴C]-2-hexanone at 200 mg/kg, the serum elimination for 2-hexanone was 6 hours; the 2-hexanone metabolites 5-hydroxy-2-hexanone and 2,5-hexanedione were eliminated from serum within 12 and 16 hours, respectively (DiVincenzo et al., 1977). Peak concentrations of 2-hexanone and 5-hydroxy-2-hexanone were reached at 2 hours, whereas the peak concentration of 2,5-hexanone was reached at 6 hours. Radioactivity was detected in most tissues with highest counts in liver > kidney > whole brain. The peak concentration of radiolabel in each of these tissues was observed at 4 hours and was reduced to less than 50% by 24 hours. No quantitative data were given on tissue distribution. An analysis of the subcellular distribution of the ¹⁴C-label in liver, brain, and kidney tissue homogenates indicated the highest counts were associated with the protein fraction, with some recovery from DNA and little or none from RNA.

Eben et al. (1979) treated male SPF-Wistar rats with 400 mg/kg 2-hexanone (98% pure, impurities not stated) daily by stomach tube for 40 weeks. The concentrations of 2-hexanone and metabolites in the blood were determined at intervals of 4 or 5 weeks. In the case of 2-hexanone, the maximum concentration was reached 1 hour after administration throughout the study; thereafter, the concentration decreased rapidly. After 7 hours, only trace amounts could be detected. During the first few weeks of the study, 2-hexanone could not be found in the urine. Only during the third week were very small concentrations of the free compound detected in urine, suggesting that the metabolic pathways for 2-hexanone were becoming saturated. A maximum (approximately 20 µg) was reached in the 17th week (Eben et al., 1979).

Granvil et al. (1994) studied the distribution and disappearance of 2-hexanone (purity not stated) from the blood and brain. Male CD-1 mice were treated with 5 mmol/kg (500 mg/kg) 2-hexanone dissolved in corn oil by intraperitoneal (i.p.) injection at a volume of 10 mL/kg. Animals were killed by decapitation, and blood and brain samples were collected at 15, 30, 60, and 90 minutes after treatment. Blood and brain concentrations at 15 minutes were ~182 µg/mL

and ~126 µg/g, respectively. By 90 minutes, the values had dropped in a uniform manner to a blood concentration of ~28 µg/mL and a brain concentration of ~25 µg/mg. The authors noted that the rapid decrease in the concentration of 2-hexanone was due to its active metabolism in these tissues (Granvil et al., 1994).

3.3. METABOLISM

2-Hexanone is hydroxylated to 5-hydroxy-2-hexanone, which is then either oxidized to 2,5-hexanedione or reduced to 2,5-hexanediol and, to a small extent, may be converted to 2,5-dimethyl-2,3-dihydrofuran. The predominant metabolite of 2-hexanone found in blood is 2,5-hexanedione. This can be reduced to 5-hydroxy-2-hexanone and further, but to a lesser extent, to 2,5-hexanediol. The formation of 2,5-hexanedione is favored over that of 5-hydroxy-2-hexanone. 5-Hydroxy-2-hexanone can be metabolized into 4,5-dihydroxy-2-hexanone (not shown in Figure 3-1) before being further converted to 2,5-dimethyl-2,3-dihydrofuran. Additionally, 4,5-dihydroxy-2-hexanone formation may be a result from 2,5-hexanedione metabolism (U.S. EPA, 2005c). Other mechanisms, such as shunting into intermediary metabolism, may accelerate metabolic clearance of 2,5-hexanedione. Reductive metabolism of 2-hexanone results in the formation of 2-hexanol, establishing an equilibrium between the two compounds. 2-Hexanol can be further metabolized to 2,5-hexanediol, 5-hydroxy-2-hexanone, and 2,5-hexanedione. The findings of Abdel-Rahman et al. (1976) that rats, guinea pigs, and rabbits exposed to 2-hexanone vapor excreted glucuronides of 2-hexanol and 2,5-hexanediol in the urine are consistent with the results by DiVincenzo et al. (1976), discussed later in this section (Abdel-Rahman et al., 1976). Although the proportions of metabolites may differ among species, ω-1-oxidation and carbonyl reduction appear to be the initial steps in the metabolism of 2-hexanone in all species tested so far (e.g., rat, cat, dog, guinea pig, and human). The metabolic pathway for 2-hexanone, as proposed by DiVincenzo et al. (1977, 1976), based on 2-hexanone metabolites identified in blood of guinea pigs, mice, and rats, is presented in Figure 3-1.

As discussed in Section 3.2, Duguay and Plaa (1995) conducted studies using male Sprague-Dawley rats exposed to 2-hexanone by gavage (0.5, 1, or 2 mmol/kg) or by inhalation (75, 150, or 300 ppm) and quantified the metabolites in the plasma, liver, and lung. The authors reported that the concentrations of metabolites, such as 2-hexanol, 5-hydroxy-2-hexanone, and 2,5-hexanedione, were readily detectable in serum. After 2-hexanone gavage or inhalation, 2-hexanol was found in low concentrations in plasma and liver (0.5–1.3 µg/mL and 0.3–1.6 µg/g, respectively). In lung, however, concentrations ranged from 2.1 to 5.1 µg/g. However, with both routes of administration, 2-hexanol concentrations did not appear to be dose dependent.

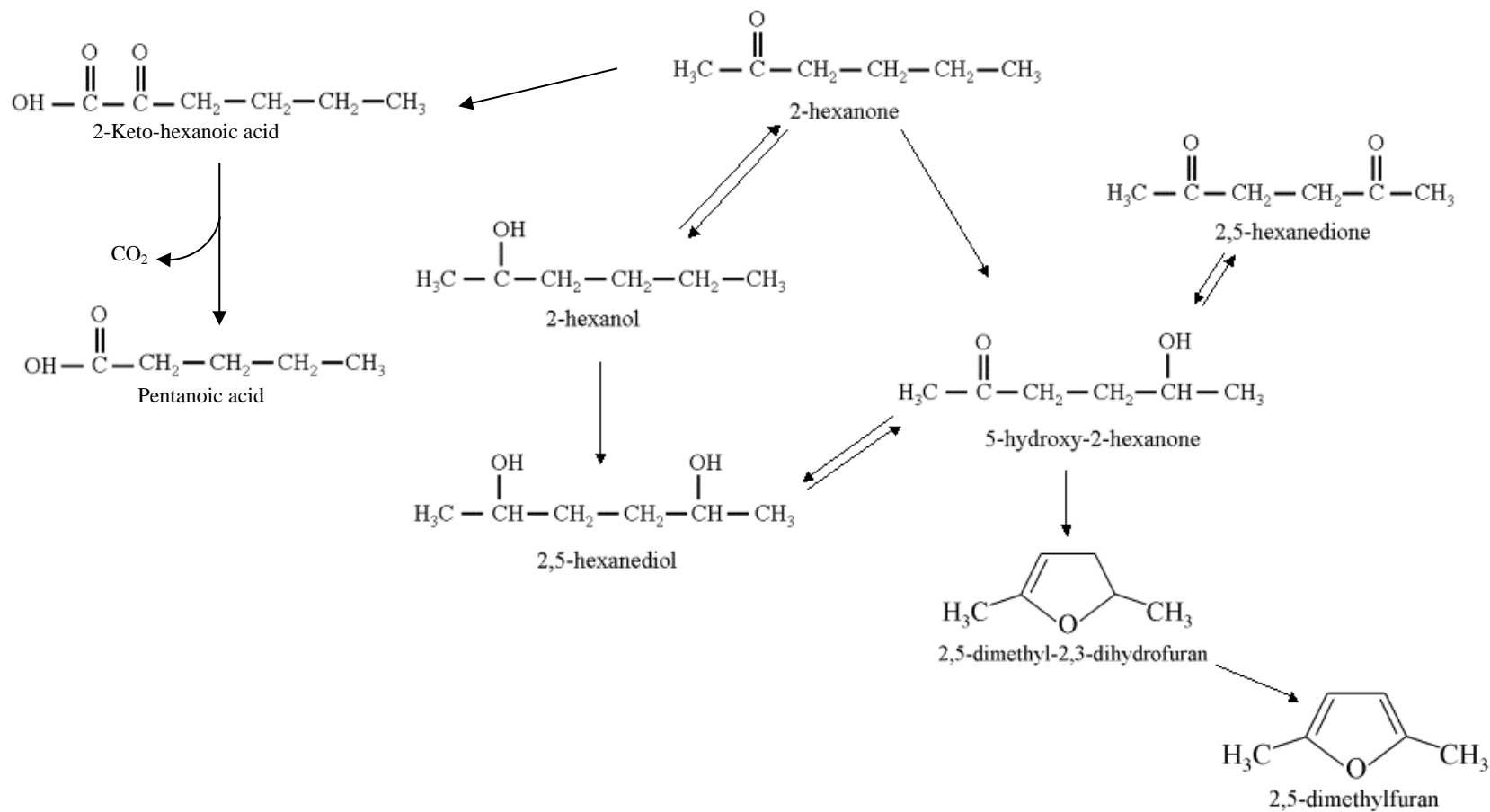


Figure 3-1. Proposed metabolic pathway for 2-hexanone.

Adapted from DiVincenzo et al. (1977, 1976).

The appearance of the 2-hexanone metabolite 2,5-hexanedione in plasma or lung, but not in liver, depended on the route of administration. The highest dose and concentration of 2-hexanone, 2 mmol/kg and 300 ppm, respectively, produced similar plasma 2-hexanone concentrations, 8.5 and 9.7 µg/mL, respectively, but the corresponding plasma 2,5-hexanedione concentrations were 7.7 µg/mL after oral and 25 µg/mL after inhalation administration. 2,5-Hexanedione was not detectable in lungs when 2-hexanone was administered orally, but significant dose-dependent amounts were found following inhalation exposure. The authors concluded that pulmonary 2-hexanone metabolism might contribute to plasma metabolite levels. 2,5-Hexanedione concentrations in liver were dose dependent but independent of the route of administration. A summary of the metabolite levels found in the plasma, liver, and lung following oral and inhalation exposures is presented in Table 3-3.

Table 3-3. 2-Hexanol and 2,5-hexanedione in the plasma, liver, and lung of male rats after oral or inhalation exposure to 2-hexanone

	Dose					
	0.5 mmol/kg (gavage) ^a	75 ppm (inhalation) ^a	1 mmol/kg (gavage) ^a	150 ppm (inhalation) ^a	2 mmol/kg (gavage) ^a	300 ppm (inhalation) ^a
<i>Plasma^b</i>						
2-HOL ^c	0.6 ± 0.2	0.5 ± 0.1	1.2 ± 0.3	0.5 ± 0.2	1.3 ± 0.4	0.8 ± 0.3
2,5-HD ^d	5.7 ± 0.5	6.7 ± 0.8	5.8 ± 0.5	13.5 ± 2.1	7.7 ± 0.4	25 ± 3.1
<i>Liver^e</i>						
2-HOL	0.4 ± 0.1	0.4 ± 0.2	1.6 ± 0.5	0.3 ± 0.1	1.2 ± 0.5	0.3 ± 0.3
2,5-HD	3.1 ± 0.2	3.1 ± 0.1	4.6 ± 0.5	4.3 ± 0.6	5.3 ± 0.5	7.3 ± 0.3
<i>Lung^e</i>						
2-HOL	2.4 ± 0.5	2.1 ± 0.6	3.0 ± 1.0	2.9 ± 0.5	5.1 ± 1.5	3.7 ± 1.1
2,5-HD	ND ^f	0.9 ± 0.2	ND	4.0 ± 0.1	ND	4.8 ± 0.7

^aRats were sacrificed 1 hour after the last oral treatment but immediately after the last inhalation exposure.

Values are mean ± SE from six animals.

^bPlasma concentrations in µg/mL.

^c2-HOL = 2-hexanol.

^d2,5-HD = 2,5-hexanedione.

^eTissue concentrations in µg/g.

^fND = not detectable (<0.25 µg/mL).

Source: Duguay and Plaa (1995).

Eben et al. (1979) administered daily oral doses of 2-hexanone (400 mg/kg) over a 40-week period to male SPF-Wistar rats. The concentrations of 2-hexanone, 2-hexanol, and 2,5-hexanedione were determined in the blood at several intervals every 4 or 5 weeks. 2-Hexanone concentrations in blood peaked at 1 hour after administration then decreased rapidly, and after 7 hours only traces could be detected. The metabolite 2-hexanol was measurable in very small quantities up to 3 hours after administration (<2 µg/mL blood). In contrast, 2,5-hexanedione concentrations were relatively high as early as 1 hour after

administration, and maximum values were recorded after 5 or 7 hours. 2,5-Hexanediol was not detectable in the blood at any time. A summary of the blood concentrations of 2-hexanone, 2-hexanol, and 2,5-hexanedione is presented in Table 3-4.

Table 3-4. 2-Hexanone, 2-hexanol, and 2,5-hexanedione in the blood of male rats after repeated administration of 400 mg/kg-day

Week	2-Hexanone (µg/mL) ^a				2-Hexanol (µg/mL) ^a				2,5-Hexanedione (µg/mL) ^a			
	1 hour	3 hours	5 hours	7 hours	1 hour	3 hours	5 hours	7 hours	1 hour	3 hours	5 hours	7 hours
2	26.5	15.2	5.8	— ^b	—	—	—	—	19.8	53.3	65.7	53.8
6	30.4	21.4	7.3	1.4	0.6	0.8	—	—	10.9	46.5	59.7	59.8
10	20.2	7.5	4.7	3.8	0.7	—	—	—	16.7	39.2	60.7	64
14	31.8	25.7	6.3	2.2	1.7	1.2	traces	—	10	35.1	55.2	59.1
19	32.2	22.5	3.4	0.1	—	—	—	—	16.7	50.3	62.1	55
23	35.1	19.8	6.6	0.3	—	—	—	—	10.4	46.7	68.9	63.7
27	37.8	21.3	2.9	0.7	1.3	traces	—	—	8	38.8	49.9	49.2
32	24.8	12.2	2.9	0.3	0.6	0.1	—	—	8.4	31.8	41.1	34.8
36	50.1	13.4	7.1	1	1.5	0.1	—	—	14.6	47.4	55.6	56.1
40	33.4	18.9	3.6	0.4	1.2	0.9	—	—	12.5	36.8	51.9	66.2

^aValues represent the averages of three animals.

^bA dash (—) indicates that the compound was below the limit of detection.

Source: Eben et al. (1979).

Granvil et al. (1994) demonstrated the rapid removal of 2-hexanone from blood and brain of male CD-1 mice following a single i.p. injection of the compound at a concentration of 5 mmol/kg. The authors observed that 2-hexanol concentrations found in whole brain at several time intervals (15, 30, and 60 minutes after dosing) were about twice as high as those found in the blood at the same time intervals and interpreted this finding as suggesting that 2-hexanol might be formed in the brain. Furthermore, the authors reported that the appearance of the reduced metabolite 2-hexanol seemed to be considerably faster than the appearance of the oxidized metabolite 2,5-hexanedione.

DiVincenzo et al. (1976) administered a single dose of 2-hexanone (450 mg/kg i.p. in corn oil) to male guinea pigs (strain not stated). Blood was collected by heart puncture from four animals at 1, 2, 4, 6, 8, 12, and 16 hours after dosing. In addition to 2-hexanone, three major metabolites were identified by gas chromatography: 5-hydroxy-2-hexanone, 2,5-hexanedione, and 2-hexanol. 2,5-Dimethyl-2,3-dihydrofuran was also detected, but additional experiments revealed that this was an artifact because 5-hydroxy-2-hexanone underwent dehydration and cyclization in the gas chromatograph. The authors noted that 5-hydroxy-2-hexanone may be

transformed also in vivo to 2,5-dimethyl-2,3-dihydrofuran, but the equilibrium favors the formation of 5-hydroxy-2-hexanone.

DiVincenzo et al. (1976) also conducted follow-up studies to determine the metabolic fate of 2-hexanone metabolites in guinea pigs. Each of the principal metabolites identified in the above study (5-hydroxy-2-hexanone, 2,5-hexanedione, 2,5-hexanediol, and 2-hexanol) was administered individually (450 mg/kg i.p.). 5-Hydroxy-2-hexanone was further metabolized to 2,5-hexanedione and 2,5-hexanediol. The half-life of 5-hydroxy-2-hexanone in serum was 156 minutes. The major metabolite 2,5-hexanedione was formed rapidly, and its concentration in serum was equivalent to or greater than that of the parent compound (5-hydroxy-2-hexanone) in all samples measured. Serum concentrations of 2,5-hexanediol were markedly lower than those of 2,5-hexanedione. 5-Hydroxy-2-hexanone was the only metabolite detected in serum of guinea pigs after an i.p. injection of 2,5-hexanedione. The half-life of 2,5-hexanedione was 100 minutes. Both 5-hydroxy-2-hexanone and 2,5-hexanedione were no longer detectable in serum by 16 hours. The principal metabolites in serum after i.p. injection with 2,5-hexanediol were 5-hydroxy-2-hexanone and 2,5-hexanedione. 2,5-Hexanediol was cleared within 8 hours and had a half-life of 84 minutes in serum. The following metabolites were identified after the administration of 2-hexanol: 2-hexanone, 5-hydroxy-2-hexanone, 2,5-hexanedione, and 2,5-hexanediol. The half-life and clearance time of 2-hexanol were 72 minutes and 6 hours, respectively.

The authors noted that the 2-hexanol was rapidly metabolized to 2-hexanone, which, in turn, was converted to the same metabolites identified above for animals treated with 2-hexanone. They determined that the conversion of 2-hexanol to 2,5-hexanediol seemed to be a minor pathway. The metabolites, 2,5-hexanediol and 2,5-hexanedione, were cleared in 8 and 16 hours, respectively. A summary of the half-life and clearance time of 2-hexanone and metabolites is presented in Table 3-5.

Table 3-5. Serum half-lives and clearance times of 2-hexanone and its metabolites in guinea pigs

Compound administered	Half-life (minutes) ^a	Clearance time (hours)
2-Hexanone	78	6
2-Hexanol	72	6
5-Hydroxy-2-hexanone	156	8 ^b
2,5-Hexanedione	100	16
2,5-Hexanediol	84	8

^aHalf-lives were determined from the linear portion of the plasma concentration curve and extrapolated to zero time.

^bEstimated value.

Source: DiVincenzo et al. (1976).

Bus et al. (1981) presented metabolism data for n-hexane that provide some insight on the metabolism of 2-hexanone. In the study, the authors exposed male F344 rats for 1 or 5 days, 6 hours/day, to 1000 ppm n-hexane. Animals were sacrificed immediately after exposure or at increasing time intervals for up to 24 hours after the end of exposure, and concentrations of the parent compound and two of its metabolites, 2-hexanone and 2,5-hexanedione, were measured in blood, liver, kidney, brain, and sciatic nerve. Kinetics of all three compounds were similar after 1 and 5 days of exposure, with tissue levels of the metabolites frequently exceeding those of the parent compound even immediately after the end of exposure. Tissue levels of n-hexane and 2-hexanone were always lower after 5 days of repeated exposures, compared with levels after a single exposure, consistent with self-induction of some metabolizing enzymes. On the other hand, tissue levels of 2,5-hexanedione were always slightly higher after 5 days of exposure, compared with single exposure. A compilation of the data for 2-hexanone and 2,5-hexanedione after 5 days of exposure to n-hexane is given in Table 3-6 (sciatic nerve data not included).

Table 3-6. Tissue levels of 2-hexanone and 2,5-hexanedione in male F344 rats following inhalation exposure to n-hexane for 5 days

Time (hours)	Blood ^{a,b}		Liver ^{a,c}		Brain ^{a,c}		Kidney ^{a,c}	
	2-Hx ^d	2,5-HxD ^e	2-Hx	2,5-HxD	2-Hx	2,5-HxD	2-Hx	2,5-HxD
0	0.46 ± 0.07	1.97 ± 0.38	0.12 ± 0.01	0.56 ± 0.10	0.78 ± 0.04	5.66 ± 0.17	22.9 ± 3.81	11.8 ± 1.03
1	0.23 ± 0.02	6.02 ± 0.56	0.20 ± 0.04	0.64 ± 0.13	0.18 ± 0.01	7.41 ± 0.31	9.73 ± 1.14	23.5 ± 1.85
2	0.06 ± 0.03	3.99 ± 0.37	—	0.69 ± 0.01	0.08 ± 0.01	7.17 ± 0.74	4.80 ± 0.39	26.4 ± 1.61
4	— ^f	2.12 ± 0.26	—	0.15 ± 0.02	—	2.75 ± 0.34	0.63 ± 0.23	16.8 ± 3.67
8	—	0.54 ± 0.19	—	0.03 ± 0.03	—	—	0.67 ± 0.15	9.08 ± 2.45
12	—	—	—	—	—	—	0.78 ± 0.21	0.86 ± 0.27
24	—	—	—	—	—	—	0.28 ± 0.00	0.01 ± 0.01

^aMean ± SE, n = 3.

^bValues in µg/mL plasma.

^cValues in µg/g wet weight.

^d2-hexanone

^e2,5-hexanedione

^fDash (—) = below detection limit.

Source: Bus et al. (1981).

This experiment, although conducted with n-hexane as the parent compound, provides some insight into the metabolism of 2-hexanone. The data shown in Table 3-6 indicate that the metabolism of 2-hexanone to 2,5-hexanedione (intermediates not considered) proceeds rapidly, while the further metabolism of 2,5-hexanedione and its elimination appear to proceed much more slowly. Both the resurgence of 2-hexanone levels in kidney between 8 and 12 hours and the precipitous drop of 2,5-hexanedione levels in kidney between 8 and 12 hours occurred in the same fashion with single exposure, suggesting rather complex compartmentalization and

toxicokinetics that, to some extent, may be governed by the lipophilic characteristics of the compounds. The authors (Bus et al., 1981) suggested that the high levels observed in kidney for both metabolites, but not the parent compound, reflect the fact that the metabolites of n-hexane, and thus 2-hexanone, are mostly eliminated via urine.

Cytochrome P450 (CYP450) enzymes catalyze the initial steps (either detoxification or bioactivation) of 2-hexanone, but their identities have not been investigated to any great detail. Oral administration of 1-[¹⁴C]-2-hexanone to humans or rats resulted in the appearance of ¹⁴CO₂ in the exhaled breath, indicating removal of the α-carbon (DiVincenzo et al., 1978, 1977). Administration of SKF525A (a mixed function oxidase inhibitor) to rats before oral administration of 2-hexanone resulted in marked decrease in the respiratory excretion of ¹⁴CO₂ for the first 4 hours after administration, followed by a marked increase at 4–8 and 12–24 hours. This suggests that this oxidative step is mediated by a microsomal mixed-function oxidase system (DiVincenzo et al., 1977).

Because inhalation exposure of humans to 1-[¹⁴C]-2-hexanone resulted in the appearance of labeled carbon dioxide in expired air and 2,5-hexanedione in serum, DiVincenzo et al. (1978) hypothesized that the metabolic pathway for 2-hexanone is similar in humans and experimental animals. Metabolically, aliphatic ketones generally are in equilibrium with the corresponding secondary alcohols, which explains the presence of 2-hexanol. An alternate pathway is oxidation of the 5-methylene group to the corresponding alcohol, 5-hydroxy-2-hexanone. Another possibility in the metabolism of 2-hexanone is the cyclization of 5-hydroxy-2-hexanone to the corresponding dihydrofuran and oxidation to 2,5-dimethylfuran (DiVincenzo et al., 1977). However, the formation of these furan moieties may be an artifact resulting from thermal dehydration and cyclization during gas chromatography (DiVincenzo et al., 1977). In addition, the γ-valerolactone found in the urine was hypothesized to result from α-oxidation of 5-hydroxy-2-hexanone to 2-keto-5-hydroxyhexanoic acid, decarboxylation and oxidation to 4-hydroxypentanoic acid, and lactonization to γ-valerolactone (not shown in Figure 3-1) (DiVincenzo et al., 1977).

Although the specific isoforms of CYP450 that catalyze the metabolism of 2-hexanone have not been fully characterized, Nakajima et al. (1991) provided some insight into the effects of 2-hexanone on CYP450 induction. The authors treated male Wistar rats with 2-hexanone at 5 mmol/kg (500 mg/kg) i.p. for 4 days and demonstrated that various CYP450 isozyme activities were induced. 2-Hexanone was effective in inducing several CYP450 isoforms as indicated by the increase in activities of benzene aromatic hydroxylase (CYP2E1) and toluene side chain oxidation (CYP2C6/11) two- to threefold and pentoxyresorufin O-depentyase (PROD; CYP2B1/2) about 30-fold but barely induced ethoxyresorufin O-deethylase activity (EROD; CYP1A1/2) (Nakajima et al., 1991). Imaoka and Funae (1991) also showed that 2-hexanone induced the immunologically measured levels of several CYP450 isozymes, foremost CYPs 2B1, 2B2, 2C6, and 2E1. Minimal or equivocal induction was observed for CYPs 1A1, 1A2,

2C7, and 4A3. The levels of CYPs 2C11 and 2C13 were slightly reduced (Imaoka and Funae, 1991). However, it is not evident to what extent 2-hexanone might affect its own metabolism via enzyme induction. Similarly, the enzymes that synthesize the glucuronides of 2-hexanone metabolites, which were identified by Abdel-Rahman et al. (1976) in the urine of 2-hexanone-exposed rats, guinea pigs, and rabbits, have not been characterized further.

3.4. ELIMINATION

In humans exposed to 2-hexanone via inhalation at 10 or 50 ppm for 7.5 hours or to 100 ppm for 4 hours, unchanged 2-hexanone (but none of its metabolites) was found in expired air, and neither 2-hexanone nor any of its metabolites was found in urine during or after exposure (DiVincenzo et al., 1978). 2-Hexanone was no longer detected in expired air 3 hours after exposure to 50 or 100 ppm. In two humans who received a single oral dose of 1- ^{14}C -2-hexanone, breath excretion of $^{14}\text{CO}_2$ reached a peak within 4 hours then decreased slowly over the next 3 to 5 days. Average overall recovery of the ^{14}C -label in 8 days was 40% in breath and 26% in urine. Feces were not analyzed (DiVincenzo et al., 1978). These results suggest slow clearance and possibly retention of 2-hexanone in humans exposed by this route.

In beagles exposed to 2-hexanone via inhalation at 50 or 100 ppm for 6 hours, 32 and 35%, respectively, of the inhaled vapor was excreted in the expired breath (DiVincenzo et al., 1978). By 3 to 5 hours after exposure, 2-hexanone was no longer detected in expired air. Excretion via other routes was not addressed.

In rats administered a single oral dose of 1- ^{14}C -2-hexanone, DiVincenzo et al. (1977) observed similar results as the above findings in humans. Radioactivity in breath accounted for about 45% of the administered dose (5% was in unchanged 2-hexanone; 40% was in $^{14}\text{CO}_2$); 38% was found in urine; 1.1% was recovered in the feces; and about 15% remained in the carcass. In male rats that received daily gavage doses of 2-hexanone at 400 mg/kg-day for 40 weeks, very low concentrations of free 2-hexanone were detected in the urine from the third week on. A maximum concentration of approximately 20 μg was reached in the 17th week (Eben et al., 1979). Similarly, free 2,5-hexanediol was found in the urine after 3 weeks and peaked in the 17th week. Free and conjugated 2,5-hexanedione were present in the 7th week, whereas excretion levels of the free form were consistent throughout the study. A strong correlation was observed in this study between the onset of neuropathy and the urinary concentration of 2,5-hexanedione when 2-hexanone, 2,5-hexanedione, or 2,5-hexanediol was administered orally to rats at 400 mg/kg-day.

Radiolabeled ^{14}C from 1- ^{14}C -2-hexanone applied to the forearms of two human volunteers was found in the breath and urine (DiVincenzo et al., 1978). In one subject, eliminated amounts in urine and breath were similar, while, in the other subject, the levels in breath were about three times higher than in urine. Fecal elimination was not measured.

3.5. PHYSIOLOGICALLY BASED TOXICOKINETIC MODELS

2-Hexanone was considered as a metabolite in two physiologically based toxicokinetic (PBTk) models for n-hexane that focus on its neurotoxic metabolite, 2,5-hexanedione (Hamelin et al., 2005; Perbellini et al., 1990). PBTk models that deal specifically with 2-hexanone were not identified. A blood/air partition coefficient of 127 for 2-hexanone measured using preserved human blood has been reported (Sato and Nakajima, 1979).

4. HAZARD IDENTIFICATION

4.1. STUDIES IN HUMANS—CASE REPORTS, EPIDEMIOLOGICAL STUDIES, AND OBSERVATIONAL STUDIES

In humans, 2-hexanone vapor caused irritation of the eyes and respiratory tract during acute exposure to relatively high concentrations. Men exposed to 0.23, 0.65, or 2% 2-hexanone in air (9422, 26,600, or 81,900 mg/m³) for 1 minute or less reported strong eye and nasal irritation (Schrenk et al., 1936). Moderate eye and nasal irritation was reported after a brief exposure to 0.1% (4100 mg/m³). Peripheral neuropathy was reported in printers, furniture finishers, and spray painters occupationally exposed to 2-hexanone (Davenport et al., 1976; Mallov 1976; Allen et al., 1974; Billmaier et al., 1974). Several studies have described the occurrence of neurological effects after the introduction of 2-hexanone into products used in the occupational setting.

Davenport et al. (1976) reported the occurrence of symmetrical polyneuropathy in a 35-year-old male who was occupationally exposed to 2-hexanone among other compounds. The patient had worked as a furniture finisher for several years, most recently spraying lacquer compounds, sometimes without using a face mask. Initially, according to the manufacturer, MiBK was present at a concentration of 20% in the finish, 12% in the thinner, and 7% in the sealer. Toluene, xylene, n-butyl alcohol, and acetone were also present in various proportions. After repeated inquiries, the manufacturer disclosed that, for the 6-month period before the onset of the man's illness, 2-hexanone had been substituted for MiBK on a volume-for-volume basis in the formulations of lacquers and solvents because of MiBK supply limitations. The patient first noticed tingling in the soles of his feet and mild clumsiness of gait. Weakness progressed rapidly to the upper extremities, resulting in a wheelchair-bound condition. Three months after the onset of the first symptoms, routine hematology, blood chemistry, urinalysis, spinal fluid, and analysis for heavy metals and porphyrins were normal. A biopsy of the sural nerve¹ at the level of the lateral malleolus revealed diffuse fibrosis and loss of nerve fibers. Several enlarged axons, with and without myelin sheaths, with neurofibrillary tangles were evident. A clinical evaluation 3 months later indicated improved strength and ability to walk unassisted, though with some residual unsteadiness of gait. Tendon reflexes distal to the elbows and knees were still absent. The case report noted that a similar progressive distal extremity weakness developed in a 19-year-old coworker of the patient. This condition also improved following removal from contact with lacquer products.

One probable and two definite cases of 2-hexanone-induced peripheral neuropathy were found during an investigation of 26 painters who worked at Cannelton or nearby Newburgh dams

¹ A sensory nerve that innervates the skin of the back of the leg, and skin and joints on the lateral side of the heel and foot.

on the Ohio River (Mallov, 1976). Two formulations of paint were used. The older formulation contained 22% (weight/weight [w/w]) MiBK and 22% (w/w) methyl isoamyl ketone. In the newer otherwise identical formulation, these solvents were replaced by 44% (w/w) 2-hexanone. While both paint formulations were reported to contain 3.1% (w/w) of the known neurotoxicant triorthocresyl phosphate, this substance was not found in two bulk samples of the 2-hexanone paint formulation. One definite case of peripheral neuropathy was that of a 42-year-old man, a painter for 10 years, who had been painting Cannelton Dam from September 1972 until August 1973. His initial signs, including weight loss, numbness and tingling of feet, and progressive weakness in both lower extremities that progressed to his upper extremities as well, began in July 1973. Weakness progressed until he could no longer stand without assistance. Lower extremity reflexes became absent and an electromyogram was abnormal. Blood and urine lead analysis indicated slightly elevated levels but not sufficient to cause effects. The second case was that of a 35-year-old man who had been painting since he was 14 years old. He painted at Cannelton Dam from April to October 1973. He felt well until about 4 weeks prior to the termination of painting at Cannelton but eventually became unable to rise from a sitting position without help. Urine lead levels were in the lower normal range. The third painter had worked at either Cannelton or Newburgh dam from September 1970 until November 1973. He also felt well until about 4 weeks prior to termination of painting. While he experienced weakness in his extremities, he remained able to walk but reported above-normal episodes of falling and dropping things. He was not examined by a physician until 3.5 months after the onset of symptoms, at which time absent ankle reflex, foot weakness, and diminished sensation were noted. None of the three patients had a history of alcoholism or family history of neurological disease or took medications.

A cross-sectional study of peripheral neuropathy among employees at a coated fabrics plant in Ohio was started when it was noted that six workers from the print department had developed severe peripheral neuropathy (five hospitalized, one seen as outpatient) between April and August 1973 (Allen et al., 1974; Billmaier et al., 1974). The plant produced plastic-coated printed fabrics that were used mainly for wall coverings and automobile interiors. Processing steps included mixing, calendering, laminating, coating, printing, embossing, inspecting, and shipping. Starting in September 1973, all 1157 employees of the plant (including the original six cases) were screened using electromyography and nerve conduction testing. A total of 192 employees were referred for detailed neurological evaluation. On the basis of these examinations, it was concluded that 68 employees had definite signs, symptoms, and electrodiagnostic findings of peripheral neuropathy. Severity ranged from mild (electrodiagnostic findings but no physical symptoms) to moderate (distal sensory loss) to severe (distal muscle weakness and sensory loss). There were a total of nine severe cases, including the original six cases. Cases with possible causes other than a toxic chemical (e.g., diabetes) were

not included in the analysis but were identified in the presentation of results. The distribution of cases within the plant is shown in Table 4-1.

Table 4-1. Prevalence of peripheral neuropathy among employees of a coated fabrics plant

Workplace	Number of cases	Number of employees examined	Prevalence (%)
Non-print departments	30 ^a	984	3
Print department (total)	38 ^b	173	22 ^c
Operators	27	69	39 ^c
Helpers	10	59	17 ^c
Foreman	0	21	0
Service helper	1	16	6
Not known	0	8	0
Total	68	1157	6

^aIncludes 18 persons with diabetes or other conditions that can cause or contribute to neuropathy.

^bIncludes one person with diabetes and one person on isoniazid therapy.

^cSignificantly elevated compared with non-print departments ($p < 0.001$) using the chi-square test.

Source: Billmaier et al. (1974).

The prevalence of peripheral neuropathy was significantly higher among print department employees than among employees from other departments (22 vs. 3%, $p < 0.001$). All nine severe cases were print department workers. Within this department, prevalence was highest among printer operators (39%, $p < 0.001$ compared with non-print department employees), who spent almost 100% of their time near the printing machines. Prevalence among helpers (17%) who spent roughly 50% of their time near the printing machines was also significantly elevated compared with non-print department employees ($p < 0.001$). There was a 6% prevalence among service helpers who were in and out of the premises (one case among service helpers was a pan washer who used the solvent for cleaning). Among manufacturing departments other than the print department, the prevalence of neuropathy ranged from 0 to 6.7%. No cases of peripheral neuropathy were observed in supervisory personnel who remained at a distance from the machines or in office personnel.

In addition to job category, incidence of neuropathy was also associated with working overtime (print operators with definite neuropathy averaged 47.2 hours/week versus 42.0 hours/week for those without neuropathy [$p < 0.01$]) and with eating on the job (data not shown). Each employee generally worked on the same machine all the time. No differences in neuropathy incidence were found based on the type of printing machine or the area in which the machine was located; data were insufficient to correlate illness with individual machines. Among print department employees, there were no significant differences in neuropathy incidence related to age or tenure in the department; 90% of cases had presented within the previous year, and only 5% of the cases were known to have medical conditions that could cause

or contribute to neuropathy. Among non-print department employees, cases were clustered in older (40+) employees ($p < 0.001$); only 53% had onset within the previous year, and 60% of these cases were known to have diabetes or other medical conditions that could cause or contribute to neuropathy unrelated to compound exposure.

In the search for the etiologic agent, other chemicals known to cause peripheral neuropathy were ruled out, either by clinical tests on workers or because they were not used in the plant. Based on an investigation into the relationship between the cases of peripheral neuropathy and the distribution of the roughly 275 chemicals used in the plant, the most likely agent appeared to be contained in the solvents used as ink thinners and cleaners. These had previously consisted of methyl ethyl ketone (MEK) and MiBK, but, starting in August 1972, the latter was phased out and gradually replaced by 2-hexanone, which reached maximal usage in December 1972. Substitution of 2-hexanone for MiBK was the only major change in the production process in the previous 7 years. In September 1973, the print department was closed for a month and 2-hexanone was removed from production materials. Thus, there was a 13-month window of exposure to 2-hexanone.

In addition to exposure to 2-hexanone, affected workers were also exposed to high concentrations of MEK that sometimes vastly exceeded threshold limit values (TLVs). MEK by itself does not produce this type of neuropathy in animal studies but can potentiate the effects produced by 2-hexanone (Saida et al., 1976). Thus, the presence of MEK in the coated fabrics plant study could contribute to an overestimation of the risk associated with exposure to 2-hexanone itself. Workplace levels for 2-hexanone and MEK from this study are presented in Table 4-2.

Table 4-2. Results of area atmospheric sampling for MEK and 2-hexanone in a coated fabrics plant

	Front of print machine ^a		Back of print machine ^a		Wind-up area ^a	
	MEK ^b	2-Hexanone ^c	MEK	MEK ^b	2-Hexanone ^c	MEK
	104	2.3	85	104.0	2.3	85.0
	109	2.6	265	3.0	44	2.0
	124	4.1	401	9.0	47	2.0
	162	5.1	440	9.8	49	2.6
Median	220	5.8	603	21.7	127	5.9
	453	9.7	608	23.9	143	6.0
	565	11.5	725	48.6	250	7.9
	570	19.8	750	49.9	289	9.8
	670	21.7	763	156.0	338	17.5

^aValues are in ppm, listed from lowest to highest result obtained for each solvent at each work location.

^bTLV = 200 ppm.

^cTLV = 100 ppm.

Source: Billmaier et al. (1974).

Another confounding factor for this study is that exposure may not have been limited to the inhalation route. Poor work practices documented at the plant included washing hands in solvent, using solvent-soaked rags to clean equipment, and eating in work areas. Dermal and even oral exposure is likely to have occurred. The significance of exposure by these routes is suggested by the observations that eating on the job was associated with the development of neuropathy and that a worker whose job involved washing pans with the solvent was the only afflicted print department worker other than the print operators and their helpers. As discussed in Sections 3.1.2 and 3.1.3, 2-hexanone is absorbed readily through the skin and gut and can produce neuropathy by both routes in animals.

The researchers reported that patients removed from 2-hexanone exposure showed significant and consistent improvements. They also performed a study of workers at a comparable coated fabrics plant in California that produced the same products as the one in Ohio but without the use of 2-hexanone. Electrodiagnostic studies were conducted on 21 solvent-exposed workers at the California plant, but no peripheral neuropathy was found.

4.2. ACUTE, SUBCHRONIC, AND CHRONIC STUDIES IN ANIMALS

4.2.1. Oral Exposure

4.2.1.1. *Acute and Short-Term Oral Exposure*

Range-finding toxicity data by Smyth et al. (1954) list an oral median lethal dose (LD₅₀) of 2.59 g/kg of 2-hexanone for rats, while Tanii et al. (1986) provide an oral LD₅₀ of 2.43 g/kg for mice. Details for either study are limited (Tanii et al., 1986; Smyth et al., 1954).

4.2.1.2. *Subchronic Toxicity Studies*

16-Week study: female Wistar rats

Homan et al. (1977) conducted a 120-day drinking water study with female Wistar rats. 2-Hexanone (purity not stated) was administered in drinking water at 0, 0.65, or 1.3% (0, 480, or 1010 mg/kg-day). A dose-dependent decrease in food consumption was observed in exposed animals versus controls. Water consumption in exposed animals was reduced to about half that of controls. Animals exposed to 0.65 or 1.3% 2-hexanone experienced a 45.5 and 68.8% reduction in body weight gain, respectively. A dose-dependent decrease in absolute liver weight was observed in exposed animals. Absolute kidney weights were increased, and there was a dose-dependent increase in relative kidney weights. A summary of the data for diet and water consumption, body weight gain, and organ weights is provided in Table 4-3. Neurotoxicity outcomes among the treated animals are outlined in Section 4.4.1.

Table 4-3. Gross observations in rats exposed to 2-hexanone in drinking water for 120 days

Dose (mg/kg-day)	Food intake (g/day)	Water intake (mL/day)	Body weight gain (g)	Liver weight		Kidney weight	
				Absolute (g)	Relative	Absolute (g)	Relative
0	17.99	32.29	110.2	10.30	3.10	1.97	0.60
480	16.90	17.98	60.0 ^a	9.01	3.35	2.21	0.82 ^a
1010	12.90	17.33	34.3 ^a	7.80 ^a	3.38	2.10	0.92 ^a

^aSignificantly different from controls, $p < 0.01$.

Source: Homan et al. (1977).

40-Week study: male Wistar rats

Eben et al. (1979) gavaged male SPF-Wistar rats daily with 400 mg/kg 2-hexanone (98% pure) for 40 weeks. Body weight gain in treated animals was less than in controls; a decrease in body weights was observed from the 17th to the 25th weeks, followed by a slight increase until study completion. There were also symptoms of neurotoxicity in the treated animals (see Section 4.4.1).

4.2.1.3. Chronic Toxicity Study

13-Month study: CD/COBS(SD) rats

O'Donoghue et al. (1978)² conducted a 13-month study in male CD/COBS(SD) rats. The animals' drinking water contained 0, 0.25, 0.5, or 1.0% (0, 143, 266, or 560 mg/kg-day) 2-hexanone (96% pure, containing 3.2% MiBK and 0.7% unknown contaminants). 2-Hexanone produced a dose-dependent reduction in body weight at all doses tested. The effect was present by the second week in the two highest dose levels and by the third week in the low-dose group. A statistically significant increase in liver weight was found in the highest dose group compared with all groups except the 0.5% group. The 0.5% and 0.25% groups showed dose-dependent increases in relative liver weights compared with controls. A statistically significant increase in relative kidney weights was present between the 1.0% 2-hexanone group and all other groups and between the 0.5% group and all other groups. Similarly, a statistically significant increase in relative testes weight was found between the 1.0% group and all other groups. A summary of the body weight and organ weight data is present in Table 4-4 (O'Donoghue et al., 1978).

Clinical neurological deficits were noted in animals exposed to either 0.5 or 1.0% 2-hexanone. Severe deficits including decreased extension of the hind limb, hind-limb weakness, and muscular atrophy of the hind-limb musculature were noted among animals treated with 1% 2-hexanone. Deficits among animals exposed to 0.5% 2-hexanone were slight and did not result in clinical progression. Evidence of axonal swelling was noted at all dosing levels of

²This study is an unpublished study; accordingly, it was externally peer reviewed by EPA in December 2007.

2-hexanone. Neurological effects are discussed in further detail in Section 4.4.1.1. Other than neural effects and changes in body weight, no nonneural clinical signs related to 2-hexanone exposure were found.

Table 4-4. Pathological changes in rats exposed for 13 months to 2-hexanone

	Body weight ^a	Liver ^b		Kidney ^b		Testes ^b	
		Absolute	Relative	Absolute	Relative	Absolute	Relative
Control	710	26.71 ± 2.02	3.64 ± 0.41	4.66 ± 0.53	0.63 ± 0.87	2.99 ± 0.81	0.40 ± 0.11
2-Hexanone (0.25% or 143 mg/kg-day)	685	24.99 ± 4.33	3.97 ± 0.43	4.58 ± 0.69	0.73 ± 0.05	3.24 ± 0.38	0.52 ± 0.08
2-Hexanone (0.5% or 266 mg/kg-day)	612	25.06 ± 2.04	4.22 ± 0.43	5.33 ± 0.31	0.90 ± 0.12 ^c	3.16 ± 1.04	0.54 ± 0.19
2-Hexanone (1.0% or 560 mg/kg-day)	448	20.73 ± 2.95	4.62 ± 0.32 ^c	4.86 ± 0.38	1.10 ± 0.23 ^c	3.29 ± 0.26	0.75 ± 0.17 ^c

^aValues are means of 10 animals.

^bValues are mean ± SE based on four or five animals per group.

^cStatistically different from controls, $p < 0.05$

Source: O'Donoghue et al. (1978).

To determine whether the concentration of MiBK, a CYP450 inducer, a contaminant in the 2-hexanone formulation used by O'Donoghue et al. (1978) may have altered the observed toxicity of 2-hexanone, other studies were evaluated that used MiBK as a test article. In a 13-week gavage study, 30 male and female Sprague-Dawley rats were treated daily with 0, 50, 250, or 1,000 mg MiBK/kg-bw (MAI, 1986). At the middle and high doses, adverse effects were observed in the liver and kidney, which progressed in severity in the high dose animals. No treatment-related effects of any kind were observed at 50 mg/kg-day. The Carnegie-Mellon Institute of Research (1977) conducted a 120-day drinking water study with 1.3% MiBK using female HLA Wistar rats. The authors estimated the dosage to be 1040 mg/kg-day. The only statistically significant finding was increased mean absolute and relative kidney weights in treated rats compared with controls. Histopathological examination revealed renal tubular cell hyperplasia in only one of five of the treated rats. No exposure-related histopathological changes were found in other organs. Based on the foregoing, it can be concluded that the dosage of MiBK received as an impurity in the study by O'Donoghue et al. (1978) did not contribute to the observed 2-hexanone related effects. O'Donoghue et al. (1978) did not observe adverse effects in the kidney or liver of treated animals, despite these organs being the target organs of toxicity in experimental studies with MiBK from both the oral and inhalation routes (U.S. EPA, 2003a).

4.2.2. Inhalation Exposures

4.2.2.1. Acute and Short-Term Toxicity Studies

No acute inhalation toxicity studies of 2-hexanone were identified. The National Library of Medicine's Hazardous Substances Data Bank states that a 4-hour exposure of rats to 4000 ppm 2-hexanone did not kill all animals, while exposure to 8000 ppm for 4 hours was an LD₁₀₀ (NLM, 2005). Abdo et al. (1982) reported the death of one out of five hens exposed continuously to 200 ppm 2-hexanone (70% purity). No deaths were reported in hens exposed to 100 ppm or lower (Abdo et al., 1982).

4.2.2.2. Subchronic Toxicity Study

11-Week study: male rats

Groups of five male rats (CrI:COBS/CD[SD]BR) were exposed to 0 or 700 ppm (0 or 2870 mg/m³) 2-hexanone (purity 96.1%) 72 hours/week for 11 weeks (Katz et al., 1980). The exposure schedule was as follows: two 20-hour periods and two 16-hour periods, Monday through Friday, separated by 8-hour nonexposure periods. Total white blood cell counts of treated animals were significantly ($p < 0.05$) lower than those of controls; no other differences were noted in clinical chemistry or hematological values. Gross examination of treated animals revealed marked atrophy of the hind-limb musculature, depletion of adipose tissue, and significantly decreased absolute and relative testicular weight ($p < 0.05$). Histopathological examination was performed on selected tissues, including lung and trachea (but not nasal cavities), eye, digestive tract, pancreas, thyroid, parathyroid, testes, epididymides, spleen, bone marrow, mesenteric lymph nodes, thymus, and nervous system. Atrophy of testicular germinal epithelium and grossly enlarged axons in the brain stem and cerebellum were observed in treated animals. No damage to bone marrow was evident despite the low white blood cell count. Although no discussion of findings in the lung or trachea was presented, the implication is that there were no treatment-related changes in these tissues. The treatment group developed signs of neurotoxicity (weakened hind- and forelimb grasp) by the second week of exposure, progressing to severe hind-limb weakness by 71 days, and showed decreased weight gain. Neurological effects are discussed in further detail in Section 4.4.1.2.

4.2.2.3. Chronic Toxicity Study

72-Week study: male Sprague-Dawley rats

Krasavage and O'Donoghue (1977) exposed groups of male Sprague-Dawley rats (18/group) to 0, 100, or 330 ppm (0, 410, or 1353 mg/m³) 2-hexanone (purity not specified) 6 hours/day, 5 days/week for 72 weeks. Clinical signs (observed daily and examined weekly), body weight (recorded weekly), and water consumption (at 15, 22, 32, and 44 weeks of exposure) were monitored. Beginning at 4 weeks and continuing at approximately 6-week intervals for the first 52 weeks, unspecified numbers of animals were killed for microscopic

examination of an extensive list of tissues, including the trachea and lung. Body weights and weight gain were comparable among groups until the 20th week. Thereafter, body weights of the high-concentration animals fell behind those of controls (data presented graphically without statistical analysis); a visual estimate of the graphic presentation suggested that the body weights of high-concentration animals were at least 10% less than those of controls. After 36 weeks of exposure, body weight gain in the low-concentration group also began to lag behind controls. Water intake was comparable among groups.

Gross postmortem findings revealed no compound-related changes. Low-concentration animals did not develop clinical signs attributed to 2-hexanone exposure or morphologic lesions of neuropathy. Histopathologic evidence for neuropathy in high-concentration rats was equivocal. Neurological effects are discussed in further detail in Section 4.4.1.2. Spontaneous lesions were present in the urogenital, cardiovascular, and endocrine systems of both treated and control animals and were therefore not attributed to 2-hexanone exposure by the study authors (Krasavage and O'Donoghue 1977).

2-Year study: cats

Groups of four domestic shorthair cats were exposed by inhalation to 0, 100, or 330 ppm (0, 410, or 1353 mg/m³) 2-hexanone (purity not specified) for 6 hours/day, 5 days/week for 2 years (O'Donoghue and Krasavage 1979). Clinical signs and body weights were monitored. Serum was sampled after 30, 90, and 128 exposures to determine the levels of 2-hexanone and two of its metabolites, 5-hydroxy-2-hexanone and 2,5-hexanedione. Each sample set involved collection serum on a Monday prior to daily exposure, the following Tuesday prior to daily exposure, the following Friday prior to daily exposure, immediately after daily exposure and one and three quarter hours after daily exposure. Sera from high-dose and control animals were also analyzed for sodium, potassium, chloride, and calcium levels. Cats were sacrificed at the end of the treatment and were subjected to necropsy and histopathologic examinations.

No clinical neurological effects attributed to exposure to 2-hexanone were identified except that cats anesthetized with sodium pentobarbital following a 6-hour exposure had prolonged sleeping times (O'Donoghue and Krasavage, 1979). No compound-related changes of body weight or serum electrolyte values were found. Serum levels of 2-hexanone and the two metabolites, 5-hydroxy-2-hexanone and 2,5-hexanedione, were below the detection limit on Monday morning following a two-day non-exposure period. With the exception of 2,5-hexanedione in the 330 ppm group (1353 mg/m³), serum levels on Tuesday morning following a 6-hour exposure after 30 days of exposure remained below the detection level. Of the three substances measured, 2-hexanone cleared the serum more quickly than 5-hydroxy-2-hexanone, which cleared more quickly than 2,5-hexanedione. Biopsy examinations through the first 9 months of exposure were unremarkable and did not serve as an early detection method for neuropathy. Gross postmortem findings revealed no compound-related changes. General

histopathologic examinations showed no compound-related changes other than in the nervous system and musculature. Neurological effects are discussed in further detail in Section 4.4.1.2.

4.2.3. Dermal Exposure

90-Day study: hens

Abou-Donia et al. (1985b) treated leghorn laying hens (n = 5) with 2-hexanone (99% pure; topical application, 1 mmol/kg-bw). The chemical was applied daily with a micropipette over an area of 10 cm² on the unprotected back of the neck for 90 days. All hens developed gross ataxia. At sacrifice, no changes were observed in treated versus control animals when compared for size, shape, weight, or color. Equivocal histopathologic changes were present in the spinal cord of two hens. These histopathologic changes were characterized by swollen axons without obvious fragmentation of the axon or myelin sheath. No precautions against licking were mentioned in the study, so ingestion of 2-hexanone may have taken place.

4.3. REPRODUCTIVE/DEVELOPMENTAL STUDIES—ORAL AND INHALATION

4.3.1. Oral Exposure

No standard two-generation studies or other studies of reproductive and developmental effects following oral administration of 2-hexanone were identified.

4.3.2. Inhalation Exposure

In a developmental study, Peters et al. (1981) exposed groups of 25 pregnant F344 rats to 0, 500, 1000, or 2000 ppm (0, 2048, 4096, or 8193 mg/m³) 2-hexanone (purity not stated), 6 hours/day on gestational days (GDs) 1–21. A separate control group was maintained for each exposure group and the high-concentration controls were pair fed. Respective controls were exposed to ambient air in similar chambers to those of their exposed counterparts. Sexually mature female rats were impregnated and placed in exposure chambers 6 hours/day throughout gestation. Four weeks postdelivery, the dam was separated from the pups. The maternal 500 ppm group along with its control was terminated before 3 weeks because of an apparent lapse in care during which offspring were “unable to reach food and water,” resulting in reduced weight gain in this group. The pups in the control, 1000, and 2000 ppm groups were observed over a lifetime. At 4 (weaning), 8 (puberty), and 14 weeks (adult) and at 18–20 months of age (geriatric), five males and five females were taken, one per litter, for gross and histopathology studies and for measurement of organ/body weight ratios. At different periods of development (weaning, puberty, and adult), offspring underwent behavioral testing. Pentobarbital sleeping time was also measured in pubescent and geriatric animals in the high-dose and control groups.

Survival in the 2000 ppm and 1000 ppm dams was not affected by treatment. High-dose dams appeared sluggish after exposure but seemed to have recovered by the next exposure. Hair loss, lack of muscular coordination, and weakness were observed in “several” dams at the

highest concentration after 20 days of exposure. Abnormal sniffing in the air was reported for dams in the 1000 ppm group. Maternal gestational body weight gain was decreased by 14 and 10% in the dams exposed to 2000 and 1000 ppm, respectively. Rats in the 2000 ppm exposure group were observed to eat less than the controls. No unusual behavior or change in maternal gestational growth was reported for the 500 ppm dams. Histopathology and neurotoxicity evaluations were not performed in the dams.

2-Hexanone exposure was found to result in statistically significant decreases (*p* value not reported) in litter size and pup weight observed at the highest exposure level (Peters et al., 1981). However, maternal toxicity, manifested as decreased maternal body weight during gestation, was also evident in high-dose dams, suggesting that maternal toxicity might have affected fetal growth. There was a significant decrease in the number and weight of live offspring of dams in the 2000 ppm exposure group. A lifelong, statistically significant, concentration-related reduction in growth was observed in male offspring. Only a slight treatment-related effect on body weight was seen in female offspring. Organ weights in weanling, pubescent, and geriatric offspring were unaffected by treatment, but brain weight in adult 1000 ppm offspring was significantly increased compared to that of control. Organ weights were not measured in high-dose adult offspring. The authors did not report any gross skeletal alterations. Beginning at 40 weeks of age, offspring of dams treated with 1000 or 2000 ppm showed a 3–5% decrease in survival relative to controls. The incidence of pathological lesions and the types of lesions contributory to death were not significantly different in treated and control groups (Table 4-5).

Table 4-5. Summary of pathological lesions in offspring of rats exposed to 2-hexanone during gestation

	Control				1000 ppm				2000 ppm			
	Male	Female	Total	%	Male	Female	Total	%	Male	Female	Total	%
Number of animals dead or sacrificed ^a	57	57	114	--	37	34	71	--	24	22	46	--
Pituitary tumor	1	3	4	3.5	1	1	2	3	1	0	1	2
Pituitary hemorrhage	2	0	2	2	0	0	0	0	1	2	3	6.5
Diaphragmatic hernia	1	1	1	2	0	1	1	1.4	1	2	3	6.5
Ovarian cysts	0	2	2	2	0	7	7	10	0	8	8	18
Mottled testes	26	0	26	23	16	0	16	23	1	0	1	2

^aAnimals include those dying subsequent to weaning in addition to those sacrificed at 78 ± 2 weeks of age.

Source: Peters et al. (1981)

Standard hematological tests (hemoglobin, red blood cell count, white blood cell count, lymphocytes, mean corpuscular hemoglobin, packed cell volume) showed no significant

treatment effect on the processes involved in blood cell formation and function (Peters et al., 1981). Clinical chemistry findings were limited to a concentration-related decrease in creatinine phosphokinase activity in pubescent offspring, with values in the 1000 ppm and 2000 ppm groups significantly lower ($p < 0.05$) than controls. In geriatric offspring, there were significant increases ($p < 0.05$) in serum alanine aminotransferase activity in the 1000 ppm and 2000 ppm groups and sodium in the 2000 ppm group. The only lesions showing a significant concentration-response relationship ($p < 0.05$, Fisher's exact test conducted for this assessment) at the time of geriatric sacrifice were ovarian cysts that had 4% (2/57), 21% (7/34), and 36% (8/22) incidences in the control, 1000 ppm, and 2000 ppm females, respectively.

In pubescent high-dose male offspring, pentobarbital sleep time was significantly increased ($p < 0.05$) compared with controls. No significant changes in pentobarbital sleep time were noted in pubescent females or geriatric offspring of either sex. Behavioral alterations were reported in the offspring of pregnant rats exposed to 1000 ppm or 2000 ppm 2-hexanone. These effects consisted of reduced activity in the open field, increased activity in the running wheel, and deficits in avoidance conditioning. Offspring of treated dams (both dose levels) clung to an inclined screen longer than offspring of controls at all ages (newborn, weanling, puberty, and adult) except geriatric in which results were similar to those of controls. For offspring in the puberty and adult categories, pronounced sex differences were noted; females in all exposure categories (including controls) were clinging from 24–100% longer than males. However, the biological significance of this observation is unknown. There was a decreased rate of avoidance learning in puberty-aged females of treated dams and increased random movement in both puberty-aged and adult offspring of treated dams. Behavioral tests in most cases indicated that maternal exposure to 2-hexanone was associated with hyperactivity in the young and decreased activity in the geriatric stage, which the authors (Peters et al., 1981) speculated to be due to premature aging resulting from the earlier hyperactivity. It is not clear whether these effects are the result of transplacental exposure to 2-hexanone or of postnatal exposure to 2-hexanone and/or its metabolites via the milk of the exposed dams.

4.4. OTHER ENDPOINT-SPECIFIC STUDIES

4.4.1. Neurotoxicity Studies

4.4.1.1. Oral Exposures

90-Day study: hens

In hens that received a single gavage dose of 2-hexanone (technical grade 2-hexanone, 70% pure, containing 30% methyl isobutyl ketone) at 2000 mg/kg, mild weakness was observed on the day of administration, followed by apparent recovery in 4–5 days. Hens that received 100 mg/kg showed no signs of neurotoxicity (Abou-Donia et al., 1982). In a subchronic (90-day) phase of the same study, hens ($n = 3$) administered 2-hexanone at 100 mg/kg-day or higher developed

severe ataxia or near paralysis. There was also evidence of histopathological changes, including swelling or degeneration of thoracic and lumbar regions of the spinal cord.

90-Day study: rats

Krasavage et al. (1980) administered 660 mg/kg 2-hexanone (96% pure) by gavage to male CD/COBS(SD) rats for up to 90 days. The authors considered severe hind-limb weakness or paralysis, as exhibited by “dragging” of at least one hind foot, to be clear indication of neuropathy. When this endpoint was reached, the treatment was terminated and the animal was processed for histological examination. There was a time- and dose-dependent depression in body weight gain and feed consumption. Treated animals consumed an average of 21 grams/day versus 28 grams/day for controls. The body weights of experimental and control animals at study completion were approximately 400 and 600 grams, respectively. Histologic examination of nerve tissue collected at termination revealed morphologic changes indicative of giant axonal neuropathy, which included multifocal axonal swellings, myelin infoldings, and paranodal myelin retraction. In this study, atrophy of the germinal epithelium of the testes was also observed, but the statistical significance of this observation was not addressed (Krasavage et al., 1980).

120-Day study: female Wistar rats

Homan et al. (1977) conducted a 120-day drinking water study with female Wistar rats (five/group). 2-Hexanone (purity not stated) was administered in drinking water at 0, 0.65, or 1.3% (0, 480, or 1010 mg/kg-day) (for further experimental details see Section 4.2.1.2). Neurological evaluations were conducted to assess balance, strength, coordination, and behavior. Performance was scored for each of the following 10 criteria: posture, gait, palpebral reflex, startle reflex, flexor reflex, extensor reflex, placing reflex, hopping reaction, righting reflex, and clinging reaction. Score ranged from 0 to 2 with 0 indicating normal and 2 being clearly deficient. The net score for each rat was calculated as the sum of the individual test scores. Scores were tabulated, ranked, and analyzed using the Kruskal-Wallis ranks sum test. The rank values (statistics generated from the Kruskal-Wallis test) for each treatment group for a given day of analysis were then averaged to generate a mean rank and standard deviation. A summary of the mean rank (mean of the values generated from the Kruskal-Wallis test) and standard deviation is provided in Table 4-6. Gross pathological evaluation revealed mild atrophy affecting skeletal muscles of the hind limbs in two of five animals in the 0.65% group and slight to severe atrophy of skeletal muscles (most pronounced in muscles of the hind limbs) affecting four of five animals in the 1.3% group (Homan et al., 1977).

Table 4-6. Time course of neuropathy scores following exposure of rats to 2-hexanone in drinking water

Treatment	Analysis after number of treatment days			
	46	57	80	110
	Mean rank value			
Control	26.1 ± 9.1	15.0 ± 0.0	22.1 ± 12.8	17.5 ± 0.0
0.65% 2-hexanone	32.0 ± 14.9	30.6 ± 12.7	30.9 ± 9.2	21.5 ± 8.0
1.3% 2-hexanone	37.5 ± 12.6	41.0 ± 5.7	40.0 ± 13.7	47.2 ± 2.8a

^aStatistically significant versus controls, $p < 0.05$.

Source: Homan et al. (1977).

24-Week study: guinea pigs

Abdel-Rahman et al. (1978) administered 2-hexanone (purity not stated) in drinking water to guinea pigs (five/group, sex not stated) at 0, 0.1, or 0.25% (approximately 0, 97, or 243 mg/kg-day) for 24 weeks. Bibs were used to prevent dermal absorption by inadvertent contact of the animals' bodies with the solvent. The body weight of the guinea pigs was monitored each week up to the eighth week of the study. At the end of the seventh week, animals exposed to 0.25% 2-hexanone weighed an average of 600 grams versus 440 grams in controls. Similarly, animals exposed to 0.1% 2-hexanone weighed 618 grams by the eighth week compared with 490 grams in controls. Decreased locomotor activity may have contributed to increased body weights. The average motor activity counts in animals exposed to 0.25% 2-hexanone were 714 ± 130 compared to 1173 ± 201 in controls. Pupillary response to light (measured by change in pupillary diameter in response to an intense 2-second light stimulus) was abnormal in high-dose animals for the first 5 weeks of treatment as shown in Table 4-7 (data not provided for 0.1% 2-hexanone). The authors reported that by the 24th week of the study, a greatly impaired pupillary response was observed for all treatment groups (data not provided in the report) (Abdel-Rahman et al., 1978).

Table 4-7. Effect of 2-hexanone on guinea pig pupillary response of both eyes

Treatment	Week							
	1		2		3		5	
	Right ^a	Left ^a	Right	Left	Right	Left	Right	Left
Control	1.83 ± 0.00	1.66 ± 0.17	1.6 ± 0.1	1.67 ± 0.19	1.6 ± 0.06	1.7 ± 0.05	1.5 ± 0.05	1.5 ± 0.1
0.25% 2-hexanone	1.33 ± 0.19	1.33 ± 0.01 ^b	1.05 ± 0.15	1.17 ± 0.01 ^b	0.67 ± 0.17	0.92 ± 0.08 ^b	0.59 ± 0.14 ^b	0.71 ± 0.04 ^b

^aValues represent the mean ± SE of the change in pupillary diameter.

^bStatistically significant from controls ($p < 0.001$) as calculated by study authors.

Source: Abdel-Rahman et al. (1978).

40-Week study: rats

Eben et al. (1979) gavaged male SPF-Wistar rats daily with 400 mg/kg 2-hexanone (98% pure) for 40 weeks. The authors stated that this treatment did not cause neuropathic symptoms; however, from the 17th week the authors noted that the animals exhibited weakness of the hind limbs, which continued until the 28th week. Thereafter, an improvement was observed. No further details were provided.

13-Month study: rats

As previously mentioned in Section 4.2.1.3, O'Donoghue et al. (1978) conducted a 13-month study in male CD/COBS(SD) rats. Each group of 10 rats was exposed to drinking water containing 0, 0.25, 0.5, or 1.0% (0, 143, 266, or 560 mg/kg-day) 2-hexanone (96% pure, containing 3.2% MiBK and 0.7% unknown contaminants). Body weight and neurological examinations were performed weekly. At the end of the study, a dose-dependent reduction in body weight was noted among all dose groups. All but one animal was found to have some evidence of neurotoxicity. Other than neural effects and body weight changes, no compound-related clinical signs were found.

Clinical neurological deficits were found only in animals receiving 0.5 or 1.0% 2-hexanone. Deficits were recorded as slight if there was incomplete extension of the hind limb and just detectable widening of the hind limb stance; moderate if there was obvious weakness, incomplete extension of the hind limbs, and waddling; and severe if there was dragging of at least one hind paw. In the 1.0% group, all the animals exhibited severe deficits. Gross pathological examination revealed observable muscle atrophy of hind-limb and lumbar muscles at this high-dose level. Progression of the clinical findings to a more severe state did not occur with time in the 0.5% group. In addition to the aforementioned changes, animals receiving 1% hexanone in their drinking water displayed loss of tone with grossly observable atrophy of the hind-limb musculature and axial muscles of the lumbar area. Weakness of the forelimbs with some muscle atrophy was observable in three of nine rats at the end of the study. Pain sensation, as judged by toe pinch, remained intact, but motor response such as flexor response was easily overcome. It was noted that tactile placing in the hind limbs could be elicited even in rats with severe weakness. Bowel and bladder functions remained normal. The clinical course was highly variable with improvements in the clinical symptoms being very common; thus, while all animals showed slight deficits on at least two of the weekly examinations, they showed improvements during other weeks.

Evidence of neuropathy was most common in the giant axons of animals of each dose level. In peripheral nerves from the 1.0% group, swelling of giant and other axons was common. Myelin infoldings into the axoplasm were more common than in controls. Myelin ovoids were

frequently found along with degenerating axons. The second most common site of neural degeneration was in the spinal cord, particularly in the ventral and ventromedial funiculi of the thoracolumbar segments. The changes were similar to those found in peripheral nerves. In plastic embedded sections, an additional early change was noted, which consisted of clumping of axonal organelles in otherwise normal peripheral or central axons. Examination of the dorsal root ganglia did not reveal any effect on cell bodies, but in three animals single swollen axons were found in adjacent roots, indicating a very minimal effect. Axonal swelling was also very rare in the brain. No neuropathologic effects were found rostral to the pons. Small numbers of swollen axons were located in the ventromedial medulla. Rare single swollen axons were located in the ventral spino-cerebellar tracts, cerebellar peduncles, and deep cerebellar white matter.

Neurogenic skeletal muscle atrophy occurred in both proximal and distal hind-limb musculature. Myofibrillar atrophy was multifocal with foci overlapping in severe cases to produce large diffuse areas of atrophy with fatty replacement. Intramuscular nerves frequently showed an obvious loss of axons and rarely a swollen axon. No difference in the severity or frequency of atrophic foci was seen between proximal and distal muscles.

In the 0.5% group, peripheral nerve changes were identical in morphology and in the number of animals affected compared with the higher-dose animals but were reduced in severity. Swollen axons were generally few in number but were found in all animals. Myelin ovoids and frankly degenerating axons were also reduced in number. In some nerve bundles, there was obvious loss of axons. Spinal effects were reduced to a few swollen axons and rare degenerating axons in the ventromedial fasciculi of the thoracolumbar cord. Effects on the brainstem and cerebellum were minimal, consisting of only single or small numbers of swollen axons and single degenerating axons in half of the animals examined. Neurogenic skeletal muscle atrophy consisted of infrequent multifocal areas of myofibrillar atrophy that were generally regarded as minor. Two animals without myofibrillar atrophy were considered normal. Three samples from the quadriceps and two from the calf muscles, while not demonstrating myofibrillar atrophy, did have early myopathic effects consisting of foci of increased numbers of angular myofibers and increased numbers of myofibers with central or internal nuclei. In one of these animals, intramuscular axonal swelling was found.

At the 0.25% level, peripheral nerve changes were less severe than at higher doses and axonal swelling was found in 8 of 10 animals examined. In these eight rats, the number of swollen axons was very low, but additional changes, such as myelin infoldings into axons, myelin ovoids, and degenerating axons, were more common. In one animal, while no axonal swelling was observed, numerous degenerating axons were found. Another rat was indistinguishable from controls. Spinal lesions were minimal, consisting of a single or very few swollen axons. A few instances of axonal swelling were found in the medullae of two rats. Neurogenic myofibrillar atrophy was also minimal, occurring as a single or very few foci in two

animals. Foci of angular myofibers were found in four additional animals but were of minimal severity. In control animals, the peripheral and central nervous system (CNS) contained a few degenerating axons and myelin ovoids, but these were minimal. A summary of animals found to have axonal swelling and the areas in which these axons or myopathic changes were found is presented in Table 4-8.

Table 4-8. Summary of neuropathologic findings in male rats administered 2-hexanone in drinking water for 13 months

Treatment	Incidence of axonal swelling				Incidence of myofibrillar atrophy	
	Brain	Spinal cord	Dorsal root ganglia	Peripheral nerve	Quadriceps muscle	Calf muscle
Control	0/10	0/5	0/5	0/10	0/10	0/10
0.25% 2-hexanone	2/10	7/10	0/7	8/10	1/10	2/10
0.5% 2-hexanone	4/10	5/5	0/5	10/10	5/10	6/10
1.0% 2-hexanone	8/10	5/5	3/5	10/10	10/10	10/10

Source: O'Donoghue et al. (1978).

4.4.1.2. Inhalation Exposures

6-Week study: rats

In a short communication, Duckett et al. (1974) reported the results of a study in which groups of nine rats (strain and sex not reported) were exposed to 200 ppm (819 mg/m³) 2-hexanone (purity unspecified) 8 hours/day, 5 days/week for 6 weeks. Four rats served as controls. Animals presented with muscular weakness of all limbs that persisted for a few hours after exposure termination each day. Only the sciatic nerve was examined histologically. Axonal hypertrophy, beading, and degeneration associated with perinodal and segmental breakdown of myelin were observed in the sciatic nerve of all treated rats.

13-Week study: rats

In the same short communication discussed above, Duckett et al. (1974) discussed results of an unpublished subchronic experiment with 2-hexanone. In this experiment, groups of 20 Wistar rats of unspecified sex were exposed to 2-hexanone for 8 hours/day, 5 days/week at 40 ppm (164 mg/m³) for 22–88 days or at 50 ppm (205 mg/m³) for 13 weeks. Similar numbers of control rats were sham exposed. No overt signs or “pathological manifestations” of peripheral or central neuropathy were seen in exposed rats, except for demyelination of the sciatic nerve in 3 of the 20 rats exposed to 50 ppm for 13 weeks. Additional details were not provided. The results at 50 ppm for 13 weeks, when compared with the results at 50 ppm for 6 months, indicate that the incidence of neuropathy increases with increasing duration of exposure.

12-Week study: cats, rats, chickens

Mendell et al. (1974) continuously exposed groups of animals of unspecified sex (four Sprague-Dawley rats, four domestic shorthair cats, and five domestic chickens) to 2-hexanone (purity not stated) for 24 hours per day, 7 days per week for up to 12 weeks. Concentrations of 2-hexanone were initially 200 ppm (820 mg/m³) for chickens and 600 ppm (2,460 mg/m³) for cats and rats but were adjusted at an unspecified time to 100 and 400 ppm (410 and 1640 mg/m³), respectively, to minimize complications from inanition and weight loss. Pair-fed controls were sacrificed when the exposed animals were sacrificed. After 5–8 weeks of exposure, the cats developed hind-limb and forelimb weakness. Focal swelling of the axon along the sciatic nerve, often associated with loss of neurotubules and denudation of myelin beginning at the nodes of Ranvier, and areas of demyelination were observed. Abnormal electromyograms were also observed in the cats exposed for 9–10 weeks; electromyograms were not measured in chickens or rats (Mendell et al., 1974).

90-Day study: hens

Abdo et al. (1982) exposed adult leghorn laying hens (*Gallus gallus domesticus*), 5 per group, to varying concentrations of 2-hexanone (10, 50, 100, 200, and 400 ppm; technical grade 2-hexanone containing 70% 2-hexanone and 30% methyl isobutyl ketone) for 90 days. Body weights were monitored weekly, and hens were examined every other day for neurological signs of 2-hexanone neurotoxicity. A 30-day observation period followed the final exposure. Clinical assessment of neurotoxicity was graded by classifying the degree of ataxia before paralysis as follows: T₁, mild ataxia, characterized by diminished leg movement and reluctance to walk, with hens tending to slide on the floor or fly; T₂, gross ataxia, characterized by a change in gait and disturbance of leg movement; T₃, severe ataxia, with severe leg weakness manifested by unsteadiness and occasional falling on the floor; T₄, ataxia, with near paralysis, marked by inability to walk (Abdo et al., 1982).

The spinal cord and the sciatic, peroneal, and tibial nerves were excised from hens that died during the experiment or were killed by heart puncture and exsanguinations. Severity of lesions was defined by the following criteria: (1) rare swollen axons without fragmentation, phagocytosis, or loss of myelin staining were designated as equivocal histological changes; (2) occasional degenerative changes of axons and myelin in peripheral nerve or within the spinal cord, which may contain nests of phagocytic cells, were termed mild to moderate degeneration; and (3) lesions were considered severe when there was almost complete destruction of axons and myelin in a given tract such as the anterior columns or within extensive areas of peripheral nerve.

Only hens exposed to one of the highest two concentrations of 2-hexanone, 400 or 200 ppm, lost significant weight at the onset of ataxia; weight loss for these two groups continued, and the hens exposed to 400 and 200 ppm 2-hexanone weighed 48.0 ± 7.4 and $63.1 \pm 5.5\%$ (mean \pm SE) of the initial weights, respectively, at the onset of paralysis. Although the group

exposed to 100 ppm 2-hexanone gained some weight at the onset of ataxia, they lost 24.4% of their initial weight after 69 days of exposure. This weight loss coincided with the development of severe ataxia. This treatment group, however, regained all lost weight by the end of the 30-day observation period. No appreciable change in weight was observed in hens exposed to 50 or 10 ppm 2-hexanone.

None of the hens continually exposed to 2-hexanone vapor showed any signs of acute toxicity that are attributed to the narcotizing effects of 2-hexanone on the CNS. All hens continually exposed to 50–400 ppm 2-hexanone developed ataxia after a latent period of 6–30 days, depending on 2-hexanone concentrations. Those exposed to 400 ppm progressed to paralysis, and two died 27 days after the beginning of exposure. The remaining three chickens were in a distressed condition and were sacrificed at 31 days. The number of days of exposure to 2-hexanone vapors before the onset of ataxia was dependent on and inversely proportional to the concentration of 2-hexanone.

All hens exposed to 200 ppm 2-hexanone developed paralysis 64–72 days after the beginning of the exposure; one of these hens died at day 72 and the other four were sacrificed on day 73. Four of the hens inhaling 100 ppm 2-hexanone developed severe ataxia (T_3), while the fifth bird progressed to ataxia with near paralysis (T_4). Three hens of the group exposed to 50 ppm 2-hexanone showed severe ataxia (T_3), while the other two developed only gross ataxia (T_2). The clinical condition of all hens in this group was gross ataxia (T_2) at termination. All hens exposed to 10 ppm 2-hexanone remained normal.

Histopathological lesions in the spinal cord were dependent on concentration, duration of exposure, and duration of intoxication. Two of the hens exposed to 400 ppm did not exhibit any histopathological alterations, while another two showed equivocal changes. Hens exposed to 100 ppm 2-hexanone exhibited clinical signs of neurotoxicity for 99 ± 2 days, and all hens showed unequivocal changes in the spinal cord. Although hens exposed to 50 ppm 2-hexanone were exposed for a mean of 97 days, only four of these hens had unequivocal changes in the spinal cord. Similarly, the presence of histopathological lesions in peripheral nerves was a function of both the level of 2-hexanone inhaled and, particularly, the total dose inhaled. Although all five hens exposed to 100 ppm for 90 days survived until termination on day 120, they showed gross to severe ataxia and each had unequivocal lesions in peripheral nerves. Hens given high doses became paralyzed and thus could not be kept alive as long as those given 100 ppm 2-hexanone.

4-Month study: rats

Groups of six young adult rats (strain and sex not specified) were exposed to 1300 ppm (5325 mg/m^3) of 2-hexanone 6 hours/day, 5 days/week for up to 4 months (Spencer et al., 1975). Three rats were exposed to air only. Animals were observed for neurological signs, and histopathological examinations of several peripheral nerves, regions of the spinal cord, medulla,

and cerebellum were completed. In the exposed rats, narcosis, loss of coordination, weight loss (data not presented), foot drop, and proximal hind-limb and forelimb weakness were observed. Pathological alterations included nerve fiber degeneration in the peripheral nerves, spinal cord, medulla, and cerebellum; axonal dilatation with localized fiber swelling; and secondary paranodal myelin retraction.

6-Month study: male rats

Duckett et al. (1979) exposed groups of Wistar rats (sex not specified) to 0 (n = 20) or 50 ppm (n = 40) 2-hexanone (0 or 205 mg/m³) 8 hours/day, 5 days/week for 6 months. No overt signs of toxicity were observed during the study. Electrophysiological evaluations were performed on 5 treated and 10 control rats at the end of the experiment. The mean sciatic motor conduction velocity (MCV) in the exposed group was significantly lower ($p = 0.005$) than in the controls. No effect on the amplitude of the evoked muscle action potential (MAP) was observed. Widespread demyelination of the sciatic nerve was reported in 32 rats from the exposed group; two of the rats also had axonal hypertrophy and beading. No abnormalities were seen in the sciatic nerves of control rats. The study authors reported that the histopathology of the CNS, liver, and kidney of all rats was normal (details were not provided) (Duckett et al., 1979).

72-Week study: male rats

Krasavage and O'Donoghue (1977) exposed male Sprague-Dawley rats (18/group) to 0, 100, or 330 ppm (0, 410, or 1353 mg/m³) 2-hexanone (purity not specified) 6 hours/day, 5 days/week for 72 weeks (for further experimental detail, see Section 4.2.2.2). Exposure to 100 ppm did not cause clinical or pathologic evidence of neurological damage. One rat exposed to the high concentration developed progressive hind-limb weakness; another three high concentration animals showed slight weakness that was not progressive. One animal in the high concentration group developed a severe polyradiculoneuritis of the nerve roots in the lumbar and sacral spinal nerves and in the sciatic and tibial nerves. The authors concluded that chronic exposure to 100 ppm 2-hexanone was not neurotoxic, while findings at 330 ppm were equivocal (Krasavage and O'Donoghue 1977).

6-Month study: male rats

Male Sprague-Dawley rats (six/group) were exposed to 0 or 100 ppm (0 or 410 mg/m³) 2-hexanone (purity 96.66%, 2.9% MiBK) 22 hours/day, 7 days/week for 6 months (Egan et al., 1980). Two animals from each group underwent microscopic examination for neuropathologic changes following 2, 4, and 6 months of exposure. No treated or control animals displayed clinical signs of neurotoxicity during the exposure period. After four months of exposure, a typical pattern of 2-hexanone-induced neuropathology began to appear in the CNS and peripheral nervous system (PNS). At this time, PNS specimens revealed giant axonal swellings

and secondary demyelination in a few large diameter fibers in the tibial nerve branches to the calf muscles. In the CNS, isolated giant axonal swellings were found in the medulla oblongata and cerebellum. By six months, more advanced degeneration was presented in teased fibers in calf muscle branches and giant axonal swelling had ascended to the level of the sciatic notch. The spinal cord revealed scattered fiber degeneration in the ventral portion of the gracile tract and the caudal portion of descending fiber tracts in the lumbar region.

10-Month study: male rats, male monkeys

Johnson et al. (1977) exposed male Sprague-Dawley rats (10/per group) and male monkeys (*Macaca fascicularis*) (8/group) to 0, 100, or 1000 ppm (0, 410, or 4100 mg/m³) commercial grade 2-hexanone (purity not stated) for 6 hours per day, 5 days per week for up to 10 months. Rats in the 1000-ppm exposure group exhibited progressive body weight loss beginning at 16 weeks and reaching statistical significance at 20 weeks ($p < 0.01$). Monkeys in the 1000-ppm group progressively lost body weight beginning at 8 weeks. No significant effect of 2-hexanone on body weight of rats or monkeys was found in the low-dose exposure groups.

Four neurological tests were conducted on both rats and monkeys: MCV of right sciatic-tibial nerves, MCV of the right ulnar nerve, absolute refractory period of these nerves, and MAP recorded in response to both sciatic and ulnar stimulation. In addition, electroencephalograms and visual evoked potentials were recorded from monkeys. All animals were administered an anesthetic prior to neurological testing: rats received an i.p. injection of 35 mg/kg of sodium pentobarbital, and monkeys were given 15 mg/kg of ketamine hydrochloride intramuscularly.

After 25 weeks, all rats and monkeys in the high-dose exposure group were removed from further exposure because neuropathy (hind-limb drag) apparently had developed. All eight monkeys in the 100 ppm group were exposed for a total of 41 weeks. Rats in the low-dose group were removed from 2-hexanone exposures after 29 weeks. Beginning at 3 months of exposure, monkeys in the 1000 ppm group showed a progressive decrease in the MCV of the sciatic-tibial nerves. After 6 months, the mean MCV of this group was 63% of the mean of control animals. Commencing at 9 months, the MCV for the sciatic-tibial nerves in monkeys in the 100 ppm group was significantly different from control values ($p = 0.05$). At the termination of the study, the MCV of monkeys from the 100 ppm group was 12% less than in the corresponding controls ($p < 0.05$).

A similar pattern of sciatic-tibial neuropathy developed in rats exposed to the higher concentration of 2-hexanone. A significant decrease in MCV was observed at approximately 3 months (13 weeks) of exposure ($p = 0.05$). A significant difference at 8 weeks between MCVs of control and 1000 ppm rats was considered spurious. In the 100 ppm group, a significant difference in MCVs between controls and treated rats occurred at 29 weeks ($p < 0.001$).

A neuropathy similar to that observed for the sciatic-tibial nerves was noted in the ulnar nerve of both the monkeys and rats. When compared with controls, commencing at 4 months,

monkeys showed a progressive decrease in the MCV of the ulnar nerve. At the end of 6 months' exposure to 1000 ppm, monkeys showed a significant decrease in ulnar MCV with values approximately 64% of those of controls ($p < 0.01$). Ulnar MCVs in the 100 ppm group showed a similar decreasing trend at about 6 months; however, these values were not statistically different from controls. In rats, ulnar MCVs were significantly decreased compared with control values ($p < 0.05$), beginning at about 17 weeks in both exposed groups.

Both monkeys and rats exposed to 1000 ppm 2-hexanone showed a continuous decrease in MAP amplitude in response to sciatic stimulation that became statistically significant in monkeys at 6 months ($p < 0.01$). This effect was not noted in the low-dose group of monkeys. Rats in the 100 ppm group had reduced MAP amplitudes for sciatic stimulation, beginning at 12 weeks. No effects of 2-hexanone on scalp-recorded electroencephalograms (EEGs) of monkeys were observed. Amplitude measures of the EEG were not affected at either exposure concentration. Visual examination of the EEG records did not reveal any abnormal patterns (e.g., spikes or abnormal waves).

Evidence of 2-hexanone-induced effects on average visual evoked potential (AVEP) was obtained in monkeys exposed to 1000 ppm. Specifically, latencies of certain AVEP components were increased beginning at 4 months. No effects on these latencies occurred as a result of the low-dose 2-hexanone exposure. The refractory time (i.e., the time that must elapse between two consecutive stimuli of a nerve in order for the second stimulus to also excite the nerve) was not affected by 2-hexanone at either level of exposure.

Only rats were examined for effects of 2-hexanone on operant behavior at 10 and 19 weeks of exposure to 100 and 1000 ppm, respectively. For operant behavior, animals were trained on a multiple fixed ratio of 5, fixed interval 3-minute (multi-FR5FI3) schedule for 20–40 days after shaping the bar press response. Once behavior was stable, animals were placed in exposure chambers and tested after exposure. A reduction in response rate in the 1000 ppm group developed by the second week of exposure; however, no effects of 2-hexanone on operant behavior were found with the 100 ppm group (Johnson et al., 1977).

2-Year study: cats

Groups of four domestic shorthair cats were exposed by inhalation to 0, 100, or 330 ppm (0, 410, or 1353 mg/m³) 2-hexanone (purity not specified) for 6 hours/day, 5 days/week for 2 years (O'Donoghue and Krasavage, 1979). Clinical signs and body weights were monitored (for details, see Section 4.2.2.2). To follow the onset of neuropathy, biopsy specimens were collected from two randomly selected cats in each group at six intervals for the first 9 months of the exposure period. All specimens were taken from alternate hind paws and included 5–6 Pacinian corpuscles and plantar interosseous muscles. Cats were sacrificed at the end of the treatment and underwent gross and histopathologic examinations, and the nervous system was examined microscopically in detail.

No clinical neurological effects attributed to exposure to 2-hexanone were identified. Neuropathologic examination results for the control and low-dose groups were comparable. All cats in the high-dose group showed evidence of neuropathologic changes in the CNS and the PNS at and below the level of the cerebellum and pons (O'Donoghue and Krasavage, 1979). In the PNS, the highest incidences of change occurred in the tibial motor nerve branches to the musculature of the lower leg and then in the tibial nerve itself. In the branches, endoneural space was enlarged with clear fluid. Swelling of giant axons with myelin retraction was evident, and degenerating axons were found infrequently. No changes were found in the dorsal root ganglion cells. In the distal portion of the PNS in the high-dose animals, unusually large preterminal axonal processes were evident, a condition not seen in controls. Examinations of tibial nerve fibers indicated comparable percentages of the four fiber pathology categories (i.e., demyelination, remyelination, swelling, and degenerative fibers) in the control and low-dose groups, but the high-dose group had notable changes in each fiber pathology category except degenerative fibers. Demyelination, remyelination, swelling, and degeneration occurred in 12.3, 3.4, 6.3, and 0.4% of high-dose axons examined (average number of high-dose axons examined = 158), compared with 0, 0.3, 0, and 0.6% of control axons (average number of control axons examined = 84). In the CNS, swollen terminals were found in the posterior cerebellar peduncles, folial white matter, nucleus gracilis, fasciculus gracilis, spino-cerebellar tracts, medullary reticular formation, and all levels of the spinal cord.

4.4.1.3. Other Routes of Exposure

11-Month study: dogs (subcutaneous injections)

O'Donoghue and Krasavage (1981) administered 2-hexanone (>97% pure, with 2.9% MiBK and trace quantities of 2-hexanol) by daily subcutaneous (s.c.) injection to purebred male beagles (n = 4) for 11 months. Each dog received 300 mg/kg of the test compound or saline at first once daily and later (time not stated) divided into two equal doses 6 hours apart. All animals developed signs of neurotoxicity to varying degrees. The patellar reflex was lost unilaterally in two of the four dogs receiving 133 grams of 2-hexanone over a period of 96 days. One month later, the patellar reflex was lost bilaterally in both dogs, and clinical signs of neurotoxicity progressed with observations of muscle weakness and difficulty walking. The condition of both dogs gradually reversed during the course of the study, following an unspecified cessation of exposure. In the remaining two dogs, the clinical signs of neurotoxicity appeared later in the study or were apparent at study completion. In one dog, the patellar reflex could not be elicited after it had received 243 grams of 2-hexanone over a period of 156 days. Following cessation of exposure, the dog returned to apparent normality in approximately 56 days. In the remaining dog, no clear neuropathic abnormality was produced, but, although the patellar reflex was present, the response appeared sluggish. There was occasional evidence of hind-limb weakness.

Mean body weights of treated animals were comparable with those of controls, but individual animals showed weight loss or decreased weight gain. Hematology, clinical chemistry, and cerebrospinal fluid analysis were not affected by the treatment. Repeated biopsy examinations of distal peripheral nerves showed typical giant axonal swelling. The biopsy findings paralleled the clinical course except during a recovery phase, where the biopsy continued to be abnormal while the clinical course improved. Electromyographic examination of the treated dogs showed the persistence of abnormalities in two recovering dogs, no abnormalities in one recovering dog, and no abnormalities in the one dog that had appeared clinically normal throughout the study (O'Donoghue and Krasavage, 1981).

90-Day study: hens (intraperitoneal injections)

Abou-Donia et al. (1982) treated five groups of leghorn laying hens (*Gallus gallus domesticus*, n = 3) with daily injections of 2-hexanone (70% 2-hexanone and 30% methyl isobutyl ketone, i.p.) at 100 or 200 mg/kg for 90 days. Hens given a daily 100 mg/kg i.p. injection of 2-hexanone progressed through all successive stages of ataxia; the clinical conditions of two of them improved after treatment was stopped, while the third hen progressed to paralysis and died after 30 days of administration. Daily i.p. injection of 200 mg/kg 2-hexanone produced ataxia with near paralysis (T₄), which progressed to paralysis in one hen. The clinical condition of this hen, however, reverted to grade T₄ after cessation of administration.

Spinal cords from hens given daily i.p. 100 mg/kg injections of 2-hexanone did not exhibit any histopathologic changes. One of these hens, however, showed unequivocal histopathologic changes in the peripheral nerves. The sites of axonal degeneration were accompanied by myelin degeneration, and macrophages were observed containing debris with the staining properties of myelin. Although none of the hens given 200 mg/kg i.p. injections of 2-hexanone showed histopathologic alterations in peripheral nerves, two of these hens developed unequivocal histopathologic lesions in the spinal cord. A longitudinal section from the ventral column of the thoracic spinal cord from one of the hens showed axons with prominent swellings. These swellings have the morphologic configuration of the paranodal swelling that suddenly ends at the nodes of Ranvier. A longitudinal section of the thoracic spinal cord from the other affected hen demonstrated extensive degeneration in the ventral column and a markedly swollen axon and nests of macrophages.

4.4.2. Immunotoxicity Studies

No studies were located regarding immunological effects in humans by any route of exposure to 2-hexanone.

A reduction in total white blood cell counts to 60% of control values ($p < 0.05$), but no changes in differential white cell counts or evidence of bone marrow damage, was found in rats intermittently exposed to 700 ppm 2-hexanone after 8 weeks, during an 11-week study (Katz et

al., 1980). These findings, although inconclusive, suggest that immunological effects may warrant some consideration in future assessments of the potential toxicity of exposure to 2-hexanone.

4.5. OTHER STUDIES

4.5.1. Mechanistic Studies

4.5.1.1. 2-Hexanone and Enzyme Induction

2-Hexanone and its neurotoxic metabolite 2,5-hexanedione are both effective inducers of microsomal enzyme activities. This can affect the toxicity of other xenobiotics and also can affect the toxicity of 2-hexanone itself (or its precursor, n-hexane) by increasing or decreasing the formation of toxic metabolites.

Nakajima et al. (1991) characterized the CYP450 enzymes in the livers of male Wistar rats that are induced following exposure to 2-hexanone (5 mmol/kg-day), 2,5-hexanedione (5 mmol/kg-day), or phenobarbital (80 mg/kg-day), administered intraperitoneally for 4 days. A control group received an equivalent volume of corn oil vehicle (4 mL/kg). All three treatments caused a statistically significant increase in microsomal protein content and overall CYP450 activity (Table 4-9).

Table 4-9. Effects of 2-hexanone, 2,5-hexanedione, and phenobarbital on microsomal protein and CYP450

Treatment	Body weight (g)	Liver weight (g)	Liver/body weight ratio (%)	Microsomal protein (mg/g liver)	CYP450 (nmol/mg protein)
Control	206 ± 7	6.6 ± 0.2	3.21 ± 0.11	21.5 ± 0.8	0.92 ± 0.002
2-Hexanone	192 ± 6	7.3 ± 0.3 ^a	3.80 ± 0.05 ^a	25.1 ± 1.5 ^a	1.49 ± 0.10 ^a
2,5-Hexanedione	184 ± 7 ^a	6.4 ± 0.3	3.49 ± 0.07 ^a	26.2 ± 1.7 ^a	1.62 ± 0.10 ^a
Phenobarbital	197 ± 5	7.9 ± 0.4 ^a	4.01 ± 0.13 ^a	31.5 ± 3.0 ^a	2.12 ± 0.19 ^a

^aSignificantly different ($p < 0.05$) from control.

Source: Nakajima et al. (1991).

The enzyme activities (i.e., benzene aromatic hydroxylase [CYP2E1], toluene side chain oxidation [CYP2C6/11], EROD [CYP1A1/2], and PROD [CYP2B1/2]) were measured as indicators of CYP450 activity. All three treatments caused a statistically significant increase in the rate of benzene hydroxylation at low (0.2 mM) and high (6.3 mM) concentrations and toluene side chain oxidation at low (0.2 mM) and high (5.0 mM) concentrations. EROD activity was not affected by pretreatment; however, a statistically significant increase in PROD activity was observed with all three treatments. A summary of the results for the CYP450 activity measured with specific substrates is listed in Table 4-10.

Table 4-10. Effect of enzyme inducers on the activities of CYP450-related enzymes in rats exposed to 2-hexanone or 2,5-hexanedione

Treatment	Enzyme activity					
	BAH ^a		TSO ^b		EROD	PROD
	0.2 mM	6.3 mM	0.2 mM	5.0 mM		
Control	0.68 ± 0.09	0.53 ± 0.11	1.87 ± 0.15	8.34 ± 0.67	0.32 ± 0.06	0.11 ± 0.02
2-Hexanone	1.10 ± 0.19 ^c	1.76 ± 0.23 ^{c,d}	5.65 ± 0.62 ^c	19.07 ± 1.64 ^{c,e}	0.41 ± 0.30	3.68 ± 0.70 ^c
2,5-Hexanedione	0.98 ± 0.16 ^c	1.57 ± 0.15 ^{c,d}	5.05 ± 0.46 ^c	19.98 ± 0.78 ^{c,e}	0.26 ± 0.44	2.92 ± 0.90 ^c
Phenobarbital	0.48 ± 0.11 ^c	2.80 ± 0.23 ^{c,d}	5.59 ± 0.87 ^c	25.36 ± 6.23 ^{c,e}	0.27 ± 0.04	5.22 ± 0.70 ^c

^aBAH = benzene aromatic hydroxylase.

^bTSO = toluene side-chain oxidase.

^cSignificantly different ($p < 0.05$) from control.

^dSignificant difference ($p < 0.05$) between 0.2 and 6.3 mM of the corresponding group.

^eSignificant difference ($p < 0.05$) between 0.2 and 5.0 mM of the corresponding group.

Source: Nakajima et al. (1991).

Using immunoblotting and immunodetection assays, Nakajima et al. (1991) did not detect CYP4501A1/2 in microsomes from treated and control animals. CYP4502B1/2 was induced by treatment with phenobarbital > 2-hexanone = 2,5-hexanedione. Only trace amounts of CYP4502E1 were detected in phenobarbital-treated rats, whereas 2-hexanone and 2,5-hexanedione both induced this isoform efficiently.

In order to explore the effects of 2-hexanone, 2,5-hexanedione, and phenobarbital on CYP4502B1/2, CYP4502E1, and CYP4502C6/11, Nakajima et al. (1991) performed immunoinhibition analyses of toluene side-chain oxidase (TSO) activity by using monoclonal antibodies directed against each of these CYP450 isoforms. Anti-CYP4502E1 inhibited TSO activity in induced microsomes as follows (values are percent of activity in the absence of anti-CYP4502E1): phenobarbital, 97 ± 2%; 2,5-hexanedione, 79 ± 3%; 2-hexanone, 75 ± 11%; and controls, 65 ± 2%. Anti-CYP4502B1/2 inhibited TSO activity in induced microsomes differently: phenobarbital, 31 ± 4%; 2-hexanone, 65 ± 3%; 2,5-hexanedione, 69 ± 5%; and controls, 99 ± 2%. Anti-CYP4502C6/11 inhibited toluene metabolism in induced microsomes as follows: phenobarbital, 75 ± 5%; 2-hexanone, 69 ± 5%; 2,5-hexanedione, 70 ± 3%; and controls, 23 ± 4%.

Similar studies were performed by Imaoka and Funae (1991). The authors treated male Sprague-Dawley rats (number of rats not provided) with 2-hexanone (purity not stated; 5 mmol/kg, i.p.; dissolved in corn oil) daily for 4 days. This dose was considered a maximum tolerated dose. Control rats were given corn oil only. Hepatic microsomes were isolated, and the activities of CYP450 enzymes were determined against specific substrates (Table 4-11).

Table 4-11. Catalytic activities of CYP450 enzyme activities in rat liver following induction by 2-hexanone

Substrate	Enzyme activity (nmol/min-mg protein) ^a	
	Uninduced control	2-Hexanone-treated
Aminopyrine	2.40 ± 0.50	4.37 ± 0.82 ^b
Aniline	0.283 ± 0.044	0.421 ± 0.070 ^b
7-Ethoxycoumarin	3.62 ± 0.13	6.01 ± 1.24 ^b
Testosterone-2 α	0.684 ± 0.114	0.431 ± 0.158 ^c
Testosterone-2 β	0.140 ± 0.039	0.240 ± 0.056 ^c
Testosterone-6 β	0.959 ± 0.176	1.45 ± 0.341 ^c
Testosterone-7 α	0.056 ± 0.006	0.062 ± 0.013
Testosterone-15 α	0.040 ± 0.007	0.056 ± 0.017
Testosterone-16 α	1.09 ± 0.203	1.07 ± 0.347
Testosterone-16 β	0.058 ± 0.006	0.250 ± 0.106 ^c

^aMean ± SD, number of rats not provided.

^bSignificantly different from control, $p < 0.01$.

^cSignificantly different from control, $p < 0.05$.

Source: Imakoa and Funae (1991).

The content of total CYP450 measured photometrically did not change much with treatment. However, the activities of aminopyrine N-demethylase, aniline hydroxylase, and 7-ethoxycoumarin O-dealkylase were increased by pretreatment with 2-hexanone. Testosterone 2 β -, 6 β -, and 16 β -hydroxylase activities were significantly increased, whereas the 2 α -hydroxylase activity was decreased by treatment with 2-hexanone. The authors also measured changes in the levels of 11 forms of CYP450 in hepatic microsomes caused by treatment with 2-hexanone (Table 4-12).

The level of CYP4502C11, a male-specific form, was decreased by treatment with 2-hexanone in parallel with a decrease in testosterone 2 α -hydroxylase activity, which is catalyzed by this isozyme (Kamataki et al., 1983) (cf. Table 4-12). CYP4502A2 is a constitutive testosterone 6 β -hydroxylase; the increase in the level of this isoform explained the increase in testosterone 6 β -hydroxylase activity, shown in Table 4-11. CYP4502B1 and 2B2 are typical phenobarbital-inducible forms. The level of CYP4502B1 in the hepatic microsomes of control rats was very low, and CYP4502B2 was detected at a slightly higher level. Both forms were strongly induced in 2-hexanone-treated rats. These results reflected the increases in testosterone 16 β -hydroxylase and aminopyrine N-demethylase activities of hepatic microsomes (cf. Table 4-11) and suggest that 2-hexanone is a phenobarbital-type inducer.

Table 4-12. Changes in CYP450 levels following treatment with 2-hexanone

CYP450 isoform	CYP450 content (pmol/mg protein) ^a	
	Uninduced control	2-Hexanone-treated
2A1	7.0 ± 1.3	7.9 ± 1.5
2A2	10.4 ± 2.3	11.7 ± 2.8
2B1	<0.5	44.3 ± 9.4 ^c
2B2	3.8 ± 1.2	29.3 ± 6.2 ^c
2C6	52.1 ± 17.7	93.4 ± 16.9 ^b
2C7	21.9 ± 3.3	24.8 ± 5.8
2C11	457.0 ± 52.6	343.8 ± 46.3 ^c
2C13	171.4 ± 35.8	159.7 ± 24.5
2E1	49.8 ± 9.6	102.6 ± 14.8 ^b
4A3	17.6 ± 3.2	16.7 ± 2.8

^aMean ± SD, number of rats not provided.

^bSignificantly different from control, $p < 0.01$.

^cSignificantly different from control, $p < 0.05$.

Source: Imaoka and Funae (1991).

Imaoka and Funae (1991) determined that the inducibility of CYP4502B1 and 2B2 was strongly correlated with the hydrophobicity (as estimated by the octanol/water partition coefficients, log K_{ow}) of several 2-hexanone homologues: 2-hexanone (1.38) > methyl n-propyl ketone (0.91) > MEK (0.29) > acetone (−0.24). In contrast, the inducibility of P4502E1 was not dependent on hydrophobicity. Each of the aforementioned chemicals, at equimolar concentrations, induced CYP4502E1 to a similar extent, approximately twofold, while acetone, a prototypical inducer of CYP2E1, induced this isoform approximately threefold.

Based on studies of 2-hexanone and the pesticide O-ethyl O-4-nitrophenyl phenylphosphonothioate (EPN) in hens, Abou-Donia et al. (1991, 1985) speculated that the potentiation of the neurotoxic effects of 2-hexanone by EPN may be due to induction of hepatic microsomal CYP450 by EPN with increased production of 2,5-hexanedione (Abou-Donia et al., 1991, 1985a). Similarly, MEK may also potentiate the toxicity of 2-hexanone through induction of CYP450 as MEK but not 2-hexanone and has been shown to decrease hexobarbital sleep time in rats (Couri et al., 1977). While MEK has been shown to potentiate the toxicity of 2-hexanone in rats (Saida et al., 1976), Shibata et al. (2002) have demonstrated that MEK depresses the metabolism of n-hexane in human volunteer subjects. If the metabolic pathways of 2-hexanone, as detailed in Section 3.3 and Figure 3-1, are common in humans and animals and MEK depresses the metabolism of n-hexane but increases the metabolism of 2-hexanone, then the step in 2-hexanone metabolism that MEK likely affects is the ω -1-oxidation to 5-hydroxy-2-hexanone. While no specific CYP450 isoenzymes have been implicated and the mechanisms are not fully elucidated, it appears that 2-hexanone has the ability to influence its own metabolism via effects on CYP450 enzymes that need more research to be fully understood.

It should be noted that, like 2-hexanone, MiBK, a common contaminant in the formulation of the 2-hexanone, has the potential to act as a CYP450 inducer. However, the 3.2% concentration of MiBK in 96% pure formulations of 2-hexanone, as reported by O'Donoghue et al. (1978), may not have a significant impact on the toxicity of 2-hexanone. To determine whether the concentration of MiBK as a contaminant may have altered the observed toxicity of 2-hexanone, other studies were evaluated that used MiBK as a test article. In a 13-week gavage study, 30 male and female Sprague-Dawley rats were treated daily with 0, 50, 250, or 1,000 mg MiBK/kg-bw (MAI, 1986). At the middle and high doses, adverse effects were observed in the liver and kidney, which progressed in severity in the high-dose animals. No treatment-related effects of any kind were observed at 50 mg/kg-day. The Carnegie-Mellon Institute of Research (1977) conducted a 120-day drinking water study with 1.3% MiBK, using female HLA Wistar rats. The authors estimated the dosage to be 1040 mg/kg-day. The only statistically significant finding was increased mean absolute and relative kidney weights in treated rats compared with controls. Histopathological examination revealed renal tubular cell hyperplasia in only one of five of the treated rats. No exposure-related histopathological changes were found in other organs. Based on the foregoing, it can be concluded that the dosage of MiBK received as an impurity in the study by O'Donoghue et al. (1978) did not contribute to the observed 2-hexanone-related effects. O'Donoghue et al. (1978) did not observe adverse effects in the kidney or liver of treated animals, despite these organs being the target organs of toxicity in experimental studies with MiBK from both the oral and inhalation routes (U.S. EPA, 2003a).

4.5.1.2. 2-Hexanone as a Sulfhydryl Reagent

Both 2-hexanone and its metabolite 2,5-hexanedione can inhibit sulfhydryl-containing enzymes such as fructose-6-phosphate kinase and glyceraldehyde-3-phosphate dehydrogenase (enzymes in the pentose phosphate pathway [oxidative phase] and glycolytic pathway [nonoxidative phase], respectively) (Sabri, 1984; Sabri et al., 1979). Both of these chemicals inhibited fructose-6-phosphate kinase from rabbit muscle or rat brain homogenates; in each case, 2,5-hexanedione was the far more potent inhibitor (Sabri et al., 1979). Preincubation with dithiothreitol protected this enzyme from inhibition, which suggests that these compounds interfere with the sulfhydryl groups required for fructose-6-phosphate kinase activity. However, dithiothreitol could not restore enzyme activity after these compounds had been added. In addition, fructose-6-phosphate kinase activity was also reduced in brain homogenates of rats that had received 2,5-hexanedione at 0.5% in their drinking water for 10–12 weeks (Sabri et al., 1979). Glyceraldehyde-3-phosphate dehydrogenase from rabbit muscle (purified to crystalline state) was also inhibited in vitro by both compounds; in this case, 2-hexanone was the more potent inhibitor (Sabri, 1984). Levels of ATP were reduced in cat sciatic nerves treated with 2,5-hexanedione (Sabri, 1984), possibly an outcome of glyceraldehyde-3-phosphate

dehydrogenase inhibition. 2-Hexanone was found to irreversibly inhibit rat brain and rabbit muscle creatine kinase and mouse brain adenylate kinase (Lapin et al., 1982).

4.5.1.3. *Studies Exploring the Development of Neuropathy*

Groups of 12 Sprague-Dawley rats (sex unspecified) were continuously exposed (24 hours/day) via inhalation to 0, 225, or 400 ppm (0, 922.5, or 1,640 mg/m³) 2-hexanone (purity not stated) for 16–66 days (Saida et al., 1976). Rats exposed to 400 ppm were sacrificed at 16, 28, and 42 days, and those exposed to 225 ppm were sacrificed at 16, 25, 35, 55, and 66 days to study the sequence of morphological changes. Paralysis was observed after 66 and 42 days at the low and high concentrations, respectively. Neuropathologic changes preceded paralysis and were observed at the initial sacrifice after 16 days of exposure. Two distinct changes occurred quite early and close to the same time: the first to appear was an increase in the number of neurofilaments and the other was an in-pouching of the myelin sheath. In animals exposed to 400 ppm, the first observable change at 16 days was, in larger diameter nerve fibers, a two- to threefold increase in the number of neurofilaments. As the duration of exposure lengthened and the number of neurofilaments increased, several interrelated morphological observations were made. In teased nerve fiber preparations, swelling of the axons could be seen frequently in the paranodal area and less often at focal sites along the internodal segment. High numbers of nerve fibers with in-pouching of the myelin sheath were found per mm² of nerve fascicle, increasing with time after administration of the high concentration. A summary of the comparative sequential clinical and pathological observations is presented in Table 4-13.

Table 4-13. Clinical and pathological observations with time of exposure to 2-hexanone in rats

Days exposed	2-Hexanone exposure						
	400 ppm			225 ppm			
	16	28	42	16	25	35	55
Clinical findings	N ^a	N	P ^b	N	N	N	N
In-pouchings (#/mm ²)	6	142	499	23	46	92	86
Denuded fibers (#/mm ²)	0	4	11	0	0	1	2
Swollen axons >11 µm (#/300 fibers)	0	1	3	0	0	0	0

^aN = normal.

^bP = paralyzed.

Source: Saida et al. (1976).

The anterior horn cells, nerve roots, nerve trunks, intramuscular nerves, and motor end plates were studied sequentially to determine the site with the earliest pathological involvement. In animals exposed for 16 days to 225 ppm, no abnormalities were found in the motor end plates

or intramuscular nerves of the intrinsic foot muscles. Only after prolonged exposure, 66 days, did the authors find typical signs of denervation in the motor end plates. These end plates showed atrophic axon terminals with Schwann cell processes interposed between the nerve terminal and postsynaptic membrane. There was also a loss of secondary synaptic clefts.

Anterior horn cells and dorsal root ganglion cells were also examined at various intervals of exposure. No changes were observed in these cell bodies, even after typical changes were seen in the main trunk of the sciatic nerve. Specifically, no abnormalities were seen that would suggest an increase in neurofilaments in these cell bodies, and no cells were observed undergoing chromatolysis.

4.5.2. Genotoxicity Studies

Mayer and Goin (1994) tested the ability of 2-hexanone to induce chromosome loss in strain D61.M of *Saccharomyces cerevisiae*. 2-Hexanone, alone or in combination with acetone and MEK, induced only a marginally positive chromosome loss (Mayer and Goin, 1994).

No data were identified for the mutagenicity of 2-hexanone with in vitro cytogenetic tests or in vivo tests.

4.5.3. Structure-Activity Relationships

A large body of toxicological information is available on n-hexane, a compound that is metabolized to 2-hexanone, on MiBK (a branched-chain homologue of 2-hexanone), and on MEK. These compounds have been reviewed in previous IRIS assessments, and a summary of the reference values derived for each is presented in Table 4-14. n-Hexane is the only compound of the above three that is also capable of producing the peripheral neuropathy similar to that observed in humans or animals exposed to 2-hexanone. Neither MiBK nor MEK can give rise to the neurotoxic metabolite 2,5-hexanedione.

Table 4-14. Summary of the toxicities of n-hexane, MiBK, and MEK

Chemical	Experimental dose	Critical effect	Reference value	Reference
n-Hexane (CASRN 110-54-3)	NOAEL ^a : 1762 mg/m ³	Peripheral neuropathy (decreased MCV at 12 weeks)	RfC: 7×10^{-1} mg/m ³	U.S. EPA (2005c)
Methyl isobutyl ketone (CASRN 108-10-1)	NOAEL: 1229 mg/m ³	Reduced fetal body weight, increased fetal death, and skeletal variations in mice and rats	RfC: 3 mg/m ³	U.S. EPA (2003a)
Methyl ethyl ketone (CASRN 78-93-3)	LEC ^b : 5202 mg/m ³	Developmental toxicity (skeletal variations)	RfC: 5 mg/m ³	U.S. EPA (2003b)
	NOAEL: 594 mg/kg-day (0.3% 2-butanol)	Decreased pup body weight	RfD: 0.6 mg/kg-day	

^aNOAEL = no-observed-adverse-effect level.

^bLEC = lowest effective concentration.

4.5.4. Potentiation and Other Interaction Studies

4.5.4.1. Methyl Ethyl Ketone

In a study of chemical interaction, Saida et al. (1976) exposed rats of unspecified sex (12/group) continuously, 24 hours/day, to 225 ppm (922 mg/m³) 2-hexanone, 1125 ppm (3318 mg/m³) MEK, or a combined exposure of 225 ppm (922 mg/m³) 2-hexanone and 1125 ppm MEK for up to 66 days. No signs of neurotoxicity were observed in the MEK-exposed rats. Paralysis occurred earlier in the rats exposed to the mixture compared with rats exposed to 225 ppm 2-hexanone alone. In addition, an elevated severity of neuropathy, in the form of increased swollen axons, denuded fibers, and in-pouching of myelin sheaths, was observed histologically in the rats coexposed to MEK and 2-hexanone. Thus, MEK appeared to potentiate the toxicity of 2-hexanone. Yu et al. (2002) showed that the potentiating effect of MEK on n-hexane-induced neurotoxicity was due to an inhibitory effect of MEK on phase II biotransformation of 2,5-hexanedione (Yu et al., 2002). Since n-hexane is a precursor to 2-hexanone, and both compounds form the highly toxic 2,5-hexanedione, it is likely that the results of Yu et al. (2002) are applicable to coexposure studies with MEK and 2-hexanone.

As a test of in vivo enzyme induction, groups of five male Wistar rats were continuously exposed via inhalation to 225 ppm 2-hexanone, 750 ppm MEK, or the combination of 225 ppm 2-hexanone and 750 ppm MEK for 7 days (Couri et al., 1977). Subsequently, the animals were given sodium hexobarbital (100 mg/kg, i.p.), a substrate for phenobarbital-inducible CYP450 isoenzymes (Adedoyin et al., 1994; Knodell et al., 1988), and sleep time was measured. The average hexobarbital-induced sleep time of 2-hexanone-treated rats was comparable to that of controls (24.8 vs. 26.0 minutes); however, the sleep times in MEK and 2-hexanone/MEK-exposed rats were significantly ($p < 0.05$) less than in controls, 13.0 and 16.0 minutes, respectively. In a study by O'Donoghue and Krasavage (1979), sodium pentobarbital-induced sleep time was increased in 2-hexanone-treated cats.

4.5.4.2. Chloroform

Oral administration of 2-hexanone, followed by i.p. administration of chloroform to rats, resulted in a variety of hepatic and renal effects, including decreased hepatic glutathione levels, increased plasma levels of glutamic pyruvic transaminase and blood urea nitrogen, and degeneration and necrosis of hepatic and renal tissue (Hewitt et al., 1990, 1987; Brown and Hewitt, 1984; Branchflower and Pohl, 1981). Similarly, oral administration of both 2-hexanone and chloroform to rats resulted in altered permeability of the biliary tree (Hewitt et al., 1986). In these studies, some or no effect on the endpoints of interest was observed after administration of 2-hexanone or chloroform alone; administration of both substances resulted in statistically significant and dramatic changes in these effects. The authors speculated that 2-hexanone potentiated the hepatic toxicity of chloroform by decreasing glutathione levels and by increasing the metabolism of chloroform to the potent hepatotoxicant phosgene.

4.5.4.3. *O-Ethyl O-4-Nitrophenyl Phenylphosphonothioate*

2-Hexanone has been shown to potentiate the neurotoxic effects of EPN. In hens, dermal or inhalation exposure to 2-hexanone in combination with dermal application of the organophosphate pesticide EPN has resulted in earlier onset and far more severe clinical and histological manifestations of neurotoxic effects than with either chemical exposure alone (Abou-Donia et al., 1991, 1985a). The authors speculated that this potentiation effect may have been due to induction of hepatic microsomal CYP450 by EPN, leading to increased metabolism of 2-hexanone to its neurotoxic metabolite 2,5-hexanedione. An alternate explanation is that local trauma to the nervous tissue produced by 2-hexanone and EPN might increase vascular permeability and thus increase the entry of these compounds and their metabolites from circulation.

4.6. SYNTHESIS AND EVALUATION OF MAJOR NONCANCER EFFECTS

4.6.1. Oral

There are no studies that have examined the possible association between oral exposure to 2-hexanone and noncancer health effects in humans. There are six subchronic or chronic studies in which 2-hexanone was administered orally to experimental animals. These include a 90-day gavage study in hens, 90-day and 40-week gavage studies in rats, 120-day and 13-month drinking water studies in rats, and a 24-week drinking water study in guinea pigs. These studies demonstrate that the nervous system is the target organ for 2-hexanone toxicity following oral exposure. For example, O'Donoghue et al. (1978), a 13-month drinking water study using COBS CD(SD)BR rats, described the characteristic neuropathologic evidence of giant axonal neuropathy in 80% of animals at the lowest dose tested (143 mg/kg-day).

There are data suggesting that the principal metabolite of 2-hexanone, 2,5-hexanedione, is responsible for the neurotoxicity associated with oral exposure to 2-hexanone. For example, Krasavage et al. (1980) compared the neurotoxicity of 2-hexanone with that of n-hexane, 5-hydroxy-2-hexanone, 2,5-hexanediol, and 2-hexanol by administering equimolar doses of each chemical by gavage to five male COBS CD(SD)BR rats/group, 5 days/week for 90 days. Judged by the time required for the rats to develop hind-limb paralysis, 2,5-hexanedione had a higher neurotoxic potency than 2-hexanone.

In summary, the chronic and subchronic studies conducted with rats, hens, and guinea pigs provide ample evidence that the nervous system is the target of toxicity following oral exposure to 2-hexanone. A summary of the oral studies with 2-hexanone is provided in Table 4-15.

Table 4-15. A synopsis of oral toxicity studies with 2-hexanone

Species, strain	Group size (sex)	Dosage; duration; purity	Effects at LOAEL	NOAEL ^a (mg/kg-day)	LOAEL ^a (mg/kg-day)	Reference
Adult leghorn laying hens (<i>Gallus gallus domesticus</i>)	3/group (female)	100 mg/kg, gavage; 7 days/week for 90 days; technical grade containing 70% 2-hexanone and 30% MiBK	Mild ataxia at 12 ± 1 days with progression to severe ataxia by 50 ± 1 days	Not identified	100	Abou-Donia et al., 1982
Rat, COBS/CD(SD)BR	6/group (male)	660 mg/kg, gavage; 5 days/week for 90 days; 2-hexanone containing 3.2% MiBK and 0.7% unknown contaminants	Clinical and histological findings of neuropathy at 55.8 ± 4.3 days	Not identified	660	Krasavage et al. (1980)
Rat, Wistar	5/group (female)	0, 0.65, or 1.3% (0, 480, or 1010 mg/kg-day) in drinking water; 120 days; purity not stated	Mild atrophy affecting skeletal muscles of the hind limbs in 2 of 5 animals examined	Not identified	480	Homan et al. (1977)
Guinea pig, English short hair	5/group (sex not stated)	0, 0.1, or 0.25% (0, 97, or 243 mg/kg-day) in drinking water; 24 weeks; purity not stated	Decreased pupillary response to light stimulus	Not identified	97	Abdel-Rahman et al. (1978)
Rat, Wistar	6/group (male)	400 mg/kg-day, gavage; 40 weeks; 2-hexanone 98% pure, contaminants not characterized	Hind-limb weakness from the 17 th –28 th week, with improvement thereafter	Not identified	400	Eben et al. (1979)
Rat, COBS/CD(SD)BR	10/group (male)	0, 0.25, 0.5, or 1.0% (0, 143, 266, or 560 mg/kg-day) in drinking water; 13 months; 2-hexanone containing 3.2% MiBK and 0.7% unknown contaminants	Clinical neurological deficits	143	266	O'Donoghue et al. (1978)
			Neuropathologic evidence of myofibrillar atrophy of the calf muscle in 1/10 animals	143	266	
			Neuropathologic evidence of myofibrillar atrophy of the quadriceps muscle in 2/10 animals	143	266	
			Neuropathologic evidence of giant axonal neuropathy in 8/10 animals	Not identified	143	

^aNo-observed-adverse-effect levels (NOAELs) and lowest-observed-adverse-effect levels (LOAELs) determined by 2-hexanone assessment authors

4.6.2. Inhalation

Several studies have established associations between inhalation exposure to 2-hexanone and human health effects. Specifically, occupational studies and case reports suggest that inhalation exposure to 2-hexanone in humans may be associated with neurotoxicity. For example, a cross-sectional study of employees at a coated fabrics plant was conducted when it was noted that six workers from the print department had developed severe peripheral neuropathy soon after the plant began phasing in the use of 2-hexanone (Allen et al., 1974; Billmaier et al., 1974). Definite signs, symptoms and electrodiagnostic findings of peripheral neuropathy were confirmed in 68 out of 192 employees. The prevalence of peripheral neuropathy was clearly increased in jobs with evident exposure to 2-hexanone vapors and with time spent at work sites with 2-hexanone exposure.

Mallov (1976) reported one probable and two definite cases of 2-hexanone-induced peripheral neuropathy that were identified during an investigation of 26 painters. Similar to the studies reported above, (Allen et al., 1974; Billmaier et al., 1974), neuropathy was observed in the painters when the formulation of paint solvents was changed from MEK and methyl isoamyl ketone, both not neurotoxic, to 2-hexanone (Mallov, 1976). In another case of occupational exposure to 2-hexanone, symmetrical polyneuropathy was reported in a furniture finisher (Davenport et al., 1976). Six months prior to the onset of the worker's illness, 2-hexanone had been substituted for MiBK. A similar progressive distal extremity weakness developed in a coworker of the patient, which also improved following the coworker's removal from contact with lacquer products.

The toxicity of 2-hexanone via inhalation was studied extensively in experimental animals. As with oral exposures, the target organ for toxicity following inhalation exposure to 2-hexanone was the nervous system, and the most sensitive measures of intoxication were histopathological and clinical findings of peripheral neuropathy. Numerous studies are available, with duration varying from subchronic to chronic, in many different test species, including monkeys, rats, and cats. A summary of the available inhalation studies with 2-hexanone is provided in Table 4-16.

Table 4-16. Synopsis of animal inhalation toxicity studies with 2-hexanone

Species, strain	Number (sex)	Concentration; duration; purity	Effects at LOAEL	NOAEL ^a (mg/m ³)	LOAEL ^a (mg/m ³)	Reference
<i>Developmental study</i>						
Rat, pregnant F-344	25/group (female)	0, 1000, or 2000 ppm (0, 4100, or 8200 mg/m ³); day 0 of gestation through day 21, 6 h/day, 7 d/wk; purity not stated	Hyperactivity in behavioral testing	Not identified	4100	Peters et al. (1981)
<i>Subchronic exposure studies</i>						
Rat, strain not stated	9/group (sex not stated)	0 or 200 ppm (0 or 819 mg/m ³); 6 weeks, 8 h/d, 5 d/wk; purity not stated	Axonal hypertrophy, beading and degeneration of sciatic nerve	Not identified	819	Duckett et al. (1974)
Rat, Wistar	20/group (sex not stated)	40 ppm (164 mg/m ³); 22–88 days, 8h/d, 5d/wk; purity not stated	Peripheral neuropathy (demyelination of sciatic nerve) in 3/20	164	205	Duckett et al. (1979)
Rat, Sprague-Dawley	12/group (sex not stated)	0, 225, or 400 ppm (0, 922.5, or 1640 mg/m ³); 42–66 days, 24 h/d, 7 d/wk; purity not stated	Increased number of fibers with in-pouchings per mm ² of nerve fascicle	Not identified	922.5	Saida et al. (1976)
Rat, COBS/CD(SD) BR	5/group (male)	0 or 700 ppm (0 or 2870 mg/m ³); 81 days, 72 h/wk; 96.1% pure with 3.2% MiBK and 0.7% unidentified contaminants	Severe neuropathy consisting of difficulty extending hind limbs and a flat-footed gait with feet splayed in 5/5 at 71 ± 9 days	Not identified	2870	Katz et al. (1980)
Adult leghorn laying hens (<i>Gallus gallus domesticus</i>)	5/group	0, 10, 50, 100, 200, or 400 ppm (0, 41, 205, 410, 820, or 1640 mg/m ³); 90 days (continual exposure); technical grade containing 70% 2-hexanone and 30% MiBK	Mild ataxia (27 ± 2 days) progressing to severe ataxia/near paralysis (89 ± 1 days)	41	205	Abdo et al. (1982)
Rat, Sprague-Dawley	4/group (sex not stated)	0 or 400 ppm (0 or 1640 g/m ³) (adjusted); 12 weeks, 24 h/d, 7 d/wk; purity not stated	Dragging of hind limbs at 11–12 weeks	Not identified	1640	Mendell et al. (1974)
Domestic Chickens	5/group (not stated)	0 or 200 ppm (0 or 820 mg/m ³), adjusted to 100 ppm (410 mg/m ³)(time not stated); 12 weeks (24 h/d, 7 d/wk); purity not stated	Inability to stand on legs at 4–5 weeks	Not identified	820	Mendell et al. (1974)
Cat, domestic, strain not stated	4/group (sex not stated)	0 or 400 ppm (1640 mg/m ³) (adjusted); 12 weeks, 24 h/d, 7 d/wk; purity not stated	Dragging of hind limbs and forelimb weakness at 5–8 weeks	Not identified	1640	Mendell et al. (1974)

Table 4-16. Synopsis of animal inhalation toxicity studies with 2-hexanone

Species, strain	Number (sex)	Concentration; duration; purity	Effects at LOAEL	NOAEL ^a (mg/m ³)	LOAEL ^a (mg/m ³)	Reference
Rat, Wistar	20/group (sex not stated)	50 ppm (205 mg/m ³); 13 weeks, 8h/d, 5 d/wk; purity not stated	Peripheral neuropathy (demyelination of sciatic nerve) in 3/20	164	205	Duckett et al. (1979)
<i>Chronic exposure studies</i>						
Rat, strain not stated	6/group (sex not stated)	0 or 1300 ppm (5325 mg/m ³); 4 months, 6 h/d, 5d/w; purity not stated	Nerve fiber degeneration in the peripheral nerves, spinal cord, medulla, and cerebellum	Not identified	5325	Spencer et al. (1975)
Rat, Wistar	40/group (sex not stated)	50 ppm (205 mg/m ³); 6 months, 8h/d, 5 d/wk; purity not stated	Widespread demyelination of the sciatic nerve in 32/40	Not identified	205	Duckett et al. (1979)
Rat, Sprague-Dawley	6/group (male)	0 or 100 ppm (0 or 410 mg/m ³); 6 months, 22 h/d, 7 d/wk; 96.66% pure, impurities not characterized	Giant axonal swelling of peripheral nerves after 4 months	Not identified	410	Egan et al. (1980)
Rat, Sprague-Dawley	10/group (male)	0, 100, or 1000 ppm (0, 410, or 4100 mg/m ³); 10 months, 6 h/d, 5d/wk; commercial grade, impurities not stated	Decreased motor conduction velocity between treated and control animals beginning at 29 weeks	Not identified	410	Johnson et al. (1977)
Monkey, <i>Macaca fascicularis</i>	8/group (male)	0, 100, or 1000 ppm (0, 410, or 4100 mg/m ³); 10 months, 6 h/d, 5d/wk; commercial grade, impurities not stated	Decreased motor conduction velocity at 9 months (right sciatic-tibial nerve, right ulnar nerve)	Not identified	410	Johnson et al. (1977)
Rat, Sprague-Dawley	18/group (male)	0, 100, or 330 ppm (0, 410, or 1353 mg/m ³); 72 weeks, 6h/d, 5d/wk; purity not stated	Severe polyradiculoneuritis in the lumbar and sacral spinal nerves and roots and the sciatic and tibial nerves in one rat	410	1353	Krasavage and O'Donoghue (1977)
Cat, domestic shorthair	4/group (female)	0, 100, or 330 ppm (0, 410, or 1353 mg/m ³); 2 years (6h/d, 5d/wk); purity not stated	Giant axonal neuropathy of the spinal cord and peripheral nerve in 4/4	410	1353	O'Donoghue and Krasavage (1979)

^aNo-observed-adverse-effect levels (NOAELs) and lowest-observed-adverse-effect levels (LOAELs) determined by 2-hexanone assessment authors.

4.6.3. Mode-of-Action Information

Exposure to 2-hexanone in humans and experimental animals demonstrates that the nervous system is the target organ of toxicity, regardless of the route of exposure. The toxicity is attributed to the neurotoxic metabolite 2,5-hexanedione. A strong relationship has been noted between the concentration of 2,5-hexanedione in the urine and the onset of neuropathic symptoms (Eben et al., 1979). Similarly, 2,5-hexanedione has been described as eliciting severe neurotoxic symptoms following oral, dermal, or i.p. administration to hens and oral administration to rats (Abou-Donia et al., 1985a; Abdo et al., 1982; Krasavage et al., 1980).

Current research supports a mode of action for γ -diketones, such as the 2-hexanone metabolite 2,5-hexanedione, which involves the covalent cross-linking of neuronal macromolecules with proteins as the primary target. The result is axonal swelling, specifically of giant axons, that ultimately ends in retrograde degeneration of the axon. 2,5-Hexanedione is an electrophilic species that reacts with nucleophilic sites of proteins via a substitution or addition reaction, with the subsequent formation of a covalent bond (Lopachin and Decaprio, 2005). Although 2,5-hexanedione has been shown to react with sulfhydryl groups of enzymes (Section 4.5.1.2), the compound causes distal axonopathy by covalent reaction with nucleophilic lysine ϵ -amine groups to form 2,5-dimethylpyrrole adducts with neurofilaments and other proteins (LoPachin et al., 2005, 2004). Oxidation of the pyrrole moiety with molecular oxygen can generate a cation intermediate that can undergo further reactions with amino- or sulfhydryl groups. This results in the development of neurofilament aggregates in the distal, subterminal axon that, as they grow larger, form massive swellings, often just proximal to the nodes of Ranvier (Graham, 1999).

One of the major hypotheses related to the mechanism of neurotoxicity of 2,5-hexanedione is covalent binding with axonal components of nerve tissue. In vitro studies in which 2,5-hexanedione was incubated with proteins demonstrated that this compound binds to the lysine ϵ -amino group, resulting in the formation of the substituted pyrrole adduct ϵ -N-(2,5-dimethylpyrrole)norleucine (DeCaprio et al., 1982). Covalent binding of 2,5-hexanedione with axonal components leading to pyrrole formation and protein cross-linking was hypothesized as a possible initiation step leading to axonal degeneration and thus may account for the neurotoxic effects observed with exposure to γ -diketones in general (DeCaprio et al., 1988; DeCaprio et al., 1982). In vivo pyrrole formation was confirmed by the demonstration of ϵ -N-(2,5-dimethylpyrrole)norleucine in the hydrolyzed serum of a hen that had received 2,5-hexanedione at 200 mg/kg-day for two weeks (DeCaprio et al., 1982). The proposed mechanism for 2,5-hexanedione in the development of progressive sensorimotor distal axonopathy is presented in Figure 4-1.

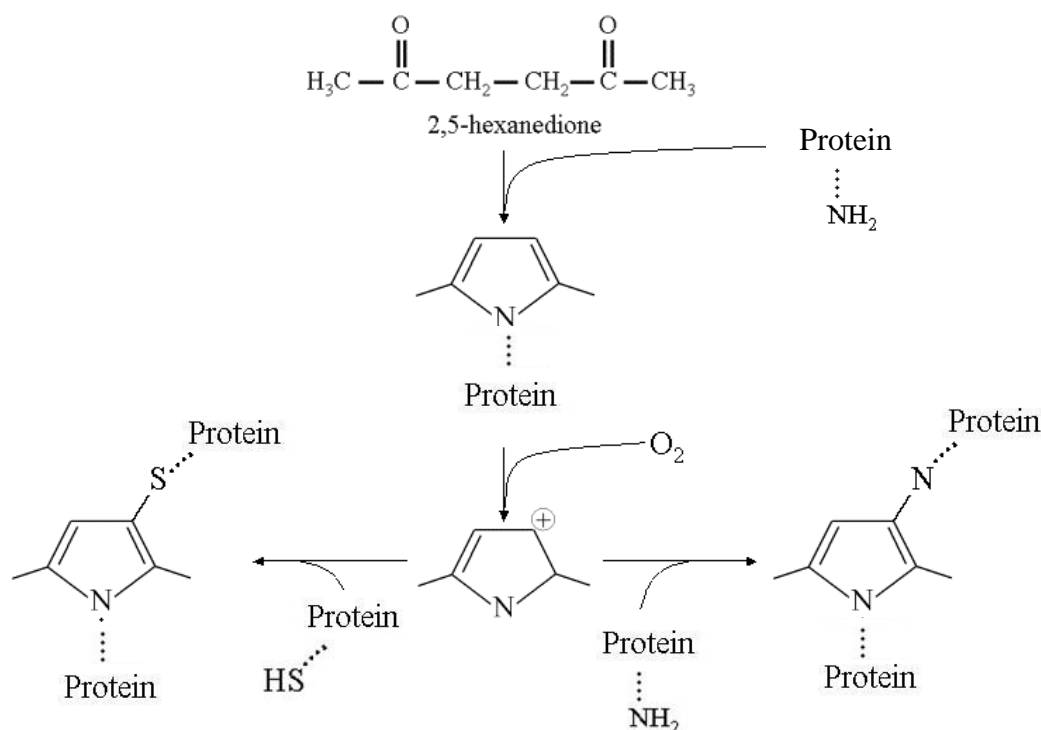


Figure 4-1. Proposed mechanism for 2,5-hexanedione-induced axonopathy.

Note: γ -Diketones, such as 2,5-hexanedione, react with amino groups in all tissues to form pyrroles. The pyrrole moiety can undergo further oxidation reactions with amino- or sulfhydryl groups. This results in the development of neurofilament aggregates (in the distal, subterminal axon), which, as they grow larger, form massive swellings of the axon.

Source: Adapted from DeCaprio et al. (1988, 1982).

4.7. WEIGHT-OF-EVIDENCE EVALUATION AND CANCER CHARACTERIZATION

4.7.1. Summary of Overall Weight of Evidence

Under EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), there is "inadequate information to assess the carcinogenic potential" of 2-hexanone. Specifically, there are no animal carcinogenicity studies available that examine exposure to 2-hexanone, and there are no studies available that assert a genotoxic potential of 2-hexanone. The available occupational studies do not present evidence for any carcinogenic action of 2-hexanone and are limited by frequent coexposure to other chemicals (e.g., MEK).

4.8. SUSCEPTIBLE POPULATIONS AND LIFE STAGES

4.8.1. Possible Childhood Susceptibility

The susceptibility of the developing brain is based on the timing of neuronal development, the rapid growth that occurs in the third trimester and early infancy, and the lack of a protective barrier early in life (Costa et al., 2004). In the cerebellum, Purkinje cells develop early, weeks 5–7 in humans, whereas granule cells are generated much later, gestational weeks

24–40 in humans. The developing brain is distinguished by the absence of a blood-brain barrier. The development of this barrier is a gradual process, beginning in utero and complete at approximately 6 months of age. Because the blood-brain barrier limits the passage of substances from blood to brain, in its absence, toxic agents can freely enter the developing brain. Since Purkinje-cell degeneration has been observed with adult rats exposed to high levels of 2,5-hexanedione, infants may be at an increased risk for this type of damage at lower levels of exposures, due to the incomplete maturation of the blood-brain barrier (Hernandez-Viadel et al., 2002). However, this would depend on the capacity of infants and small children to bioactivate 2-hexanone to 2,5-hexanedione.

Metabolism of 2-hexanone may vary between children and adults due to differences in the development and maturity of phase I and phase II enzymes (Johnsrud et al., 2003). Studies indicate that the mode of action of 2-hexanone toxicity involves the metabolism to a more toxic metabolites, namely 2,5-hexanedione. Several enzymes, such as CYP2E1, CYP2B1/2, and CYP2C6/11, are inducible following administration of 2-hexanone in animal models (Imaoka and Funae 1991; Nakajima et al., 1991); however, the individual isoforms involved in its metabolism have not been fully elucidated. Toftgard et al. (1986) found that the formation of 2,5-hexanediol from 2-hexanol was catalyzed by a CYP isozyme different from CYP2B and present in liver but not in lung microsomes. The authors concluded that 2-hexanol must be transported to the liver before the neurotoxic metabolite 2,5-hexanedione can be formed (Toftgard et al., 1986). Because of this, changes in CYP protein levels and phase II enzymes during development may likely have an impact on susceptibility to 2-hexanone. As mentioned above, the possible susceptibility of 2-hexanone may be influenced by life stage, but there are few studies to confirm the impact and severity of such exposure. Thus, the evidence of possible childhood susceptibility is inconclusive.

4.8.2. Possible Gender Differences

Evaluations of human occupational exposures have not provided evidence that 2-hexanone acts in a gender-specific way. Most animal studies also have not brought forth strong evidence of a sex-specific action of 2-hexanone. However, it should be mentioned that in a few rat studies 2-hexanone appeared to affect the male reproductive system (Katz et al., 1980; Krasavage et al., 1980; O'Donoghue et al., 1978).

5. DOSE-RESPONSE ASSESSMENTS

5.1. ORAL REFERENCE DOSE (RfD)

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a no-observed-adverse-effect level (NOAEL), lowest-observed-adverse-effect level (LOAEL), or benchmark dose (BMD), with uncertainty factors generally applied to reflect limitations of the data used.

5.1.1. Choice of Principal Study and Critical Effect—with Rationale and Justification

The 13-month drinking water study (10 animals/dose/sex) conducted by O'Donoghue et al. (1978) is the most suitable study for deriving a 2-hexanone RfD assessment. Five other subchronic studies are available and are considered as supporting studies. Of these five studies, Krasavage et al. (1980) and Eben et al. (1979) both observed neurotoxicity after administration of single doses of 2-hexanone via gavage. These two studies were not considered as principal studies because only single relatively high doses were administered and gavage administration is less relevant to human exposure than administration in drinking water. Abdo et al. (1982) observed mild ataxia, which progressed to severe ataxia, in hens gavaged daily with 100 mg/kg 2-hexanone. This study was not chosen as the principal study because the hen's digestive system is anatomically distinct from humans and thus a poor model for assessing the effects of human oral exposure.³ Finally, two subchronic drinking water studies that utilized multiple doses of 2-hexanone and identified neurotoxicological outcomes were considered. The first study, conducted by Homan et al. (1977), utilized doses that were higher than those used by O'Donoghue et al. (1978), and the purity of 2-hexanone was not stated. The second study, by Abdel-Rahman et al. (1978), utilized lower doses than the chronic study by O'Donoghue et al. (1978); however, the authors did not include complete data sets; that is, only data from the first 4 weeks of the study were presented. Further, the purity of the compound used was not stated.

O'Donoghue et al. (1978) conducted a 13-month study in male COBS/CD(SD) rats. The animals' drinking water contained 0, 0.25, 0.5, or 1.0% (0, 143, 266, or 560 mg/kg-day) 2-hexanone (96% pure, containing 3.2% MiBK and 0.7% unknown contaminants). In this study, 2-hexanone produced a dose-dependent reduction in body weight at all doses tested. The critical endpoints evaluated from this study were the incidences of myofibrillar atrophy of the quadriceps muscle and the calf muscle in male rats. These endpoints were chosen over the other neuropathologic endpoints in Table 5-1 because they occur due to axonal atrophy, an endpoint

³ The lower portion of a hen's esophagus forms a pouch called the crop, which serves as a temporary storage site for food prior to passage to the stomach. The two-part structure of the hen's stomach, which consists of the proventriculus and the gizzard, further alters the absorption and distribution of chemicals.

identified as the best correlate of nerve dysfunction, regardless of route of exposure (Lehning et al., 2000). Though axonal swelling was observed with high incidence in the peripheral nerve and spinal cord at the lowest dose tested, axonal swelling poorly correlates with nerve dysfunction and can occur without progression to nerve dysfunction (LoPachin et al., 2004, 2003; Lehning et al., 2000, 1995). Because myofibrillar atrophy of the quadriceps and calf muscles displayed a clear dose-dependent response, these data were evaluated further by BMD modeling.

5.1.2. Method of Analysis: Benchmark Dose Modeling

The animal data evaluated for derivation of an RfD for 2-hexanone are displayed in Table 5-1. These data are from a chronic toxicity study in rats in which 10 animals per dose group were administered 2-hexanone in drinking water at four different concentrations (i.e., 0, 0.25, 0.5, and 1.0%) for 13 months (O'Donoghue et al., 1978). The critical endpoints evaluated from this study were the incidences of myofibrillar atrophy of the quadriceps muscle and the calf muscle in male rats, which displayed a clear dose-dependent response.

Table 5-1. Summary of neuropathologic findings in male rats administered 2-hexanone in drinking water for 13 months

Treatment (dose)	Incidence of axonal swelling				Incidence of myofibrillar atrophy	
	Brain	Spinal cord	Dorsal root ganglia	Peripheral nerve	Quadriceps muscle ^a	Calf muscle ^a
Control	0/10	0/5	0/5	0/10	0/10	0/10
0.25% 2-hexanone (143 mg/kg-day)	2/10	7/10	0/7	8/10	1/10	2/10
0.5% 2-hexanone (266 mg/kg-day)	4/10	5/5	0/5	10/10	5/10	6/10
1.0% 2-hexanone (560 mg/kg-day)	8/10	5/5	3/5	10/10	10/10	10/10

^aThe data in bold were further evaluated for RfD derivation.

Source: O'Donoghue et al. (1978).

The BMD software (BMDS, version 1.3.2) (U.S. EPA, 1999) was used to estimate a point of departure (POD) for deriving an RfD for 2-hexanone from data on myofibrillar atrophy of the quadriceps and calf muscles. This POD, called the BMDL, is defined as the 95 percent lower bound on the benchmark dose (BMD) associated with the benchmark response (BMR). For this study, a BMR of 10% extra risk (ER) was selected because it represents a response at the lower end of the observable range of the data, and provides a consistent basis of comparison across assessments. Table 5-2 presents the “best-fit” model results based on data on the incidence of myofibrillar atrophy of the quadriceps and calf muscles in rats exposed to 2-hexanone in drinking water. A more detailed presentation of the BMD modeling results is

contained in Appendix B-1. In the absence of any compelling biological reason to choose one of these endpoints over the other for RfD derivation, myofibrillar atrophy of the quadriceps muscle was used because this endpoint yielded a slightly lower BMDL than myofibrillar atrophy of the calf muscle.

Table 5.2. Best fit BMD modeling results for data on myofibrillar atrophy of the quadriceps muscle and calf muscle

Endpoint (myofibrillar atrophy)	Model	AIC ^a	<i>p</i> Value	BMD (mg/kg-day)	BMDL (mg/kg-day)	BMD/BMDL
Quadriceps muscle	Multistage	22.3952	0.9995	141.4	49.9	2.8
Calf muscle	Quantal quadratic	25.8664	0.9701	88.7	69.2	1.3

^aAIC = Akaike Information Criterion.

5.1.3. Derivation of Human Equivalent Doses

For 2-hexanone, no PBTK model is currently available. Therefore, the first step required for the final chronic RfD derivation is to determine whether intermittent doses were employed in the animal study and, if so, to adjust these doses to reflect continuous exposures, based on the assumption that the product of dose and time is constant (U.S. EPA, 2002). In the principal study (O'Donoghue et al., 1978), animals were administered 2-hexanone in drinking water 24 hours/day, 7 days/week for 13 months. Therefore, in this case, a duration adjustment is not required (i.e., the POD [adjusted BMDL or BMDL_{ADJ}] for 2-hexanone equals the study BMDL) as follows:

$$\text{BMDL}_{\text{ADJ}} = \text{BMDL} \times (\# \text{ of hours per day exposed} / 24 \text{ hours}) \times (\# \text{ of days per week exposed} / 7 \text{ days})$$

$$\text{BMDL}_{\text{ADJ}} = 50 \text{ mg/kg-day} \times (24 \text{ hours} / 24 \text{ hours}) \times (7 \text{ days} / 7 \text{ days}) = 50 \text{ mg/kg-day}$$

EPA currently does not provide a specific procedure for calculating a human equivalent dose for oral (or dermal) exposure scenarios that parallel calculation of the inhalation human equivalent concentration (HEC). Hence, the BMDL_{ADJ} is used as the point of departure from which to apply uncertainty factors.

5.1.4. Calculation of the RfD—Application of Uncertainty Factors

The RfD for myofibrillar atrophy of the quadriceps muscle as the critical effect is calculated from the BMDL_{ADJ} by application of uncertainty factors (UFs) as follows:

$$\text{RfD} = \text{BMDL}_{\text{ADJ}} \div \text{UF}$$

$$\text{RfD} = 50 \text{ mg/kg-day} \div 300 = 0.166 \text{ mg/kg-day} = 2 \times 10^{-1} \text{ mg/kg-day}$$

The composite UF of 300 was derived as follows:

- An intraspecies uncertainty factor (UF_H) of 10 was applied to adjust for potentially sensitive human subpopulations. A default value is warranted because insufficient information is currently available to assess human-to-human variability in 2-hexanone toxicokinetics or toxicodynamics.
- A default interspecies uncertainty factor (UF_A) of 10 was applied for extrapolation from animals to humans. No suitable data on the toxicity of 2-hexanone to humans exposed by the oral route only were identified. Insufficient information is currently available to assess rat-to-human differences in 2-hexanone toxicokinetics or toxicodynamics.
- A UF of 3 was applied to account for database deficiencies (UF_D). The database includes subchronic animal studies in rats and hens and a chronic study in rats but does not include multigenerational reproductive and developmental studies. Though no 2-hexanone-specific developmental studies are available, supporting developmental studies with n-hexane, a precursor of 2-hexanone, and 2,5-hexanedione, the ultimate toxic metabolite of n-hexane and 2-hexanone, have been primarily negative. Mouse reproductive/developmental and teratological studies with n-hexane have been negative with doses administered on GDs 6–15 by gavage of up to 2200 mg/kg-day or by daily injection up to 9900 mg/kg-day (Marks et al., 1980), and rat developmental neurotoxicity studies with 2,5-hexanedione have found minimal effects (e.g., aggregated and fused axons, identified with electron microscopy) from daily s.c. injections on GDs 12–20 with 340 mg/kg 2,5-hexanedione (Ogawa et al., 1993). It should be noted that the available studies with 2-hexanone following inhalation exposure suggest the possibility of immunotoxicity and reproductive toxicity as areas of potential concern with human exposure. For example, a reduction in total white blood cell count to ~60% of control values was reported in rats intermittently exposed to 700 ppm 2-hexanone via inhalation for 8 weeks (Katz et al., 1980). In addition, behavioral alterations observed in offspring of pregnant rats exposed to 1000 ppm 2-hexanone (Peters et al., 1981) and atrophy of testicular germinal epithelium observed in male rats exposed to 700 ppm 2-hexanone (Katz et al., 1980) suggest there may be cause for concern. However, there are no studies that evaluate immunotoxicity following oral exposure to 2-hexanone. Because of the absence of studies evaluating the possible immunotoxicity or reproductive toxicity of 2-hexanone following exposure via the oral route, a UF_D of 3 is warranted.
- An uncertainty factor for LOAEL-to-NOAEL extrapolation was not used because the current approach is to address this factor as one of the considerations in selecting a BMR for benchmark dose modeling. In this case, a BMR of a 10% extra risk of myofibrillar atrophy of the quadriceps muscle was selected under an assumption that it represents a minimal biologically significant change.

- A subchronic-to-chronic UF (UF_S) was not applied because the principal study involved a chronic exposure.

5.1.5. RfD Comparison Information

Figure 5-1 presents PODs, applied UFs, and derived RfD for the endpoints considered for 2-hexanone. As stated previously, of the available chronic and subchronic studies, the 13-month drinking water study conducted by O'Donoghue et al. (1978) was considered the most suitable study to derive an RfD. Within this study, two potential endpoints, myofibrillar atrophy of either the quadriceps muscle or the calf muscle were considered. The points of departure based on the best fit models from BMD models from Table 5.2 are presented in Figure 5-1. Axonal swelling in the brain, spinal cord and dorsal root ganglia were endpoints noted in O'Donoghue et al. (1978). These endpoints are also illustrated in Figure 5-1, but as previously mentioned, axonal swelling poorly correlates with nerve dysfunction and can occur without progression to nerve dysfunction and thus is deemed less relevant endpoints. The supporting studies outlined in Table 4-15 were deemed less relevant to human exposure as they either involved single relatively high doses via gavage in rodents (Krasavage et al., 1980; Eben et al., 1979), used a test species such as hens (which might be a poor model for assessing human exposure) (Abdo et al., 1982), were subchronic in design with higher doses administered than the chronic study by O'Donoghue et al. (1978) (Homan et al, 1977), or did not include complete data (Abdel-Rahman et al., 1978).

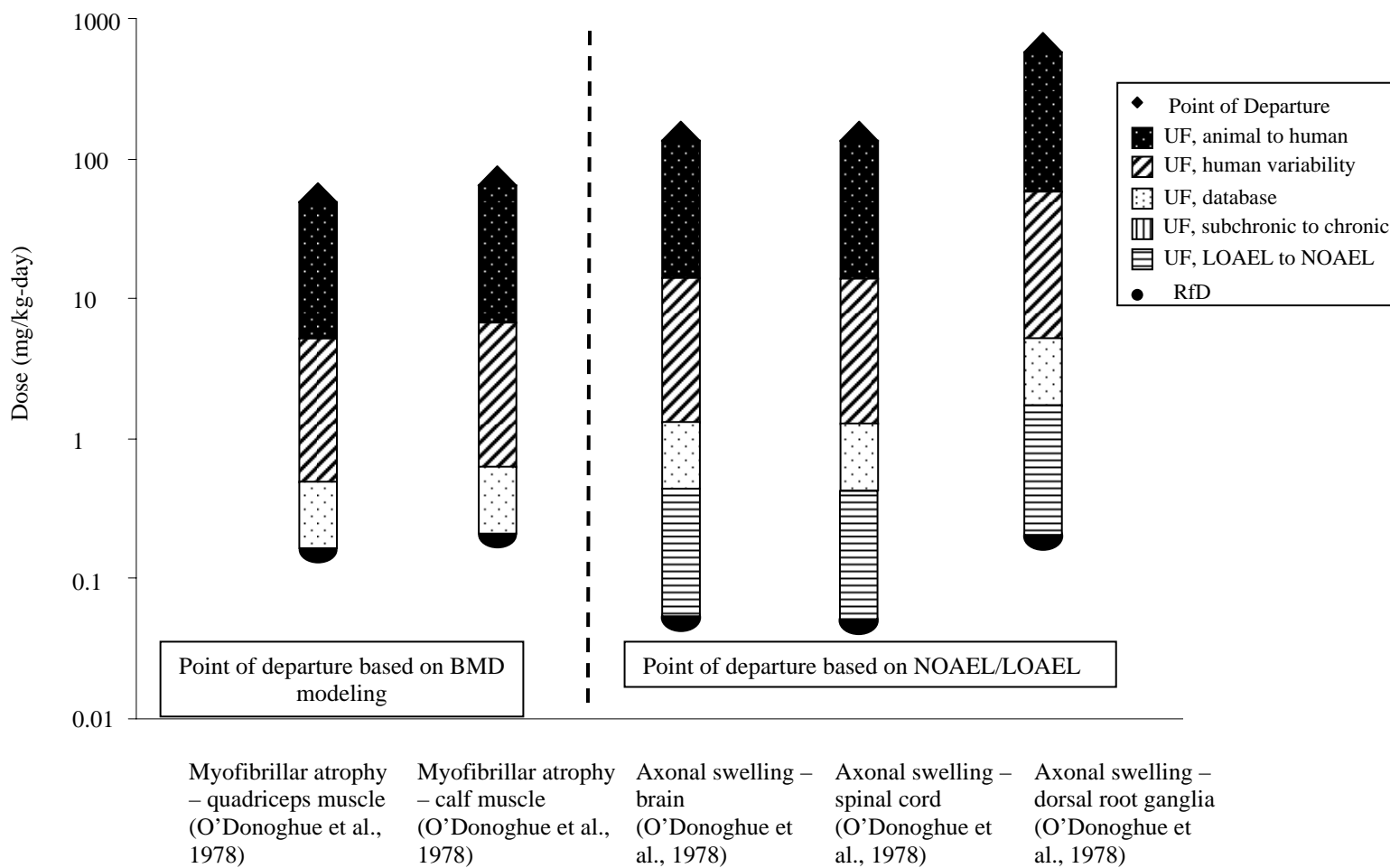


Figure 5-1. Points of departure for endpoints from O'Donoghue et al. (1978) with corresponding applied uncertainty factors and derived RfD.

5.1.6. Previous Oral Assessment

There was no previous RfD assessment for 2-hexanone with which to compare and contrast the RfD developed in this assessment.

5.2. INHALATION REFERENCE CONCENTRATION

The inhalation RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human general population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects over a lifetime. It can be derived from a NOAEL, a LOAEL, or a benchmark concentration (BMC), with UFs generally applied to reflect uncertainties and/or limitations in the data used.

5.2.1. Choice of Principal Study and Critical Effect, with Rationale and Justification

An inhalation study that exposed monkeys and rats to 0, 100, or 1000 ppm (0, 410, or 4100 mg/m³) commercial grade 2-hexanone for 6 hours per day, 5 days per week for up to 10 months was used as the principal study in the derivation of the RfC (Johnson et al., 1977).

As discussed in Chapter 4, human and animal data indicate that neurological effects are a characteristic and sensitive endpoint of inhalation exposure to 2-hexanone. Neuropathy has been observed in humans following inadvertent occupational exposure (Allen et al., 1975; Billmaier et al., 1974; Gilchrist et al., 1974) and has been demonstrated repeatedly in laboratory animals (Katz et al., 1980; Egan et al., 1980; O'Donoghue and Krasavage, 1979; Johnson et al., 1979, 1977; Duckett et al., 1979, 1974; O'Donoghue et al., 1978; Krasavage and O'Donoghue, 1977; Spencer et al., 1975; Mendell et al., 1974).

Several studies of workers in a coated fabrics plant (Allen et al., 1975; Billmaier et al., 1974; Gilchrist et al., 1974) provide evidence in humans of a concentration-dependent neurotoxic response to 2-hexanone exposure. Although personal air samples were not collected in these studies, the available measures of exposure were sufficient to produce quantitative estimates of 2-hexanone inhalation exposure for two groups of workers (i.e., print operators and print helpers), both of whom exhibited peripheral neuropathy. In these workers, exposure to 2-hexanone also occurred via oral and dermal routes, as the study authors noted that individuals frequently ate at the work site and were accustomed to washing their hands with 2-hexanone. Because the magnitude of exposure to 2-hexanone from these two other exposure routes (i.e., oral and dermal), which could have been considerable, was not quantified by the study authors and the workers were coexposed with MEK, which can potentiate the toxicity of 2-hexanone, this study was deemed unsuitable for use in RfC derivation.

Of the available animal studies on 2-hexanone, the subchronic studies by Abdo et al. (1982), Duckett et al. (1979, 1974), Krasavage and O'Donoghue (1977), Saida et al. (1976), Mendell et al. (1974) and the chronic study by O'Donoghue and Krasavage (1979) were not selected for use in deriving the RfC. Duckett et al. (1979, 1974) did not report the sex of the

animals or the purity of 2-hexanone used. Further, the authors used only one exposure concentration per series of experiments. Krasavage and O'Donoghue (1977) utilized two exposure concentrations (e.g., 100 and 330 ppm); however, the purity of 2-hexanone was not stated and limited data were provided. As mentioned previously, MiBK, a potential inducer of CYP450, is a common contaminant in the formulations of 2-hexanone. Without the more information on the purity of the 2-hexanone administered, it is difficult to ascertain if MiBK impacted the toxicity of 2-hexanone in Krasavage and O'Donoghue (1977). Saida et al. (1976) used two exposure concentrations (e.g., 225 ppm and 400 ppm) but did not state the sex of the animals or the purity of 2-hexanone used. Finally, Mendell et al. (1974) and Abdo et al. (1982) reported findings using hens. Although the exposure-concentration regimen used by Abdo et al. (1982) included five exposure concentrations (i.e., 10, 50, 100, 200, and 400 ppm), hens are not a suitable model for extrapolating experimental results to humans.⁴ The remaining studies by Johnson et al. (1977), Katz et al. (1980), and Egan et al. (1980) were all considered further as possible principal studies.

The study by Johnson et al. (1977) was performed in two different animal species, monkeys and rats, with 8 and 10 animals per dose group, respectively. Two concentrations of commercial grade 2-hexanone were employed (100 and 1000 ppm in air) with exposures occurring 6 hours per day, 5 days per week for a duration of 10 months. Concurrent control groups were used in both species. As part of this study, Johnson et al. (1977) conducted four neurological tests in each species (usually once per month) to identify adverse effects in treated versus control animals. These four tests were MCV of the right sciatic-tibial nerve, MCV of the right ulnar nerve, absolute refractory period of these two nerves, and muscle action potentials in response to both sciatic and ulnar nerve stimulation.

The animal studies by Katz et al. (1980) and Egan et al. (1980) consisted of exposure to 2-hexanone (purity > 96%) at a single concentration for a period of 6 months or less, using only one strain and sex of rats. Both Katz et al. (1980) and Egan et al. (1980) utilized clinical chemistry and histopathologic changes to identify treatment-related effects of 2-hexanone. Despite the use of commercial grade 2-hexanone, the study by Johnson et al. (1977) was chosen as the most suitable study on which to base the RfC because Johnson et al. (1977) used two different animal species, including nonhuman primates, and two 2-hexanone exposure concentrations, while also employing larger treatment groups and longer exposure durations than either Katz et al. (1980) or Egan et al. (1980). Although duration of the study by Krasavage and O'Donoghue (1979) was longer than the study by Johnson et al. (1977), the latter utilized monkeys, a more biologically relevant species than rats, when assessing inhalation exposure. Also, Krasavage and O'Donoghue (1979) provide limited information to serve as the basis for a reference concentration.

⁴ Birds have an intricate respiratory system that is exclusive to birds and includes a system of air sacs that surround the internal organs and provide reserve air space to increase lung capacity.

As previously discussed, the toxic effects seen in humans and experimental animals following exposure to 2-hexanone via inhalation provide evidence that the nervous system is the primary target of 2-hexanone toxicity. Data from Johnson et al. (1977) on both sciatic-tibial and ulnar nerve MCVs in 2-hexanone-exposed monkeys and rats were considered for use in deriving the RfC, but, ultimately, the monkey sciatic-tibial nerve MCV data were selected for the following reasons. Both monkeys and rats exhibited significant decrements in sciatic-tibial nerve MCVs at the lowest administered concentration of 2-hexanone, beginning at 9 and 7 months on study, respectively. A neuropathy similar to that observed for the sciatic-tibial nerves occurred in the ulnar nerves of both monkeys and rats. Although monkeys in the low exposure group exhibited statistically significant decreases in ulnar nerve MCVs relative to control values at 1 and 3 months, beginning at 6 months on study, this decline was not statistically significant. As monkeys have a similar respiratory tract and breathing patterns to humans, and the 2,5-hexanedione, the metabolite of 2-hexanone, typically affects long axons such as the sciatic-tibial nerve prior to other nerves, the sciatic-tibial nerve motor conduction velocity in monkeys is used to derive the RfC, though both sciatic-tibial MCV and ulnar MCV for both monkeys and rats were modeled.

5.2.2. Methods of Analysis: Benchmark Concentration Modeling

Table 5-3 displays monthly mean MCV values (in m/second) for both the sciatic-tibial and ulnar nerves of monkeys exposed to three different concentrations of 2-hexanone in air (i.e., 0, 100, or 1000 ppm) for durations ranging from 1 to 10 months. These data were extracted (via digitization⁵) from Figure 1 (for the sciatic-tibial nerve) and Figure 3 (for the ulnar nerve) of Johnson et al. (1977). Similarly, Table 5-4 displays monthly mean MCV values (in m/second) for both the sciatic-tibial and ulnar nerves of rats exposed to three different concentrations of 2-hexanone in air (i.e., 0, 100, or 1,000 ppm) for durations ranging from 2 to 29 weeks. These data were extracted (via digitization) from Figure 2 (for the sciatic-tibial nerve) and Figure 4 (for the ulnar nerve) of Johnson et al. (1977).

Because MCV values are continuous (as opposed to dichotomous), the data in Tables 5-3 and 5-4 were subjected to BMD modeling employing the available continuous models in EPA's

⁵ Values from Johnson et al. (1977) were digitized using the line tool on Microsoft Office Word 2003, followed by measuring the values with the distance tool function on Adobe® Acrobat® 6.0 Professional (version 6.0.0, 5/19/2003). To accomplish this task, the figures from Johnson et al. (1977) were inserted into a Word document using the snapshot tool from Adobe® Acrobat® 6.0 Professional. Then, horizontal lines were applied over the data points, the measurement markers on the y-axis, and extended through the y-axis. Lines from the data points to the x-coordinates were not traced over, since Johnson et al. (1977) provided the absolute values in the text. Once all of the lines were traced from the data points through the y-coordinates, a vertical line was traced over the y-axis. Then the Word document was saved in portable document format (pdf) and opened using Adobe® Acrobat® 6.0 Professional. The y-axis was viewed at 300% magnification, and the distance tool was used to measure from the origin to each y-coordinate for each horizontal line, including data points and measurement markers. The distance tool allows measurements to be made down to one hundredth of a millimeter, and repeated measures placed the reproducibility of this technique at greater than 99%.

BMDS, version 1.3.2 (i.e., linear, polynomial, power, and Hill models). The BMR was defined as a 10% relative change in nerve conduction velocity from the control mean. Changes in nerve conduction velocity are thought to represent a clinically significant effect.

A difficulty encountered in conducting a BMD analysis on these data was that no information was provided regarding the standard errors or confidence limits for the mean nerve conduction velocities shown in Figures 1 through 4 in Johnson et al. (1977) nor was any of the raw data on which these means were based presented in the paper. Attempts to obtain the raw data from the investigators were unsuccessful. In BMDS, estimates of the standard deviation of the response in each dose group are needed to calculate BMDs and their corresponding BMDLs. Therefore, an indirect method for estimating this missing information on response variability was devised.

Information regarding the variability in MCV measurements in Johnson et al. (1977) can be derived from the results of statistical tests that are reported in the paper. In this study, two different statistical procedures were employed. An ANOVA was used to test for statistically significant differences in mean MCVs at specific test periods (usually monthly) whenever data across the three exposure groups (i.e., 0, 100, or 1000 ppm) were compared. After approximately 6 months on study, however, animals (both monkeys and rats) in the highest exposure group (1000 ppm) were removed from further 2-hexanone exposure. Consequently, with termination of this 1000 ppm exposure group, only two dose groups remained for each species. Thus, subsequently, the Student's *t*-test was used to test for statistically significant changes in mean MCVs across these two groups (i.e., 0 and 100 ppm) for the remaining test periods.

In an ANOVA, an *F* statistic is used to test for a significant difference among the means of *g* groups. An *F* statistic is defined as, $F(g-1, N-g) = \text{between-group variance} / \text{within-group variance}$, where *g*-1 represents the numerator degrees of freedom and *N*-*g* represents the denominator degrees of freedom (*g* is the number of groups and *N* is the sample size within each group). In the specific case where only two group means are being compared, the *F* statistic reduces to a *t* statistic (i.e., $F(1, N-g) = t(N-g)^2$), where *t* has a Student's *t*-distribution. In order to fit a continuous dose-response model in BMDS, an estimate of the within-group variance or s^2 is needed from which the estimated standard deviation can be obtained simply by taking the square root of this variance estimate.

The estimated within-group variance can be derived using the following procedure. If the within-group means and the numbers of observations on which each of these means are based are known, the between-group variance can be calculated. Once the between-group variance has been determined and the corresponding value of the *F* or *t* statistic is known, an estimate of the within-group variance or s^2 can be derived from the following equation: $s^2 = (\text{between-group variance}) / F(g - 1, N - g)$ or $s^2 = (\text{between-group variance}) / t(N-g)^2$. In Johnson et al. (1977), for monkeys, *F* statistics were reported for mean MCVs at both 4 and 6 months, while *t* statistics

were reported for mean MCVs at both 9 and 10 months. These data yielded four estimates of the within-group variance or standard deviation. The arithmetic average of these four estimates was then used in BMD modeling as the estimated standard deviation for MCVs in each dose group, assuming a constant variance across dose groups. For rats, *F* statistics were reported in Johnson et al. (1977) for mean MCVs at both 13 and 17 weeks, while a *t* statistic was reported for mean MCVs at 29 weeks. These data yielded three estimates of the within-group variance or standard deviation. The arithmetic average of these three estimates was then used in BMD modeling as the estimated standard deviation for MCVs in each dose group, assuming a constant variance across dose groups.

Table 5-3. Effect of 2-hexanone inhalation exposure on the MCV of the sciatic-tibial and ulnar nerves in monkeys

Exposure duration (months)	2-Hexanone concentration (ppm in air)	Mean MCV: sciatic-tibial nerve (m/s) ^a	Mean MCV: ulnar nerve (m/s) ^b
1	0	42	54
	100	42	46^c
	1000	40	47^c
2	0	51	61
	100	46	63
	1000	44	49^c
3	0	54	53
	100	48	47^c
	1000	46	45^c
4	0	56	63
	100	50	58
	1000	41^c	49^c
5	0	53	61
	100	48	63
	1000	36^c	43^c
6	0	50	58
	100	47	56
	1000	33^c	41^c
7	0	51	65
	100	48	62
8	0	50	58
	100	46	58
9	0	53	63
	100	49^c	60
10	0	53	58
	100	48^c	57

^aValues extracted from Figure 1 in Johnson et al. (1977).

^bValues extracted from Figure 3 in Johnson et al. (1977).

^cStatistically significantly different compared with corresponding controls ($p < 0.05$), as determined by the study authors.

Table 5-4. Effect of 2-hexanone inhalation exposure on the MCV of the sciatic-tibial and ulnar nerves in rats

Exposure duration (weeks)	2-Hexanone concentration (ppm in air)	Mean MCV: sciatic-tibial nerve (m/s) ^a	Mean MCV: ulnar nerve (m/s) ^b
13	0	34	
	100	37	
	1000	40 ^c	
17	0		42
	100		36 ^c
	1000		38 ^c
25	0	42	40
	100	41	37
	1000	27 ^c	31 ^c
29	0	39	45
	100	25 ^c	30 ^c

^aValues extracted from Figure 2 in Johnson et al. (1977).

^bValues extracted from Figure 4 in Johnson et al. (1977).

^cStatistically significantly different compared with corresponding controls ($p < 0.05$), as determined by the study authors.

The “best-fit” model from BMDS was selected by examining the results of the chi-squared goodness-of-fit test and comparing the magnitudes of the Akaike’s Information Criterion (AIC). All models with chi-squared p values ≥ 0.1 were considered to exhibit an adequate fit to the data. Of the models exhibiting adequate fit, the model with the lowest AIC was selected as the best-fit model. These criteria for model selection are consistent with those described in the *Benchmark Dose Technical Guidance Document* (U.S. EPA, 2000c). For the MCV data in both monkeys and rats, the 1st-degree polynomial model provided the best fit for both sciatic-tibial and ulnar nerve MCVs.

The 95% lower confidence limits on the benchmark concentration estimates (BMCLs) derived from the best-fit models for sciatic-tibial and ulnar nerve MCV values in monkeys and rats are presented in Table 5-4. Detailed BMDS outputs from the BMD of the monkey and rat MCV data are contained in Appendix B-1.

5.2.3. Exposure Duration Adjustments and Conversion to Human Equivalent Concentrations

Because the RfC is a metric that assumes continuous human exposure for a lifetime, adjustments need to be made to animal (or human) data obtained from intermittent and/or less-than-lifetime exposures, as outlined in the *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994). The first step in this process is adjusting intermittent inhalation exposures to continuous inhalation exposures, based on the assumption that the product of exposure concentration and time is constant (U.S. EPA, 2002). In Johnson et al (1977), animals were exposed to 2-hexanone for 6 hours/day, 5 days/week. Therefore, the BMCL_{ADJ}, reflecting continuous inhalation exposure to 2-hexanone,

is derived as follows:

$$\begin{aligned}\text{BMCL}_{\text{ADJ}} &= \text{BMCL} \times \text{hours exposed per day}/24 \text{ hours} \times \text{Days exposed per week}/7 \text{ days.} \\ \text{BMCL}_{\text{ADJ}} &= 243 \times 6/24 \times 5/7 = 43 \text{ ppm, based on monkey sciatic-tibial nerve MCV} \\ &= 278 \times 6/24 \times 5/7 = 50 \text{ ppm, based on monkey ulnar nerve MCV} \\ &= 232 \times 6/24 \times 5/7 = 41 \text{ ppm, based on rat sciatic-tibial nerve MCV} \\ &= 352 \times 6/24 \times 5/7 = 63 \text{ ppm, based on rat ulnar nerve MCV}\end{aligned}$$

Furthermore, because RfCs are typically expressed in units of mg/m^3 , the above ppm values need to be converted to mg/m^3 using the conversion factor specific to 2-hexanone of $1 \text{ ppm} = 4.1 \text{ mg}/\text{m}^3$. Thus, the final BMCL_{ADJ} values are as follows:

$$\begin{aligned}\text{BMCL}_{\text{ADJ}} &= 43 \times 4.1 = 176.3 \text{ mg}/\text{m}^3, \text{ monkey sciatic-tibial nerve MCV} \\ &= 50 \times 4.1 = 205 \text{ mg}/\text{m}^3, \text{ based on monkey ulnar nerve MCV} \\ &= 41 \times 4.1 = 168.1 \text{ mg}/\text{m}^3, \text{ based on rat sciatic-tibial nerve MCV} \\ &= 63 \times 4.1 = 258.3 \text{ mg}/\text{m}^3, \text{ based on rat ulnar nerve MCV}\end{aligned}$$

Finally, this BMCL_{ADJ} value must be converted to an HEC. The HEC that elicits decreased MCV, which is not a respiratory (or portal-of-entry) effect, but a systemic effect, is derived based on the following. For systemic effects, 2-hexanone is classified as a category 3 gas under EPA guidelines (U.S. EPA, 1994). According to this guidance, in order to convert the concentration effective in animals to human equivalents, a multiplicative factor based on the ratio of blood:gas partition coefficients is employed as follows:

$$\text{HEC} = \text{BMCL}_{\text{ADJ}} \times [(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}]$$

Where,

$(\text{H}_{\text{b/g}})_{\text{A}}$ = blood:gas partition coefficient for 2-hexanone in animals

$(\text{H}_{\text{b/g}})_{\text{H}}$ = blood:gas partition coefficient for 2-hexanone in humans.

The blood:gas partition coefficient $(\text{H}_{\text{b/g}})_{\text{H}}$ for 2-hexanone in humans is 127 (Sato and Nakajima, 1979); however, no value has been reported for monkeys or rats. In the absence of a measured blood:gas partition coefficient in the test species, the ratio $[(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}]$ defaults to unity, and the conversion to a HEC becomes the following:

$$\text{BMCL}_{\text{HEC}} = \text{BMCL}_{\text{ADJ}} \times [(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}] = 176.3 \times 1 = 176.3 \text{ mg}/\text{m}^3 \text{ based on monkey}$$

sciatic-tibial nerve MCV

= $205 \times 1 = 205 \text{ mg/m}^3$ based on monkey ulnar nerve MCV

= $168.1 \times 1 = 168.1 \text{ mg/m}^3$ based on rat sciatic-tibial nerve MCV

= $258.3 \times 1 = 258.3 \text{ mg/m}^3$ based on rat ulnar nerve MCV

These HEC values are presented in the last column of Table 5-5.

Table 5-5. Summary of BMCLs and HECs for 2-hexanone

Study reference	Study duration and type	2-Hexanone exposure (ppm)	Species/sex	Toxicological endpoint	BMDS “best fit” continuous model	BMC (ppm)	BMCL or POD ^a (ppm)	Adjusted BMCL (BMCL _{ADJ}) ^b	HEC ^c
Johnson et al. (1977)	10-month inhalation (subchronic)	0, 100, 1000	Male monkeys (n = 8 per dose group)	Sciatic-tibial nerve motor conduction velocity (at 6 months)	1 st degree polynomial	293	243	176	176
				Ulnar nerve motor conduction velocity (at 6 months)	1 st degree polynomial	335	278	205	205
Johnson et al. (1977)	29-week inhalation (subchronic)	0, 100, 1000	Male rats (n = 10 per dose group)	Sciatic-tibial nerve motor conduction velocity (at 25 weeks)	1 st degree polynomial	271	232	168	168
				Ulnar motor nerve conduction velocity (at 25 weeks)	1 st degree polynomial	471	352	258	258

^aBMCLs or PODs were estimated at a BMR of 0.1 or 10% relative change from controls.

^bConversion factors and assumptions: molecular weight (2-hexanone) = 100.16 and 1 ppm = 100.16/24.45 = 4.1 mg/m³ (at 25°C and 760 mm Hg). Duration adjustment of exposure concentrations and conversion to mg/m³ was accomplished as follows: BMCL_{ADJ} = 243 ppm × 6h/24h × 5 d/7d = 43 ppm × 4.1 = 176 mg/m³.

^cThe BMCL_{HEC} was calculated for an extraratory effect of a category 3 gas. The blood:gas partition coefficient (Hb/g) value for 2-hexanone in humans is 127 (Sato and Nakajima, 1979); however, no value has been reported for monkeys or rats. According to EPA’s RfC methodology (U.S. EPA, 1994), when the ratio of animal to human blood:gas partition coefficients [(Hb/g)_A/(Hb/g)_H] is greater than one or the values are unknown, a value of one is used for the ratio by default. Thus, BMCL_{HEC} = 176 × [(Hb/g)_A/(Hb/g)_H] = 176 mg/m³.

5.2.4. Calculation of the RfC: Application of Uncertainty Factors

As monkeys have a similar respiratory tract and breathing patterns to humans, and the 2,5-hexanedione, the metabolite of 2-hexanone, typically affects long axons such as the sciatic-tibial nerve prior to other nerves, the BMCL_{HEC} based on sciatic-tibial nerve motor conduction velocity in monkeys (Table 5-4) is used to derive the RfC. It should be noted that ulnar nerve motor conduction velocity in monkeys and sciatic-tibial nerve motor conduction velocity in rats were found to have similar BMCL_{HEC} as the endpoint selected above and would not result in significantly different RfCs if those alternatives were utilized.

The RfC for 2-hexanone based on peripheral neuropathy as the critical effect is derived from the BMCL_{HEC} by application of UFs as follows:

$$\text{RfC} = \text{BMCL}_{\text{HEC}} \div \text{UF}$$

$$\text{RfC} = 176 \div 1000 = 0.168 \text{ mg/m}^3 \approx 2 \times 10^{-1} \text{ mg/m}^3$$

This composite UF of 1000 is composed of the following:

- A default intraspecies uncertainty factor (UF_H) of 10 was applied to adjust for potentially sensitive human subpopulations (intraspecies variability).
- A default subchronic-to-chronic uncertainty factor (UF_S) of 10 was applied to account for the less-than-lifetime exposure (10 months) in the principal study.
- A factor of 3 was selected to account for uncertainties in extrapolating from rats to humans. This value is adopted by convention where an adjustment from an animal-specific NOAEL_{ADJ} to a NOAEL_{HEC} has been incorporated. Application of a full uncertainty factor of 10 would depend on two areas of uncertainty (i.e., toxicokinetic and toxicodynamic uncertainties). In this assessment, the toxicokinetic component is mostly addressed by the determination of a human equivalent concentration as described in the RfC methodology (U.S. EPA, 1994b). The toxicodynamic uncertainty is also accounted for to a certain degree by the use of the applied dosimetry method and an UF of 3 is retained to fully address this component. An uncertainty factor for LOAEL-to-NOAEL extrapolation was not used because the current approach is to address this factor as one of the considerations in selecting a BMR for benchmark dose modeling. In this case, a BMR of a 10% change in nerve conduction velocity from the control mean was selected under an assumption that it represents a minimal biologically significant change. An uncertainty factor of 3 was applied to account for database deficiencies (UF_D). The database includes a human occupational exposure study (with coexposure to MEK), subchronic animal studies in rats and hens, and a chronic study in cats. One postnatal development and behavior study on 2-hexanone (Peters et al., 1981) exists, identifying a LOAEL of 1000 ppm (no NOAEL

reported), but no other developmental or teratology studies on 2-hexanone exist. However, support for applying a UF_D of 3 in the absence of other 2-hexanone-specific studies is based on the availability of developmental and teratology studies with n-hexane, a precursor of 2-hexanone, and 2,5-hexanedione, a metabolite of n-hexane and 2-hexanone. The rationale for the UF_D of 3 is based on the following: (1) developmental studies with n-hexane concentrations of 100 (GDs 6–15), 400 (GDs 6–15), or 1000 ppm (GDs 8–16) have been negative (Bus et al., 1979; Litton Bionetics, 1979); (2) a teratology study conducted on behalf of the National Toxicology Program, in which dams were exposed on GDs 6–19 to 200, 1000, or 5000 ppm n-hexane, identified a NOAEL of 200 ppm (Mast, 1987), a concentration nearly double the highest NOAEL identified from inhalation studies with 2-hexanone (See Table 4-16); and (3) rat developmental neurotoxicity studies with 2,5-hexanedione, the ultimate toxic metabolite of n-hexane and 2-hexanone, have found minimal effects (e.g., aggregated and fused axons, identified with electron microscopy) from daily s.c. injections on GDs 12–20 with 340 mg/kg 2,5-hexanedione (Ogawa et al., 1993).

5.2.5. RfC Comparison Information

Figure 5-2 presents PODs, applied UFs, and derived RfCs for several studies and endpoints considered for 2-hexanone. Of the chronic and subchronic studies available on inhalation exposure to 2-hexanone, Johnson et al. (1977) was deemed the most suitable to derive an RfC. The endpoints considered from Johnson et al. (1977) include MCV for both sciatic-tibial and ulnar nerves of both rats and monkeys. The PODs based on the best fit models from BMD models from Table 5-4 are presented in Figure 5-2. Subchronic rodent studies by Katz et al. (1980) and Egan et al. (1980) were also considered; however, both studies evaluated exposure to a single concentration of 2-hexanone for a period of less than 6 months, using clinical chemistry or histopathologic changes to identify treatment-related effects. The unpublished study by Krasavage and O'Donoghue (1977) was longer in exposure duration than the study by Johnson et al. (1977) and utilized two exposure concentrations, though purity of 2-hexanone was not specified. The Johnson et al. (1977) study is preferred because the study involved nonhuman primates that are more relevant to assessing human exposure than obligatory nose-breathing species such as rats. Figure 5-2 provides LOAEL and NOAEL PODs from Katz et al. (1980), Egan et al. (1980), and Krasavage and O'Donoghue (1977) as a comparison to the four BMCL endpoints from the Johnson et al. (1977) study.

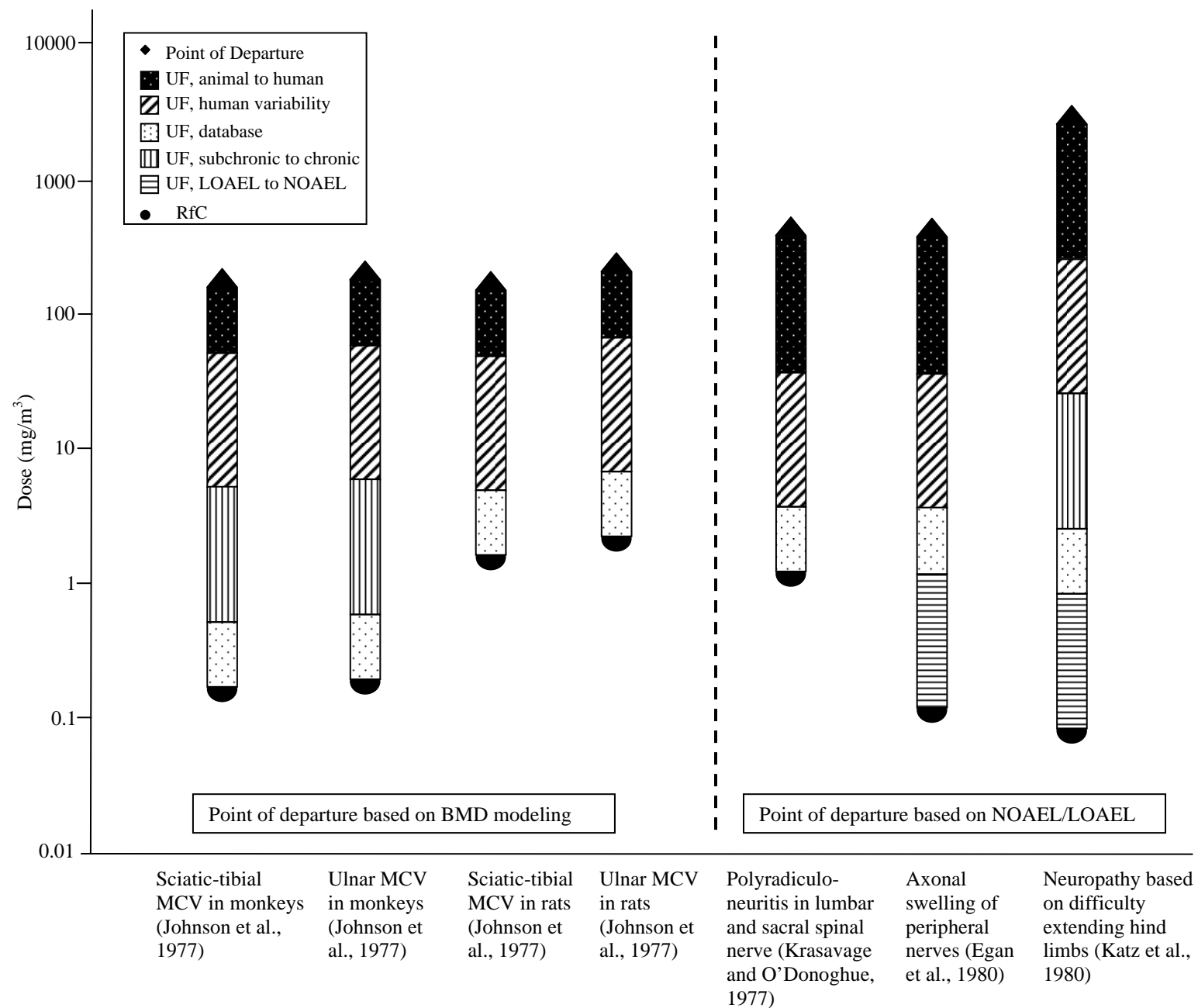


Figure 5-2. PODs for endpoints from select studies from Table 4-16, with corresponding applied UFs and derived RfCs.

5.2.6. Previous Inhalation Assessment

No previous RfC assessment for 2-hexanone exists on IRIS.

5.3. CANCER ASSESSMENT

As discussed in Section 4.6.1, the available database for 2-hexanone contains inadequate information to assess the carcinogenic potential according to *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a).

6. MAJOR CONCLUSIONS IN THE CHARACTERIZATION OF HAZARD AND DOSE RESPONSE

6.1. HUMAN HAZARD POTENTIAL

2-Hexanone (methyl butyl ketone, CASRN 591-78-6) has the chemical formula $C_6H_{12}O$ and a molecular weight of 100.16. It is a clear, volatile, flammable fluid with a pungent, acetone-like odor. 2-Hexanone is most commonly used as a paint or printing ink thinner, as a solvent for oils, waxes, and resins, or as a cleaning agent. It is currently not produced commercially in the U.S., and no information on importation is available (ATSDR, 1992).

2-Hexanone is well absorbed by the inhalation route and in the gastrointestinal tract. Animal studies suggest that 2-hexanone does not penetrate skin very efficiently, but there is evidence that it penetrates human skin easily (Bos et al., 1991). The distribution of 2-hexanone has not been studied thoroughly. In a rat study, it appeared in the plasma and the lung at higher concentrations than in the liver after both oral and inhalation administration (Duguay and Plaa, 1995) but did not show an affinity for a lipid rich tissue such as the brain (Granvil et al., 1994). In guinea pigs, 2-hexanone was eliminated quite rapidly, with a half-life of a little more than 1 hour for the parent compound and values not exceeding 2½ hours for the major metabolites (DiVincenzo et al., 1976). In rats, on the other hand, 2-hexanone was eliminated more slowly (Bus et al., 1981). The biological half-life of 2-hexanone in humans is not known. A PBTK model has not been proposed.

Metabolites of 2-hexanone include 2-hexanol, 2,5-hexanediol, 5-hydroxy-2-hexanone, 2,5-hexanedione, and some cyclic furan derivatives. The enzymes that metabolize 2-hexanone have not been characterized well. Among the metabolites of 2-hexanone, 2,5-hexanedione is the most important because it is a well-known neurotoxicant. It causes neuropathy specifically of the peripheral giant axons that involves neurofilament cross-linking and axonal swelling and proceeds to retrograde axonal degeneration. 2-Hexanone-induced neuropathy has been observed clinically in occupationally exposed humans (Davenport et al., 1976; Mallov, 1976; Allen et al., 1974; Billmaier et al., 1974), but the findings are frequently obscured by coexposure to other solvents, most frequently MEK, which is known to potentiate the toxicity of 2-hexanone.

A significant number of studies have been conducted in which laboratory animals were exposed orally or via inhalation for up to 2 years. Oral exposure studies used rats (Krasavage et al., 1980) and guinea pigs (Abdel-Rahman et al., 1978), with doses ranging up to 600 mg/kg-day by gavage or up to 1.3% in drinking water (amounting to 1010 mg/kg-day). The 13-month study in rats by O'Donoghue et al. (1978) that gave a detailed report of neuropathy incidences was used in the RfD assessment for 2-hexanone. Inhalation studies employed rats (Egan et al., 1980; Katz et al., 1980; Duckett et al., 1979, 1974; Johnson et al., 1977; Krasavage and O'Donoghue, 1977; Saida et al., 1976; Spencer et al., 1975), cats (O'Donoghue and Krasavage, 1979), and

monkeys (Johnson et al., 1977), with exposures ranging from 10–1300 ppm (41–5325 mg/m³). The hallmark symptom observed in any of these studies was neuropathy. The 2-hexanone-induced neuropathy also has been characterized mechanistically in animal studies (DeCaprio et al., 1988, 1982). Also a study in beagles that received 2-hexanone via the subcutaneous route reported neuropathy (O'Donoghue and Krasavage, 1981).

It is not clear whether 2-hexanone causes any other significant illness in humans. The available animal studies do not provide sufficient information to assess carcinogenicity of 2-hexanone. Currently, there is no evidence that this chemical causes cancer in humans or animals. According to *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), there is “inadequate information to assess the carcinogenic potential” of 2-hexanone.

Estimates of risks from other organizations

Estimates of risk for 2-hexanone derived by other organizations are compiled by the National Library of Medicine and can be found on the TOXNET Web page at <http://toxnet.nlm.nih.gov/>.

6.2. DOSE RESPONSE

6.2.1. Noncancer/Oral

There are no chronic or subchronic data for oral exposure of humans to 2-hexanone. There is no standard 2-year bioassay for 2-hexanone in any animal species; the only study with a chronic exposure time, the 13-month study in rats by O'Donoghue et al. (1978), was adequately conducted and reported critical, chemical-related effects with sufficient detail to be eligible as the principal study. Myofibrillar atrophy of the quadriceps muscle and the calf muscle in male rats is the critical endpoint evaluated. This endpoint was chosen over other neuropathic endpoints because it occurs due to axonal atrophy, an endpoint that correlates best to nerve dysfunction regardless of route of exposure. Another endpoint described in O'Donoghue et al. (1978) was peripheral nerve axonopathy. This endpoint was not well characterized, with incidences jumping from 0% (0/10 animals) in controls to 80% (8/10 animals) at the lowest dose (143 mg/kg-day) and 100% (10/10 animals) at both higher doses (266 and 560 mg/kg-day). Though peripheral nerve axonopathy may have exhibited the most sensitive response to 2-hexanone exposure, axonal swelling may not be an appropriate proximal marker of neuropathy because this endpoint poorly correlates with nerve dysfunction.

The RfD of 2×10^{-1} mg/kg-day was derived from myofibrillar atrophy of the quadriceps muscle in male COBS/CD(SD)BR rats following 13 months of oral exposure to 2-hexanone (O'Donoghue et al., 1978). There is sufficient evidence from other studies in experimental animals to confirm that the nervous system is the primary target for the toxicological effects of 2-hexanone (Abdo et al., 1982; Krasavage et al., 1980; Eben et al., 1979; Abdel-Rahman et al.,

1978; Homan et al., 1977). Because there are no compelling biological reasons to choose myofibrillar atrophy of the quadriceps muscle over myofibrillar atrophy of the calf muscle, the slightly lower BMDL was chosen. A graphical comparison of the RfDs from these two endpoints is illustrated in Figure 5-1.

A composite UF of 300 was applied; 10 for intraspecies (interindividual) variability, 10 for interspecies variability, and 3 for database uncertainty. Information was unavailable to quantitatively assess toxicokinetic or toxicodynamic differences between animals and humans and the potential variability in human susceptibility; thus, the interspecies and intraspecies UFs of 10 were applied. A threefold database deficiency UF was applied to reflect that, though chronic and subchronic information on 2-hexanone was available, there are no 2-hexanone-specific multigenerational reproductive and developmental studies. Developmental studies on n-hexane, a precursor of 2-hexanone and 2,5-hexanedione, have shown low risk of toxicity, but there is still a level of concern because available studies on 2-hexanone via inhalation exposure have suggested the possibility of immunotoxicity and reproductive toxicity. Rat developmental neurotoxicity studies with 2,5-hexanedione have found minimal effects (e.g., aggregated and fused axons, identified with electron microscopy) from daily s.c. injections on GDs 12–20 with 340 mg/kg 2,5-hexanedione. Thus, due to the absence of studies specifically evaluating immunotoxicity or reproductive toxicity of 2-hexanone via an oral route of exposure, a UF of 3 was applied to account for database deficiency.

The overall confidence in this RfD assessment is medium. Confidence in the principal study (O'Donoghue et al., 1978) is medium. The study involves a comparatively low but acceptable number of animals per group (10) and reports clinical neurological deficits and neuropathologic effects within a dose range in which LOAEL could be identified for the critical effect. Animal studies in two additional species (guinea pigs and hens) corroborate the primacy of the neurological endpoint and confirm the validity of peripheral neuropathy as the critical effect. Confidence in the database is medium. The database lacks chronic exposure information on pure 2-hexanone via any route of exposure, as well as a multigenerational developmental and reproductive toxicity study and a developmental neurotoxicity study. The chronic drinking water study of O'Donoghue et al. (1978) satisfies the minimum oral database requirements for deriving an RfD for 2-hexanone. Reflecting medium confidence in the principal study and medium confidence in the database, confidence in the RfD is medium.

6.2.2. Noncancer/Inhalation

Dose-dependent development of 2-hexanone-induced neuropathy was confirmed in numerous subchronic studies in rats (Egan et al., 1980; Katz et al., 1980; Duckett et al., 1979, 1974; Johnson et al., 1977; Krasavage and O'Donoghue, 1977; Saida et al., 1976; Spencer et al., 1975) and one chronic study in cats (O'Donoghue and Krasavage, 1979). One 10-month study was Johnson et al. (1977), using two different species (monkeys and rats; n = 8 and n = 10 per

group, respectively) with two concentrations of 2-hexanone (commercial grade). Johnson et al. (1977) utilized four sensitive neurological tests to identify subtle changes in treated versus control animals. The study by Johnson et al. (1977) was chosen as the most suitable study for RfC development. Both sciatic-tibial MCV and ulnar MCV in 2-hexanone-exposed monkeys and rats were considered in deriving the RfC. A graphical comparison of the potential RfCs from these endpoints, as well as other endpoints and studies considered, are illustrated in Figure 5-2. Because monkeys have a similar respiratory tract and breathing patterns to humans and 2,5-hexanedione, the metabolite of 2-hexanone, typically affects long axons such as the sciatic-tibial nerve prior to other nerves, sciatic-tibial nerve MCV in monkeys was used to derive the RfC. It should be noted that ulnar nerve MCV in monkeys and sciatic-tibial nerve MCV in rats were found to have similar BMCL_{HEC} as the endpoint selected above and would not result in significantly different RfCs if those alternatives were utilized.

The RfC of $2 \times 10^{-1} \text{ mg/m}^3$ was derived from the decrease in sciatic-tibial MCV in monkeys exposed to 2-hexanone for 10 months (Johnson et al., 1977). A composite UF of 1000 was applied in the derivation of the RfC: a default of 10 for intraspecies (interindividual) variability, a default of 10 for subchronic-to-chronic uncertainty, 3 for interspecies variability, and 3 for database uncertainty. Information was unavailable to predict potential variability in susceptibility among the population; thus, the intraspecies variability UF of 10 was applied. A subchronic-to-chronic UF of 10 was applied to account for the less-than-lifetime exposure in the principal study, because the data utilized for calculating the RfC were based on values obtained at 25 weeks. An interspecies UF of 3 (rather than 10) was applied because a dosimetric adjustment was made. A UF of 3 was applied to account for database deficiencies. Although a developmental study exists that did not identify a NOAEL (a LOAEL of 1000 ppm was identified), the available developmental studies with n-hexane, a precursor of 2-hexanone, provide support for applying a UF_D of 3. Namely, developmental studies with n-hexane concentrations of 100 (GDs 6–15), 400 (GDs 6–15), or 1000 ppm (GDs 8–16) have been negative; a teratology study in which dams were exposed to n-hexane identified nearly double the highest NOAEL identified from inhalation studies with 2-hexanone. Also, rat developmental neurotoxicity studies with 2,5-hexanedione, the ultimate toxic metabolite of n-hexane and 2-hexanone, have found minimal effects from daily s.c. injections on GDs 12–20 with 340 mg/kg 2,5-hexanedione.

The overall confidence in this RfC assessment is medium. Confidence in the principal study is medium; it involves exposures in two species via the inhalation route and sensitive diagnostic tests for determining treatment-related neurotoxicity. In addition, animal studies in four different species (monkeys, rats, cats, and hens) and occupational exposures corroborate the primacy of the neurological endpoint and confirm the relevance of the critical effect for decreased MCV values.

6.2.3. Cancer

Under the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), there is inadequate information to assess the carcinogenic potential of 2-hexanone. As such, data are unavailable to calculate quantitative cancer risk estimates.

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APPENDIX B-1. DOSE-RESPONSE MODELING FOR DERIVATION OF AN RfD FOR 2-HEXANONE

B-1.1. METHODS

The models in U.S. EPA's benchmark dose (BMD) software (BMDS) (version 1.3.2) were fit to data sets for myofibrillar atrophy of the quadriceps and calf muscle in a 13-month drinking water study with exposure to 2-hexanone (O'Donoghue, 1978). The dose levels used were those reported in the study. A BMR of a 10% extra risk of myofibrillar atrophy of the quadriceps muscle or calf muscle was selected under an assumption that it represents a minimal biologically significant change. Models were run using the default restrictions on parameters built into the BMDS.

B-1.2. RESULTS

The BMD modeling results for myofibrillar atrophy of the quadriceps and calf muscles are summarized in Table B-1.1 and Table B-1.2, respectively. The tables show the BMDs and the 95% lower bounds on the doses (BMDLs) derived from each endpoint modeled. The remainder of this section shows detailed summaries of the best-fit models for myofibrillar atrophy of the quadriceps and calf muscles, presented sequentially.

Table B-1.1. BMD modeling results for animals with myofibrillar atrophy of the quadriceps muscle

Model	AIC ^a	<i>p</i> Value	BMD	BMDL	BMD/BMDL
Gamma multi-hit	24.6565	0.9034	150.665	86.2822	1.746189
Log logistic	25.1541	0.7602	156.315	96.3847	1.621782
Logistic	24.5648	0.9392	155.905	94.5301	1.649263
Multistage	22.3952	0.9995	141.38	49.9434	2.830804
Log probit	24.9892	0.7974	152.728	98.9646	1.543259
Probit	24.4241	0.9822	148.53	86.9997	1.707247
Quantal linear	29.9156	0.1628	34.9514	22.8722	1.528117
Quantal quadratic	23.7952	0.8118	100.711	78.3857	1.284813
Weibull	24.3816	0.9945	145.517	78.2565	1.859488

^aAIC = Akaike Information Criterion.

Table B-1.2. BMD modeling results for animals with myofibrillar atrophy of the calf muscle

Model	AIC^a	<i>p</i> Value	BMD	BMDL	BMD/BMDL
Gamma multi-hit	27.7837	0.8972	117.834	48.9374	2.407852
Log logistic	28.36	0.7402	122.843	63.2262	1.942913
Logistic	27.9769	0.8457	120.47	70.8106	1.701299
Multistage	27.4841	0.9956	95.8576	30.1238	3.182122
Log probit	28.0906	0.8019	123.737	67.2193	1.840796
Probit	27.7116	0.9227	114.43	65.7944	1.739206
Quantal linear	30.2036	0.3498	28.7546	19.0312	1.510919
Quantal quadratic	25.8664	0.9701	88.7125	69.2097	1.281793
Weibull	27.5386	0.9756	109.348	45.8927	2.382688

^aAIC = Akaike Information Criterion.

Multistage Model
MYOFIBRILLAR ATROPHY OF THE QUADRICEPS MUSCLE

BMDS MODEL RUN

The form of the probability function is:

$$P[\text{response}] = \text{background} + (1 - \text{background}) * [1 - \exp(-\beta_1 * \text{dose} - \beta_2 * \text{dose}^2 - \beta_3 * \text{dose}^3)]$$

The parameter betas are restricted to be positive

Dependent variable = Incidence

Independent variable = Dose

Total number of observations = 4

Total number of records with missing values = 0

Total number of parameters in model = 4

Total number of specified parameters = 0

Degree of polynomial = 3

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

Default Initial Parameter Values

Background = 0
 Beta(1) = 0
 Beta(2) = 0
 Beta(3) = 5.88262e+011

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -Background -Beta(1) -Beta(2)
 have been estimated at a boundary point, or have been specified by the user,
 and do not appear in the correlation matrix)

Beta(3)

Beta(3) 1

Parameter Estimates

Variable	Estimate	Std. Err.
Background	0	NA
Beta(1)	0	NA
Beta(2)	0	NA
Beta(3)	3.72829e-008	2.10431e-008

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-10.1823			
Fitted model	-10.1976	0.0306013	3	0.9986
Reduced model	-26.9205	33.4763	3	<.0001

AIC: 22.3952

Goodness of Fit

	Dose	Est._Prob.	Expected	Observed	Size	Chi^2 Res.
i: 1	0.0000	0.0000	0.000	0	10	0.000
i: 2	143.0000	0.1033	1.033	1	10	-0.036
i: 3	266.0000	0.5043	5.043	5	10	-0.017
i: 4	560.0000	0.9986	9.986	10	10	1.001

Chi-square = 0.02 DF = 3 P-value = 0.9995

Benchmark Dose Computation

Specified effect = 0.1

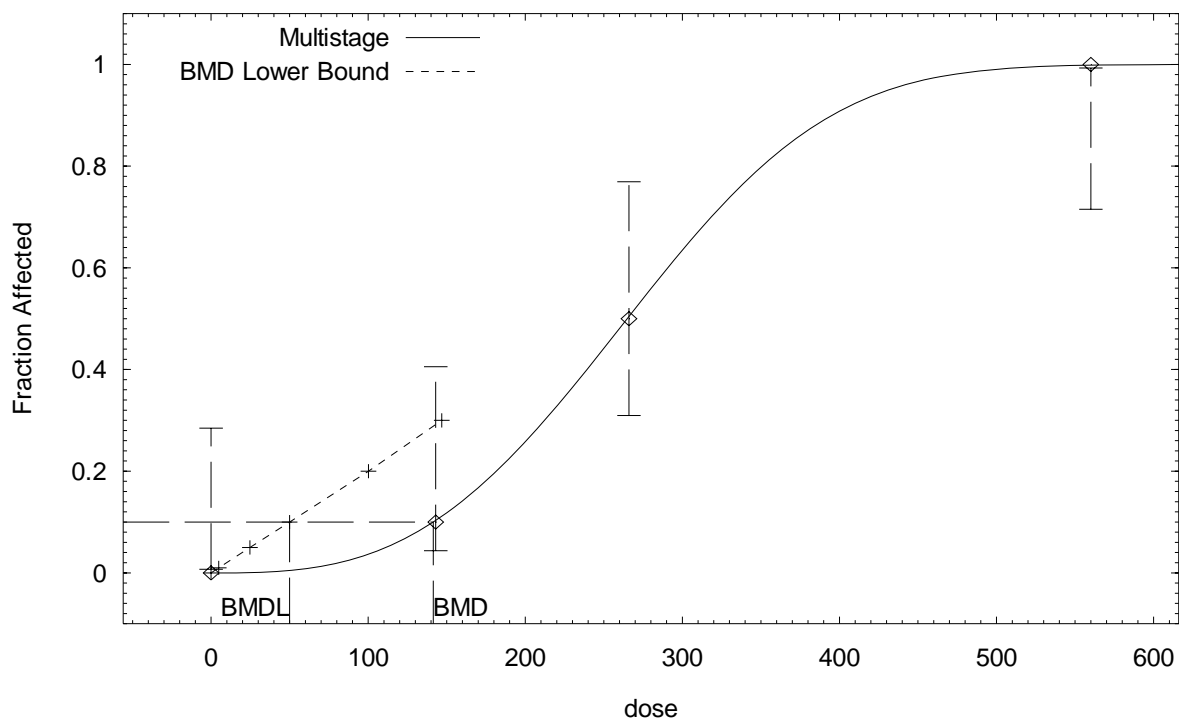
Risk Type = Extra risk

Confidence level = 0.95

BMD = 141.38

BMDL = 49.9434

Multistage Model with 0.95 Confidence Level



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Quantal Quadratic Model MYOFIBRILLAR ATROPHY OF THE CALF MUSCLE

BMDS MODEL RUN

The form of the probability function is:

$$P[\text{response}] = \text{background} + (1 - \text{background}) * [1 - \text{EXP}(-\text{slope} * \text{dose}^2)]$$

Dependent variable = Incidence

Independent variable = Dose

Total number of observations = 4

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

Default Initial (and Specified) Parameter Values

Background = 0.0454545

Slope = 9.7083e-006

Power = 2 Specified

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -Background -Power
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

Slope

Slope 1

Parameter Estimates

Variable	Estimate	Std. Err.
Background	0	NA
Slope	1.33878e-005	4.17409e-006

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-11.7341			
Fitted model	-11.9332	0.398099	3	0.9406
Reduced model	-27.5256	31.5828	3	<.0001

AIC: 25.8664

Goodness of Fit

Dose	Est._Prob.	Expected	Scaled		Residual
			Observed	Size	
0.0000	0.0000	0.000	0	10	0
143.0000	0.2395	2.395	2	10	-0.2926
266.0000	0.6122	6.122	6	10	-0.07918
560.0000	0.9850	9.850	10	10	0.3905

Chi-square = 0.24 DF = 3 P-value = 0.9701

Benchmark Dose Computation

Specified effect = 0.1

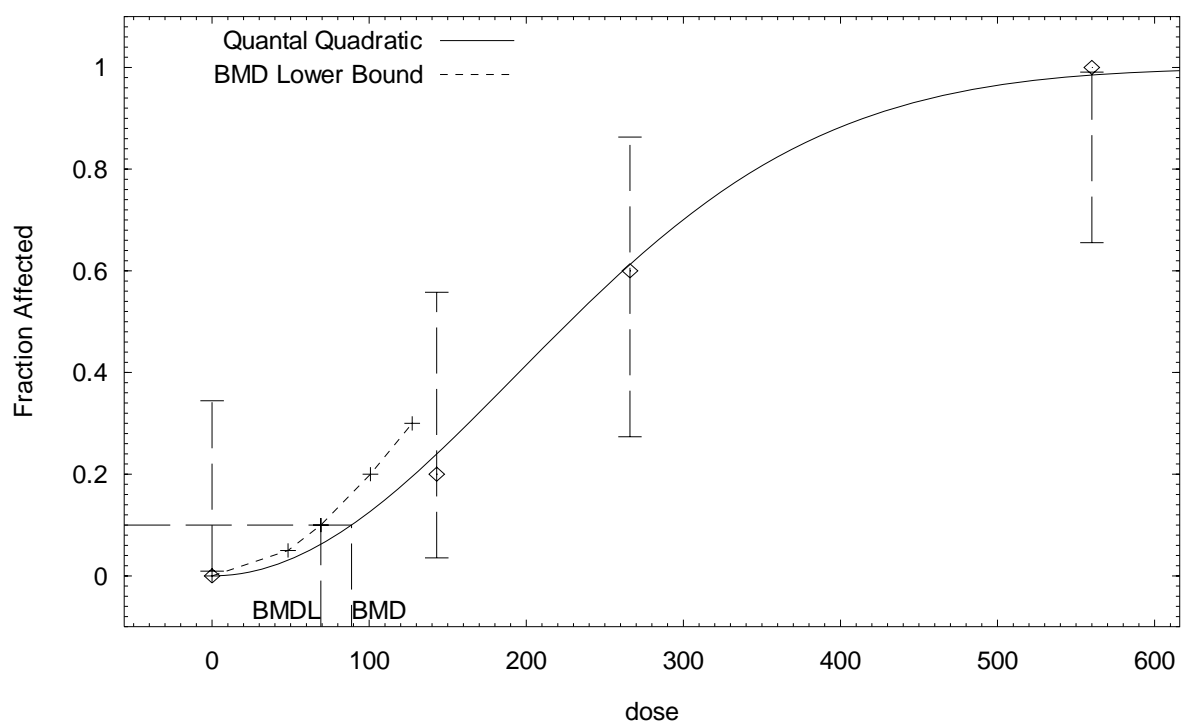
Risk Type = Extra risk

Confidence level = 0.95

BMD = 88.7125

BMDL = 69.2097

Quantal Quadratic Model with 0.95 Confidence Level



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APPENDIX B-2. EXPOSURE-RESPONSE MODELING FOR DERIVATION OF AN RfC FOR 2-HEXANONE

B-2.1. METHODS

The models in U.S. EPA's benchmark dose (BMD) software (BMDS) (version 1.3.2) were fit to multiple data sets presented in an inhalation study with exposure to monkeys and rats (Johnson et al., 1977). Motor conduction velocity (MCV) was determined to be the most relevant endpoint in both species and was modeled for the sciatic and tibial nerves. The exposure concentrations used were those reported in the study. The U.S. EPA (2000c) BMD methodology suggests that in the absence of any other idea of what level of response to consider adverse, a change in the mean equal to one control standard deviation from the control should be used as the benchmark response (BMR). A BMR of a 10% change in nerve conduction velocity from the control mean was selected under an assumption that it represents a minimal biologically significant change. Thus, a 10% BMR was utilized in the derivation of the reference concentration (RfC).

B-2.2. RESULTS

The BMD modeling results are summarized in Table B-2.1. This table shows the BMDs and 95% lower bounds on doses (BMDLs) derived from each endpoint modeled in monkeys and rats. The remainder of this section shows detailed summaries of the modeling results for monkey sciatic and ulnar nerves for both monkeys and rats (all 1st degree polynomial), presented sequentially.

Table B-2.1. Summary of BMDS modeling results for 2-hexanone

Animal/endpoint	Model ^a	<i>p</i> Value	AIC ^b	BMD	BMDL
Monkey sciatic-tibial nerve (MCV at 6 months)	1st degree polynomial	0.59	105.59	293.184	243.262
	2 nd degree polynomial	--	107.29	169.254	68.8063
	Power	--	105.58	293.184	243.262
	Hill	--	--	--	--
Monkey ulnar nerve (MCV at 6 months)	1st degree polynomial	0.90	105.31	334.691	278.471
	2 nd degree polynomial	--	107.29	293.8	94.1174
	Power	--	109.31	334.691	278.471
	Hill	--	--	--	--
Rat sciatic-tibial nerve (MCV at 25 weeks)	1st degree polynomial	0.79	121.55	270.959	232.105
	2 nd degree polynomial	--	123.48	355.94	100.045
	Power	--	125.48	329.704	232.56
	Hill	--	--	--	--
Rat ulnar nerve (MCV at 25 weeks)	1st degree polynomial	0..26	122.77	470.964	352.274
	2 nd degree polynomial	--	606.18	133.181	87.3758
	Power	--	125.48	177.555	14.1388
	Hill	--	--	--	--

^aBolded values were the models used for further evaluation and RfC derivation.

^bAIC = Akaike Information Criterion.

=====

1st Degree Polynomial Model
MONKEYS MCV SCIATIC TIBIAL

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BMDS MODEL RUN

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The form of the response function is:

$$Y[\text{dose}] = \text{beta}_0 + \text{beta}_1 * \text{dose} + \text{beta}_2 * \text{dose}^2 + \dots$$

Dependent variable = MEAN

Independent variable = Dose

rho is set to 0

Signs of the polynomial coefficients are not restricted

A constant variance model is fit

Total number of dose groups = 3

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

Default Initial Parameter Values

alpha = 28.6225

rho = 0 Specified

beta\_0 = 49.3606

beta\_1 = -0.0168361

Parameter Estimates

| Variable | Estimate   | Std. Err. | 95.0% Wald Confidence Interval |                   |
|----------|------------|-----------|--------------------------------|-------------------|
|          |            |           | Lower Conf. Limit              | Upper Conf. Limit |
| alpha    | 25.351     | 7.31821   | 11.0076                        | 39.6944           |
| beta_0   | 49.3606    | 1.3264    | 46.761                         | 51.9603           |
| beta_1   | -0.0168361 | 0.0023421 | -0.0214265                     | -0.0122456        |

Asymptotic Correlation Matrix of Parameter Estimates

|        | alpha     | beta_0   | beta_1    |
|--------|-----------|----------|-----------|
| alpha  | 1         | 4.8e-015 | -5.1e-015 |
| beta_0 | 4.8e-015  | 1        | -0.63     |
| beta_1 | -5.1e-015 | -0.63    | 1         |

Table of Data and Estimated Values of Interest

| Dose | N | Obs Mean | Obs Std Dev | Est Mean | Est Std Dev | Chi^2 |
|------|---|----------|-------------|----------|-------------|-------|
| Res. |   |          |             |          |             |       |
| 0    | 8 | 50       | 5.35        | 49.4     | 5.03        | 0.359 |

|     |   |    |      |      |      |        |
|-----|---|----|------|------|------|--------|
| 98  | 8 | 47 | 5.35 | 47.7 | 5.03 | -0.399 |
| 976 | 8 | 33 | 5.35 | 32.9 | 5.03 | 0.0401 |

Model Descriptions for likelihoods calculated

Model A1:  $Y_{ij} = \mu(i) + e(ij)$   
 $\text{Var}\{e(ij)\} = \sigma^2$

Model A2:  $Y_{ij} = \mu(i) + e(ij)$   
 $\text{Var}\{e(ij)\} = \sigma(i)^2$

Model R:  $Y_i = \mu + e(i)$   
 $\text{Var}\{e(i)\} = \sigma^2$

Warning: Likelihood for model A1 larger than the Likelihood for model A2.

#### Likelihoods of Interest

| Model  | Log(likelihood) | DF | AIC        |
|--------|-----------------|----|------------|
| A1     | -50.647941      | 4  | 109.295882 |
| A2     | -50.647941      | 6  | 113.295882 |
| fitted | -50.793824      | 2  | 105.587648 |
| R      | -65.085067      | 2  | 134.170135 |

Test 1: Does response and/or variances differ among dose levels

(A2 vs. R)

Test 2: Are Variances Homogeneous (A1 vs A2)

Test 3: Does the Model for the Mean Fit (A1 vs. fitted)

#### Tests of Interest

| Test | $-2 \cdot \log(\text{Likelihood Ratio})$ | Test df | p-value |
|------|------------------------------------------|---------|---------|
|------|------------------------------------------|---------|---------|

|        |               |   |        |
|--------|---------------|---|--------|
| Test 1 | 28.8743       | 4 | <.0001 |
| Test 2 | -1.42109e-014 | 2 | <.0001 |
| Test 3 | 0.291767      | 1 | 0.5891 |

The p-value for Test 1 is less than .05. There appears to be a difference between response and/or variances among the dose levels.  
It seems appropriate to model the data

The p-value for Test 2 is less than .05. Consider running a non-homogeneous variance model

The p-value for Test 3 is greater than .05. The model chosen appears to adequately describe the data

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Relative risk

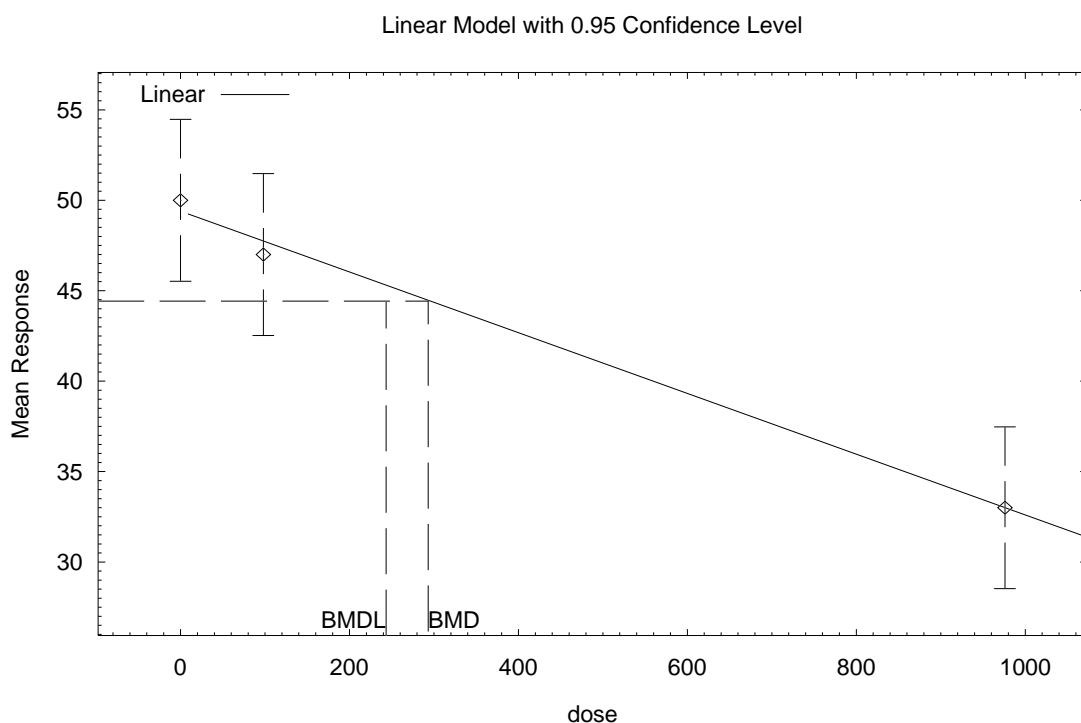
Confidence level = 0.95

BMD = 293.184

BMDL = 243.262

BMDL computation failed for one or more points on the BMDL curve.

The BMDL curve will not be plotted



09:06 05/01 2007

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**1<sup>st</sup> Degree Polynomial Model.**  
MONKEYS MCV ULNAR

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BMDS MODEL RUN

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The form of the response function is:

$Y[\text{dose}] = \text{beta}_0 + \text{beta}_1 * \text{dose} + \text{beta}_2 * \text{dose}^2 + \dots$

Dependent variable = MEAN
 Independent variable = Dose
 rho is set to 0
 Signs of the polynomial coefficients are not restricted
 A constant variance model is fit

Total number of dose groups = 3
 Total number of records with missing values = 0
 Maximum number of iterations = 250
 Relative Function Convergence has been set to: 1e-008
 Parameter Convergence has been set to: 1e-008

Default Initial Parameter Values

alpha = 28.6225
 rho = 0 Specified
 beta_0 = 57.8551
 beta_1 = -0.0172861

Parameter Estimates

Variable	Estimate	95.0% Wald Confidence Interval		
		Std. Err.	Lower Conf. Limit	Upper Conf. Limit
alpha	25.0604	7.23432	10.8814	39.2394
beta_0	57.8551	1.31877	55.2704	60.4399
beta_1	-0.0172861	0.00232864	-0.0218502	-0.0127221

Asymptotic Correlation Matrix of Parameter Estimates

	alpha	beta_0	beta_1
alpha	1	-4.3e-012	8.7e-012
beta_0	-4.3e-012	1	-0.63
beta_1	8.7e-012	-0.63	1

Table of Data and Estimated Values of Interest

Dose	N	Obs Mean	Obs Std Dev	Est Mean	Est Std Dev	Chi^2
Res.						
0	8	58	5.35	57.9	5.01	0.0819
98	8	56	5.35	56.2	5.01	-0.091
976	8	41	5.35	41	5.01	0.00914

Model Descriptions for likelihoods calculated

Model A1: $Y_{ij} = \mu(i) + e(ij)$
 $\text{Var}\{e(ij)\} = \sigma^2$

Model A2: $Y_{ij} = \mu(i) + e(ij)$
 $\text{Var}\{e(ij)\} = \sigma(i)^2$

Model R: $Y_i = \mu + e(i)$
 $\text{Var}\{e(i)\} = \sigma^2$

Warning: Likelihood for model A1 larger than the Likelihood for model A2.

Likelihoods of Interest

Model	Log(likelihood)	DF	AIC
A1	-50.647941	4	109.295882
A2	-50.647941	6	113.295882
fitted	-50.655476	2	105.310953
R	-65.478867	2	134.957734

Test 1: Does response and/or variances differ among dose levels

(A2 vs. R)

Test 2: Are Variances Homogeneous (A1 vs A2)

Test 3: Does the Model for the Mean Fit (A1 vs. fitted)

Tests of Interest

Test $-2 \cdot \log(\text{Likelihood Ratio})$ Test df p-value

Test 1	29.6619	4	<.0001
Test 2	-1.42109e-014	2	<.0001
Test 3	0.0150715	1	0.9023

The p-value for Test 1 is less than .05. There appears to be a difference between response and/or variances among the dose levels.

It seems appropriate to model the data

The p-value for Test 2 is less than .05. Consider running a non-homogeneous variance model

The p-value for Test 3 is greater than .05. The model chosen appears to adequately describe the data

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Relative risk

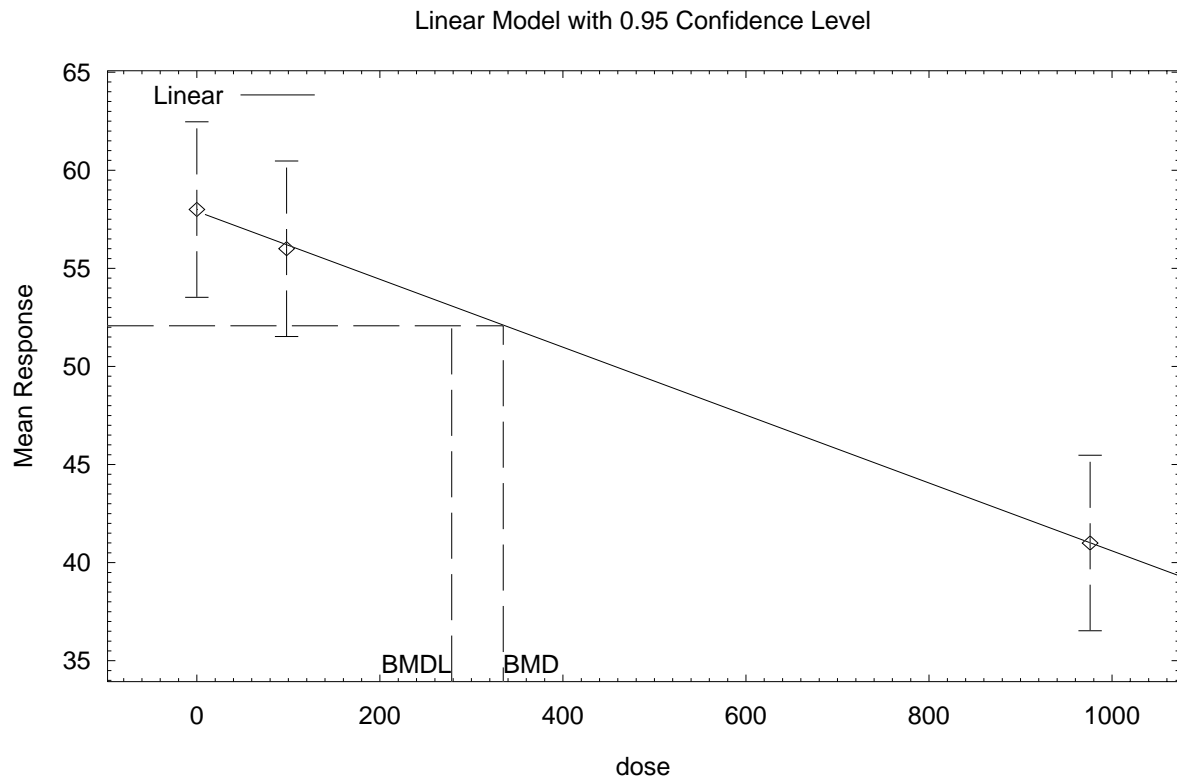
Confidence level = 0.95

BMD = 334.691

BMDL = 278.471

BMDL computation failed for one or more points on the BMDL curve.

The BMDL curve will not be plotted



10:20 05/01 2007

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1st Degree Polynomial Model.
RATS MCV SCIATIC TIBIAL
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BMDS MODEL RUN

~~~~~  
The form of the response function is:

$$Y[\text{dose}] = \text{beta}_0 + \text{beta}_1 * \text{dose} + \text{beta}_2 * \text{dose}^2 + \dots$$

Dependent variable = MEAN

Independent variable = Dose

rho is set to 0

Signs of the polynomial coefficients are not restricted

A constant variance model is fit

Total number of dose groups = 3

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

#### Default Initial Parameter Values

alpha = 20.5209  
 rho = 0 Specified  
 beta\_0 = 42.2427  
 beta\_1 = -0.0155901

#### Parameter Estimates

| Variable | Estimate   | Std. Err.  | 95.0% Wald Confidence Interval |                   |
|----------|------------|------------|--------------------------------|-------------------|
|          |            |            | Lower Conf. Limit              | Upper Conf. Limit |
| alpha    | 18.5129    | 4.78001    | 9.14425                        | 27.8815           |
| beta_0   | 42.2427    | 1.01325    | 40.2568                        | 44.2287           |
| beta_1   | -0.0155901 | 0.00178935 | -0.0190972                     | -0.012083         |

#### Asymptotic Correlation Matrix of Parameter Estimates

|        | alpha     | beta_0    | beta_1   |
|--------|-----------|-----------|----------|
| alpha  | 1         | -4.2e-008 | 6.6e-008 |
| beta_0 | -4.2e-008 | 1         | -0.63    |
| beta_1 | 6.6e-008  | -0.63     | 1        |

#### Table of Data and Estimated Values of Interest

| Dose | N  | Obs Mean | Obs Std Dev | Est Mean | Est Std Dev | Chi^2   |
|------|----|----------|-------------|----------|-------------|---------|
| Res. |    |          |             |          |             |         |
| 0    | 10 | 42       | 4.53        | 42.2     | 4.3         | -0.178  |
| 97   | 10 | 41       | 4.53        | 40.7     | 4.3         | 0.198   |
| 976  | 10 | 27       | 4.53        | 27       | 4.3         | -0.0197 |

#### Model Descriptions for likelihoods calculated

Model A1:  $Y_{ij} = \mu(i) + e(ij)$   
 $\text{Var}\{e(ij)\} = \sigma^2$

Model A2:  $Y_{ij} = \mu(i) + e(ij)$   
 $\text{Var}\{e(ij)\} = \sigma(i)^2$

Model R:  $Y_i = \mu + e(i)$   
 $\text{Var}\{e(i)\} = \sigma^2$

#### Likelihoods of Interest

| Model  | Log(likelihood) | DF | AIC        |
|--------|-----------------|----|------------|
| A1     | -58.741250      | 4  | 125.482501 |
| A2     | -58.741250      | 6  | 129.482501 |
| fitted | -58.777017      | 2  | 121.554034 |

R       -78.206652     2    160.413304

Test 1: Does response and/or variances differ among dose levels

(A2 vs. R)

Test 2: Are Variances Homogeneous (A1 vs A2)

Test 3: Does the Model for the Mean Fit (A1 vs. fitted)

#### Tests of Interest

| Test | -2*log(Likelihood Ratio) | Test df | p-value |
|------|--------------------------|---------|---------|
|------|--------------------------|---------|---------|

|        |         |   |        |
|--------|---------|---|--------|
| Test 1 | 38.9308 | 4 | <.0001 |
|--------|---------|---|--------|

|        |   |   |   |
|--------|---|---|---|
| Test 2 | 0 | 2 | 1 |
|--------|---|---|---|

|        |           |   |        |
|--------|-----------|---|--------|
| Test 3 | 0.0715333 | 1 | 0.7891 |
|--------|-----------|---|--------|

The p-value for Test 1 is less than .05. There appears to be a difference between response and/or variances among the dose levels.

It seems appropriate to model the data

The p-value for Test 2 is greater than .05. A homogeneous variance model appears to be appropriate here

The p-value for Test 3 is greater than .05. The model chosen appears to adequately describe the data

Benchmark Dose Computation  
Specified effect =        0.1

Risk Type        =    Relative risk

Confidence level =        0.95

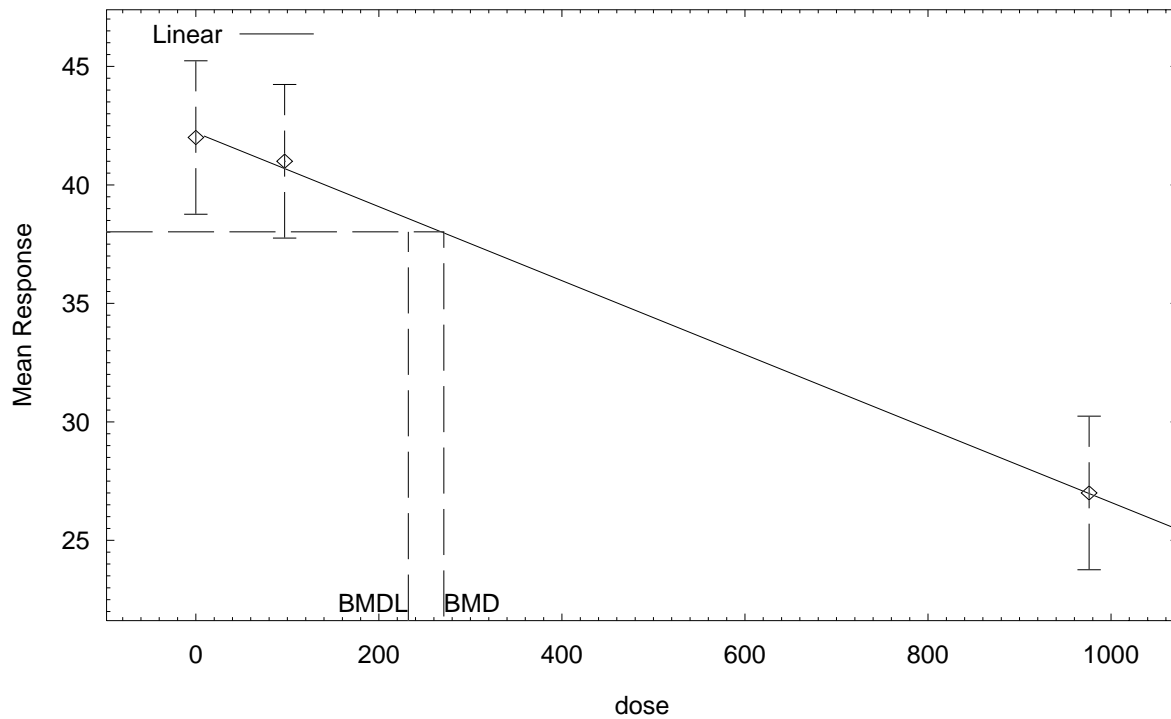
BMD =        270.959

BMDL =        232.105

BMDL computation failed for one or more points on the BMDL curve.

The BMDL curve will not be plotted

Linear Model with 0.95 Confidence Level



12:45 05/01 2007

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**1<sup>st</sup> Degree Polynomial Model.**

RATS MCV ULNAR

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BMDS MODEL RUN

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The form of the response function is:

$$Y[\text{dose}] = \text{beta}_0 + \text{beta}_1 * \text{dose} + \text{beta}_2 * \text{dose}^2 + \dots$$

Dependent variable = MEAN

Independent variable = Dose

rho is set to 0

Signs of the polynomial coefficients are not restricted

A constant variance model is fit

Total number of dose groups = 3

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

Default Initial Parameter Values

alpha = 20.5209
 rho = 0 Specified
 beta_0 = 38.9587
 beta_1 = -0.0082721

Parameter Estimates

Variable	Estimate	Std. Err.	95.0% Wald Confidence Interval	
			Lower Conf. Limit	Upper Conf. Limit
alpha	19.2803	4.97816	9.52331	29.0373
beta_0	38.9587	1.03404	36.932	40.9853
beta_1	-0.0082721	0.00182606	-0.0118511	-0.00469309

Asymptotic Correlation Matrix of Parameter Estimates

	alpha	beta_0	beta_1
alpha	1	7.6e-015	3.4e-015
beta_0	7.6e-015	1	-0.63
beta_1	3.4e-015	-0.63	1

Table of Data and Estimated Values of Interest

Dose	N	Obs Mean	Obs Std Dev	Est Mean	Est Std Dev	Chi^2 Res.
0	10	40	4.53	39	4.39	0.75
97	10	37	4.53	38.2	4.39	-0.833
976	10	31	4.53	30.9	4.39	0.0828

Model Descriptions for likelihoods calculated

Model A1: $Y_{ij} = \mu(i) + e(ij)$
 $\text{Var}\{e(ij)\} = \sigma^2$

Model A2: $Y_{ij} = \mu(i) + e(ij)$
 $\text{Var}\{e(ij)\} = \sigma(i)^2$

Model R: $Y_i = \mu + e(i)$
 $\text{Var}\{e(i)\} = \sigma^2$

Likelihoods of Interest

Model	Log(likelihood)	DF	AIC
A1	-58.741250	4	125.482501
A2	-58.741250	6	129.482501
fitted	-59.386277	2	122.772554
R	-67.712722	2	139.425445

Test 1: Does response and/or variances differ among dose

levels (A2 vs. R)

Test 2: Are Variances Homogeneous (A1 vs A2)

Test 3: Does the Model for the Mean Fit (A1 vs. fitted)

Tests of Interest

Test	-2*log(Likelihood Ratio)	Test df	p-value
------	--------------------------	---------	---------

Test 1	17.9429	4	0.000127
--------	---------	---	----------

Test 2	0	2	1
--------	---	---	---

Test 3	1.29005	1	0.256
--------	---------	---	-------

The p-value for Test 1 is less than .05. There appears to be a difference between response and/or variances among the dose levels. It seems appropriate to model the data

The p-value for Test 2 is greater than .05. A homogeneous variance model appears to be appropriate here

The p-value for Test 3 is greater than .05. The model chosen appears to adequately describe the data

Benchmark Dose Computation

Specified effect = 0.1

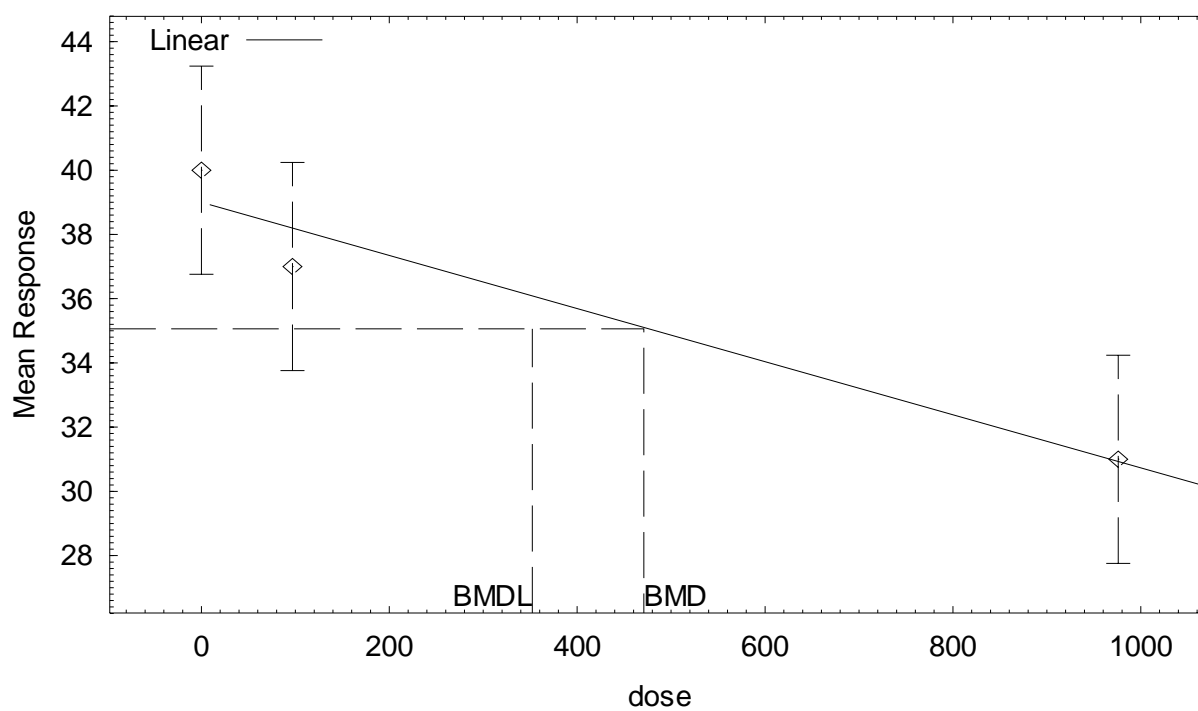
Risk Type = Relative risk

Confidence level = 0.95

BMD = 470.964

BMDL = 352.274

Linear Model with 0.95 Confidence Level



16:02 02/20 2008



Superfund Technical Support Center

National Center for Environmental Assessment

U.S. Environmental Protection Agency

26 West Martin Luther King Drive, MS-AG41

Cincinnati, Ohio 45268

Jon Reid/Director, Pat Daunt/Administrator

Hotline 513-569-7300, FAX 513-569-7159, E-Mail: STSC.Superfund@epa.gov

March 14, 2008

Michael Sivak
U.S. EPA, Region 2

ASSISTANCE REQUESTED: PPRTVs for 1,1,2-Trichloroethane, 1,2,4-Trimethylbenzene,
1,2-Dichloroethane, 1,2-Dichloropropane and 2,4-Dichlorophenol

ENCLOSED INFORMATION: Attachment 1: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR 1,1,2-TRICHLOROETHANE (CASRN
79-00-5) Derivation of Subchronic and Chronic
Inhalation RfCs**

Attachment 2: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR 1,2,4-TRIMETHYLBENZENE
(CASRN 95-63-6)**

Attachment 3: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR 1,2-DICHLOROETHANE
(ETHYLENE DICHLORIDE) (CASRN 107-06-2)
Derivation of a Chronic Oral RfD**

Attachment 4: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR 1,2-DICHLOROPROPANE (CASRN
78-87-5) Derivation of a Carcinogenicity Assessment**

Attachment 5: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR 2,4-DICHLOROPHENOL (CASRN
120-83-2)**

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (5)

cc: STSC Files

7-5-2006

Provisional Peer Reviewed Toxicity Values for

1,1,2-Trichloroethane (CASRN 79-00-5)

Derivation of Subchronic and Chronic Inhalation RfCs

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
1,1,2-TRICHLOROETHANE (CASRN 79-00-5)
Derivation of Subchronic and Chronic Inhalation RfCs**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

An RfC for 1,1,2-trichloroethane is not listed on IRIS (U.S. EPA, 2006) or in the HEAST (U.S. EPA, 1997). EPA review documents for 1,1,2-trichloroethane that were identified in the CARA list (U.S. EPA, 1991, 1994) and examined for relevant information were a Health Effects Assessment (U.S. EPA, 1984) and a Drinking Water Health Advisory (U.S. EPA, 1987), neither of which attempted an inhalation toxicity assessment. ATSDR (1989) declined to derive minimal risk levels for inhalation exposure to 1,1,2-trichloroethane. ACGIH (2001, 2003), NIOSH (2003), and OSHA (2003) all list TWA occupational exposure levels of 10 ppm (55 mg/m³) for 1,1,2-trichloroethane to protect against central nervous system depression, eye and upper respiratory tract irritation, and liver damage. The toxicity of 1,1,2-trichloroethane has been studied by NTP (2003) by oral exposure, and has been reviewed by IARC (1991, 1999). WHO (2003) does not have a toxicological review for this chemical. Literature searches were

conducted for the period from 1988 to July 2003 in the following databases: TOXLINE (including NTIS and BIOSIS updates), CANCERLIT, MEDLINE, CCRIS, GENETOX, HSDB, EMIC/EMICBACK, DART/ETICBACK, RTECS and TSCATS. Additional literature searches from July 2003 through October 2004 were conducted by NCEA-Cincinnati using MEDLINE, TOXLINE, Chemical and Biological Abstracts databases.

REVIEW OF PERTINENT LITERATURE

Human Studies

No relevant data were located regarding the toxicity of 1,1,2-trichloroethane to humans following inhalation exposure.

Animal Studies

On June 7, 2002 HAP Task Force of Millwood, VA presented to the EPA's Office of Pollution Prevention and Toxics a non-peer reviewed subchronic inhalation study on 1,1,2-Trichloroethane (1,1,2-TCE) conducted by WIL Laboratories, Ashland, OH. In this study 8 week old 10 male and 10 female Fischer 344 CDF (F-344) Crl:BR rats in each group were exposed to filtered air, 15, 40 and 100 ppm of 1,1,2-Trichloroethane through inhalation route for 13 weeks. Minimal hepatocellular vacuolation was observed in 15 and 40 ppm dosed groups. According to the author of the study the presence of minimal hepatocellular vacuolation, which might represent lipid accumulation, without centrilobular necrosis or an increase in serum levels of enzymes indicative of hepatocellular injury suggest that this degree of vacuolation lacks toxicological significance. Olfactory epithelium, including atrophy and respiratory epithelial metaplasia of the nasal turbinate were found in the 40 and 100 ppm dosed groups of animals. The authors of this study concluded that based on the results of the study for whole-body inhalation for 1,1,2-Trichloroethane to rats for 13 weeks, the NOEL was less than 15 ppm and NOAEL was less than 40 ppm. According to this study the hepatic and nasal effects observed in this study were not considered to be toxicologically significant. Olfactory epithelial atrophy and respiratory epithelial metaplasia are considered to be precancerous lesions. Such precancerous lesions are not adequate for developing an RfC.

In an unpublished study by Dow Chemical Company (briefly summarized by ACGIH, 2001; ATSDR, 1989; and U.S. EPA, 1984), unspecified numbers of male and female rats, guinea pigs, and rabbits were exposed to 1,1,2-trichloroethane vapors at a concentration of 15 ppm, 7 hours per day, 5 days per week for 6 months. No treatment-related adverse effects were noted regarding growth, mortality, organ weight, hematology, or clinical chemistry. Nor were there indications of treatment-related histopathologic changes. Sixteen 7-hour exposures of rats to a 1,1,2-trichloroethane vapor concentration of 30 ppm resulted in minor fatty changes and cloudy

swelling in the liver of female rats, but male rats appeared unaffected. The secondary accounts of these unpublished studies do not provide sufficient detail to provide a basis for an RfC for 1,1,2-trichloroethane.

A single male dog and 24 Sprague-Dawley rats (12/sex) were exposed to 1,1,2-trichloroethane at a target vapor concentration of 100 ppm (mean measured concentration of 84 ppm) 7 hours per day (on alternate days) for up to 6 months (Mellon Institute, 1947). Air-exposed animals (1 dog and 12 male and 12 female rats) served as controls. Endemic lung infection in the entire rat colony resulted in high mortality among treated and control rats (57 and 62%, respectively) during the study and rendered it unuseable for determining the toxicity of 1,1,2-trichloroethane. The treated dog exhibited a 13.2% decrease in body weight gain relative to the control dog, but no obvious treatment-related effects on hematology or clinical chemistry, and no pathological signs. Inclusion of only a single treated dog is an obvious limitation of this study.

DERIVATION OF A PROVISIONAL SUBCHRONIC AND CHRONIC RfC FOR 1,1,2-TRICHLOROETHANE

Due to the lack of adequate health effects data for either subchronic or chronic duration inhalation exposure, it is not feasible to derive a subchronic or chronic p-RfC for 1,1,2-trichloroethane.

REFERENCES

ACGIH (American Conference of Governmental Industrial Hygienists). 2001. Documentation for the Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices, 7th edition. Cincinnati, OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 2003. 2003 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH.

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6-11-2007

Provisional Peer Reviewed Toxicity Values for
1,2,4-Trimethylbenzene
(CASRN 95-63-6)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 1,2,4-TRIMETHYLBENZENE (CASRN 95-63-6)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Neither a reference dose (RfD) nor a reference concentration (RfC) are available for 1,2,4-trimethylbenzene in the Integrated Risk Information System (IRIS) database (U.S. EPA, 2007) or the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997). There is no Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile on 1,2,4-trimethylbenzene, other trimethylbenzene isomers, or mixtures of trimethylbenzene isomers (ATSDR, 2006). The Chemical Assessments and Related Activities (CARA) list (U.S. EPA, 1991, 1994a) and the HEAST (U.S. EPA, 1997) list a Health and Environmental Assessment (HEA) for trimethylbenzenes (U.S. EPA, 1987a); however, the available toxicity data were considered inadequate for quantitative risk assessment (U.S. EPA, 1997). The CARA (U.S. EPA, 1991, 1994a) lists a Drinking Water Health Advisory for 1,2,4-trimethylbenzene (U.S. EPA, 1987b). Because available human and animal toxicity data were considered inadequate for longer-term and lifetime quantitative risk assessment, the U.S. EPA (1987b) derived an RfD of 0.64 mg/kg-day for 1,2,4-trimethylbenzene based on assumptions that the Threshold Limit Value (TLV) of 25 ppm (125 mg/cu.m) for mixed trimethylbenzenes recommended by the American Conference of Governmental Industrial Hygienists (ACGIH, 2001, 2005) represents a NOAEL for 1,2,4-trimethylbenzene and that exposure results in 50% absorption. The National Institute of Occupational Safety and Health (NIOSH) adopted a recommended exposure limit (REL) time-weighted average (TWA) of 25 ppm (123 mg/m³) for 1,2,4-trimethylbenzene (NIOSH, 2006). The Occupational Safety and Health Administration (OSHA) has not adopted a permissible exposure limit (PEL) for 1,2,4-trimethylbenzene (OSHA, 2006). Health assessments for 1,2,4-trimethylbenzene are not available from other major sources, including CalEPA (2006), the

National Toxicology Program (NTP, 2006), the World Health Organization (WHO, 2006), and the International Agency for Research on Cancer (IARC, 2006).

A Group D (not classifiable as to human carcinogenicity) cancer classification is included in the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). A cancer assessment for 1,2,4-trimethylbenzene is not available on IRIS (U.S. EPA, 2007) or the HEAST (U.S. EPA, 1997). A cancer assessment for 1,2,4-trimethylbenzene is not available from CalEPA (2006), the National Toxicology Program (NTP, 2006), the World Health Organization (WHO, 2006), or the International Agency for Research on Cancer (IARC, 2006). Occupational exposure limits for 1,2,4-trimethylbenzene listed by NIOSH (2006) include no cancer notation.

Literature searches were performed to identify relevant information for 1,2,4-trimethylbenzene for the years 1986-1998 in the databases HSDB, RTECS, TSCATS, MEDLINE, and TOXLINE. Update literature searches were conducted in TOXLINE, MEDLINE (plus PubMed cancer subset), and DART/ETICBACK for the time period including January, 1998 to December, 2005. Update search of the TOXCENTER database was performed for the time period of August, 2000 to December, 2005. Databases searched without date limitations in December, 2005, included TSCATS, RTECS, GENETOX, HSDB and CCRIS. Search of Current Contents encompassed July to December, 2005.

REVIEW OF PERTINENT DATA

Human Studies

Oral Exposure

No data were located regarding the oral toxicity or carcinogenicity of 1,2,4-trimethylbenzene in humans.

Inhalation Exposure

Data regarding the inhalation toxicity of 1,2,4-trimethylbenzene in humans come from an occupational exposure study in which workers were exposed to a mixture of trimethylbenzene isomers. Bättig et al. (1958) examined 27 workers exposed to Fleet-X DV 99 solvent in the painting shop of a Swiss transportation plant. The solvent was analyzed spectrographically and was found to consist primarily of aromatic hydrocarbons (97.5%) and paraffinic and naphthenic hydrocarbons (2.5%). The aromatic hydrocarbon portion was composed of 1,2,4-trimethylbenzene (>50%), 1,3,5-trimethylbenzene (>30%), and possibly included 1,2,3-trimethylbenzene, 1-methyl-2-ethyl benzene, 1-methyl-3-ethyl benzene, and 1-methyl-4-ethyl benzene. Based on analysis of air samples collected from the plant, the concentration of the solvent was roughly estimated at 10-60 ppm (49-295 mg/m³). The control group consisted of 10 unskilled workers employed in a different section of the plant. Although the authors stated that the Fleet-X DV 99 solvent was used for "a period of some ten years," the average exposure duration of the workers was not reported. The workers reported CNS symptoms (vertigo, headaches and drowsiness) more often than the control group (70% versus 30% in the controls).

Chronic asthma-like bronchitis (30% of workers versus 10% of controls), anemia [defined as < 4.5 million erythrocytes/mm³ and usually combined with normal hemoglobin] (52% versus 20%) and alterations in blood clotting (30% versus 10%) were also observed in the exposed workers. The incidence of CNS symptoms was statistically significantly higher in the exposed workers than in the control group (Fisher's exact test conducted for this assessment; $p < 0.05$). For the other effects, the incidences did not significantly differ between the groups. A higher incidence of vitamin C deficiency was observed in the control group, suggesting that the two groups may not have been matched for socioeconomic status. If the assumption is made that the solvent exclusively contained trimethylbenzene isomers, then this study identifies a LOAEL in the range of 10-60 ppm (49-295 mg/m³) for signs of neurotoxicity.

Animal Studies

Oral Exposure

The database of repeated oral exposure studies in animals for 1,2,4-trimethylbenzene is limited to a 4-week study (Borrison Laboratories, 1984) and a chronic exposure carcinogenicity study (Maltoni et al., 1997). No oral developmental or reproductive toxicity studies were located for 1,2,4-trimethylbenzene.

The primary focus of the Borrison Laboratories (1984) study was the ability of 1,2,4-trimethylbenzene to induce nephrotoxicity. In this study, groups of 10 male Fischer-344 rats were administered doses of 0.5 or 2.0 g/kg neat 1,2,4-trimethylbenzene by gavage 5 days/week for 4 weeks; the duration-adjusted doses were 357 and 1429 mg/kg-day, respectively. A group of rats serving as controls were gavaged with saline. Gross necropsy was conducted in all rats, but only the kidneys underwent histopathologic examination. Mortality rates during treatment in the control, low-, and high-dose groups were 0/10, 1/10, and 10/10, respectively. Deaths in the high-dose group occurred as early as the third day of treatment. Final body weight and absolute kidney weight of low-dose rats were not significantly different than controls. Gross necropsy findings in low-dose animals included speckled cortical surfaces in the kidneys and white gelatinous material inside the urinary bladders. High-dose rats exhibited mottled and red thymus, spotty kidney and liver surfaces, enlarged adrenals, gas filled and yellow intestines and lung congestion. The presence or absence of hydrocarbon nephropathy was determined by examining the incidence of hyaline droplet changes, regenerative epithelium and tubular dilation with granular material. Treatment with 1,2,4-trimethylbenzene did not significantly increase the incidence or severity of nephropathy relative to controls; however, according to the authors, it is possible that high-dose rats died before nephropathy could develop. A NOAEL or LOAEL could not be determined due to the limited scope of the study, although the high dose of 1429 mg/kg-day was clearly a frank effects level (FEL) for mortality.

Maltoni et al. (1997) investigated the carcinogenicity of 1,2,4-trimethylbenzene (99% pure) in a long-term oral exposure experiment. Male and female Sprague-Dawley rats (50/sex/group) received doses of either 0 or 800 mg/kg (4 days/week) of 1,2,4-trimethylbenzene by gavage in 1 ml olive oil for 104 weeks. Food and water consumption and body weights were recorded throughout the experiment. Upon death or terminal sacrifice at 123 weeks, the animals were subjected to systemic necropsy. Histopathology was performed on brain, pituitary gland,

Zymbal glands, salivary glands, Harderian glands, head, tongue, thymus, mediastinal lymph nodes, lung, heart, diaphragm, liver, spleen, pancreas, kidneys, adrenal glands, esophagus, stomach, intestine (four levels), bladder, prostate, uterus, vagina, gonads, interscapular fat pad, subcutaneous and mesenteric lymph nodes, sternum, femur, spinal cord and any other organs and tissues with pathological lesions. No statistical analysis of the data was presented.

“Slight” reduction in the survival of the female Sprague-Dawley rats and an “intermediate” reduction in the survival of male rats were reported (Maltoni et al., 1997). However, quantitative survival data were not presented in the report and no statistical analysis of the decreases in survival were presented. Although the study report indicated that food and water consumption and body weight data were recorded, these data were not included in the report. There was no significant increase in the incidence of animals bearing either malignant or benign + malignant tumors (Table 1). Fisher’s exact tests conducted for EPA indicated that the differences in total tumors between the exposed and treated animals were not statistically significant ($p < 0.05$). Neuroesthesioepitheliomas were observed in the nasal cavity of 3/100 exposed animals (M + F). This tumor was not seen in concurrent controls, and a Fisher’s exact test of the data showed that the increase in incidence of neuroesthesioepitheliomas was not statistically significant ($p < 0.05$). The authors, however, indicated that these tumors are quite rare in the colony of Sprague-Dawley rats used for these experiments and suggested that this finding presents some evidence for carcinogenicity of 1,2,4-trimethylbenzene.

Table 1. Incidences of Benign and Malignant Tumors in Male and Female Sprague-Dawley Rats after a Long-term (104 week) Oral Exposure to 1,2,4-Trimethylbenzene.^a

Dose (mg/kg bw) ^b	Animals		Percent of animals with tumors	
	Sex	Number	Benign + Malignant	Malignant
800	M	50	62	26
	F	50	66	24
	M + F	100	64	25
0	M	50	54	24
	F	50	70	22
	M + F	100	62	23

^aSource: Maltoni et al., 1997

^bGavage dose administered 4 days/week for 104 weeks and animals were terminated after 123 weeks.

Inhalation Exposure

Korsak and Rydzyński (1996) examined the neurotoxic effects of acute exposure of male Wistar rats (10/group) to 1,2,4-trimethylbenzene (>97% pure) and other trimethylbenzene isomers, and also examined the neurotoxic effects of subchronic exposure to 1,2,4-trimethylbenzene and 1,2,3-trimethylbenzene. In the acute experiment, rats were exposed to concentrations of 250-2000 ppm (1227-9816 mg/m³) for 4 hours. Acute exposure to 1,2,4-trimethylbenzene caused concentration-related impairment in a rotarod performance test (EC₅₀ = 4693 mg/m³) and concentration-related decreased pain sensitivity (as measured by increased paw-lick response latency; EC₅₀ = 5682 mg/m³).

In the subchronic experiment, rats were exposed to 1,2,4-trimethylbenzene at concentrations of 0, 25, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³), 6 hours/day, 5 days/week for 3 months and observed for exposure-related clinical signs and body weight effects (Korsak and Rydzyński, 1996). Rotarod performance and hot-plate behavior were measured as indices of the neurotoxicity of trimethylbenzene isomers. Rotarod performance was tested prior to start of the study, weekly during exposure, and 2 weeks after the termination of the exposure. Hot-plate behavior was tested immediately after termination of the exposure. Fisher's exact test was used for analysis of rotarod performance and the Kruskal-Wallis test used for changes in pain sensitivity (hot plate behavior). Exposures to 1,2,4-trimethylbenzene did not result in any apparent body weight effects or clinical signs of toxicity. However, exposure-related indicators of neurotoxicity were noted. Rotarod performance failure increased in a concentration-related manner in the groups exposed to 1,2,4-trimethylbenzene, but reached the level of statistical significance (40% failure; p<0.05) only in the highest (1227 mg/m³) exposure group following 8 or 13 weeks of exposure. The incidence of rotarod performance failure in control rats was 0% throughout the study period. Although the mean rotarod performance failure rate in the highest exposure group remained at 30% after a 2-week recovery period, the rate was not significantly different from controls. Pain-sensitivity was also decreased in a concentration dependent manner (evidenced by increased latency of the paw-lick response). As shown in Table 2, the increased latency reached the level of statistical significance in the 491- and 1227-mg/m³ groups. After a 2-week recovery period, the highest (1227 mg/m³) exposure group no longer exhibited a significant difference in pain sensitivity, relative to controls. This study identified a NOAEL of 123 mg/m³ and a LOAEL of 491 mg/m³ (6 hours/day, 5 days/week) for significantly decreased pain sensitivity.

Table 2. Exposure-Related Effect on Latency of the Paw-Lick Response in Rats Exposed to 1,2,4-Trimethylbenzene Vapors 6 Hours/Day, 5 Days/Week for 3 Months.^a

Number of rats	Exposure level (mg/m ³)	Mean latency of paw-lick response (seconds)
9	0	15.4 ± 5.8 ^b
10	123	18.2 ± 5.7
9	491	27.6 ± 3.2 ^c
10	1227	30.1 ± 7.9 ^c

^a Source: Korsak and Rydzyński, 1996

^b The authors did not specify whether standard deviation or standard error of the mean is presented

^c Statistically significantly different from controls (p≤0.01)

Gralewicz et al. (1997a) investigated 1,2,4-trimethylbenzene-induced behavioral effects on groups of male Wistar rats (15/group) exposed to vapor concentrations of 0, 50, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³) for 6 hours/day, 5 days/week for 4 weeks. To assess the effect of exposure on short-term working memory, choice accuracy in a radial arm maze was tested. Effects on spontaneous activity were evaluated with an open field test. Effects on long-term memory and learning ability were assessed on the basis of conditioned passive and active avoidance tests. The hot-plate test was performed to compare the groups with respect to the decrease in responsiveness to a thermal stimulus following a brief intermittent foot shock. Animals were subjected to the following sequence of behavioral testing:

1. radial maze: 2 weeks before exposure and on days 14-18 after exposure,
2. open field activity: day 25 after exposure,
3. passive avoidance: days 35-45 after exposure,
4. hot-plate test: days 50 and 51 after exposure,
5. active avoidance: day 54 after exposure.

The data were analyzed by ANOVA and comparisons among treatments were made using Sheffe's test, or Tukey's test for 2-way ANOVA.

There was no significant effect of 1,2,4-trimethylbenzene exposure on body weight gain during the 4-week exposure. Passive-avoidance learning was significantly ($p < 0.001$) retarded in groups exposed to 491 or 1227 mg/m³ of 1,2,4-trimethylbenzene and tested 35-45 days after the end of the exposure period. Retardation of passive-avoidance learning was more pronounced in the 491 mg/m³ exposure group than in the 1227 mg/m³ group. In the hot-plate test following foot shock, evaluation of rats 50 days following termination of exposures to 491 or 1227 mg/m³ of 1,2,4-trimethylbenzene revealed significantly ($p < 0.01$) increased paw-lick latency times, in comparison to unexposed controls. There was no significant change in the active avoidance test, although there was a trend toward decreased avoidance responses with increasing 1,2,4-trimethylbenzene exposure concentration. Short-term working memory did not appear to be adversely affected by 1,2,4-trimethylbenzene exposure. In the open field test there was no significant effect on spontaneous movement or on rearing behavior; however, there was a significant ($p < 0.05$) increase in grooming behavior of animals exposed to 1,2,4-trimethylbenzene at 491 mg/m³. Although grooming behavior also was increased above controls in the 123 and 1227 mg/m³ groups, the difference was not statistically significant. The results of these experiments suggest that 4-week exposures at concentrations that produced no overt clinical signs of toxicity can produce long-term effects on the functional state of the rat central nervous system. Based on findings of significantly retarded passive avoidance learning and increased paw-lick latency in rats of the 491 and 1227 mg/m³ exposure groups, the 123 mg/m³ group represented a NOAEL and the 491 mg/m³ group represented a LOAEL (6 hours/day, 5 days/week) for persistent neurotoxic effects.

Gralewicz and Wiaderna (2001) employed the same general protocol used by Gralewicz et al. (1997a) in a comparative study of the behavioral effects of repeated inhalation exposure to individual trimethylbenzene isomers or *m*-xylene. The study included a group of 11 adult male Wistar rats exposed to 100 ppm (491 mg/m³) of 1,2,4-trimethylbenzene (purity not stated) and a control (air only) group of 10 male rats. Exposures were for 6 hours/day, 5 days/week for 4

weeks. The sequence of behavioral testing varied slightly from that employed by Gralewicz et al. (1997a) and included:

1. radial maze: 1 week before exposure and on days 14-18 after exposure,
2. open field activity: day 8 before exposure and day 25 after exposure,
3. passive avoidance: days 39-48 after exposure,
4. hot-plate test: days 50 and 51 after exposure,
5. active avoidance: days 54 and 60 after exposure.

No significant exposure-related effects were seen regarding body weights or short-term working memory (as determined in the radial arm maze test) for any of the trimethylbenzene isomers or *m*-xylene. Acquisition, but not retention, of the two-way active avoidance response was significantly impaired in all solvent-exposed groups. Results of other behavioral tests demonstrated exposure-related effects for each of the solvents. In the case of 1,2,4-trimethylbenzene, significantly increased spontaneous locomotor activity in the open field, impaired passive avoidance learning and significantly longer paw-lick latencies in the hot-plate test 24 hours after foot shock were observed. These results support the findings of the earlier study (Gralewicz et al., 1997a) in which the 491 mg/m³ (6 hours/day, 5 days/week) exposure level represented a LOAEL for neurotoxic effects in 1,2,4-trimethylbenzene-exposed rats.

Gralewicz et al. (1997b) investigated the effect of a 4-week (6 hours/day, 5 days /week) inhalation exposure to 1,2,4-trimethylbenzene (purity not stated) at concentrations of 0, 25, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³) on the occurrence of spike-wave discharges (SWD) in the neurocortex of male Wistar rats (9-10/group). Bursts of SWD increase in number and/or duration with advancing age and it was hypothesized that exposure to neurotoxic solvents may accelerate the aging process in the brain. Electrodes were implanted into the fronto-parietal cortex and into the dorsal hippocampus. One-hour EEG recordings were performed immediately before initiation of exposure, at the end of the exposure period, 1 month later and 3 months later. The occurrence of SWD bursts is limited to the state of awake immobility. The number and total duration of SWD bursts were determined from each EEG. The data were analyzed by ANOVA and multiple comparisons among treatments was performed with Tukey's test. The study results included information regarding mean body weights, but the study report did not provide details of body weight data collection.

The study authors (Gralewicz et al., 1997b) indicated that 1,2,4-trimethylbenzene exposure resulted in no statistically significant body weight effects. In the control and lowest (123 mg/m³) exposure groups, the total duration of SWD showed an increasing trend with time, in comparison to pre-exposure SWD and reached statistical significance ($p < 0.05$) at 3 months after exposure. In contrast, the total duration of SWD tended to decline with time in the mid- and high-exposure groups after exposure. The decrease in SWD occurrence, however, was statistically significant only for the measurements performed 1 month after the end of exposure in the mid-exposure (491 mg/m³) group. A similar trend was seen when the number of SWD bursts per hour was determined. The frequency of SWD bursts increased with age in the control and lowest exposure groups and tended to decline with time in the mid- and high-exposure groups. The data, however, were highly variable and were statistically significantly different from pre-exposure SWD only for the highest exposure level at 3 months after exposure. Thus,

there were no clear 1,2,4-trimethylbenzene induced concentration-related effects on SWD, although the results are suggestive that long-term effects on brain activity may have occurred.

Korsak et al. (2000) exposed groups of male and female rats (10/sex/group; 20/sex/group at the highest exposure concentration) of outbred Imp:WIST to 1,2,4-trimethylbenzene (97% pure) vapors at target concentrations of 0, 123, 492 or 1230 mg/m³ for 6 hours/day, 5 days/week for 3 months. Animals were observed twice daily for clinical signs of toxicity. Body weights were recorded prior to initiation of exposures and weekly thereafter and food consumption was measured weekly. Blood was drawn for hematological examination prior to initiation of exposures and 1 week prior to exposure termination. Clinical chemistry testing was performed at the end of the 3-month exposure period. Organ weights were determined for lungs, liver, spleen, kidneys, adrenals, heart and gonads. Histopathological examinations were performed on tissues from brain, nose, larynx, trachea, thymus, lungs, heart, liver, spleen, kidney, adrenals, thyroid, pancreas, gonads, urinary bladder, stomach, duodenum, small and large intestines and salivary glands.

Clinical findings were unremarkable (Korsak et al., 2000). No significant exposure-related effects were seen regarding food consumption or body weights. The few differences in some relative or absolute organ weights did not appear to be of toxicological relevance. Significant concentration-related trends ($p < 0.01$) for decreased numbers of red blood cells and increased numbers of white blood cells were noted for male (but not female) rats. In the male rats of the highest 1,2,4-trimethylbenzene exposure group (1230 mg/m³), both red and white blood cell counts were significantly different ($p < 0.01$) from those of control males. A significant trend ($p < 0.01$) for concentration-related decreased reticulocyte counts was observed in female rats and the difference was significant ($p < 0.05$) in the 1230 mg/m³ group. Hematological testing also revealed a significant trend ($p < 0.01$) for decreased clotting time in female (but not male) rats; the decrease reached the level of statistical significance ($p < 0.05$) in the 492 and 1230 mg/m³ groups. Clinical chemistry results were unremarkable, with the exception of significantly increased serum sorbitol dehydrogenase in all 1,2,4-trimethylbenzene-exposed groups of male rats (not concentration related). Histopathological examinations revealed exposure-related significantly increased severity of pulmonary lesions, which included increased proliferation of peribronchial lymphatic tissue in males of the mid- (but not highest) exposure level, increased alveolar macrophages in males of the highest exposure level and increases in interstitial lymphocytic infiltrations in males of the mid- (but not highest) exposure level and females of the highest exposure level. No significant exposure-related changes were seen in the other examined organs and tissues. The mid exposure level of 492 mg/m³ can be considered a LOAEL for hematological and respiratory effects in this study. The low exposure level of 123 mg/m³ is a NOAEL.

Korsak et al. (1997) exposed male Wistar rats of IMP:DAK outbred stock (10/group) to 1,2,4-trimethylbenzene ($\geq 97\%$ pure) at concentrations of 0, 25, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³) for 90 days (6 hours/day, 5 days/week). Lung lavage fluid was collected 24 hours after termination of the subchronic exposure and centrifuged at 400 g for 10 minutes. Differential counts of bronchoalveolar lavage (BAL) cell smears were determined by light microscopy after staining and the trypan blue test was used to determine cell viability. Total

protein concentration, mucoprotein concentration, lactate dehydrogenase and acid phosphatase activity were determined in the BAL supernatant.

All rats exposed to 1,2,4-trimethylbenzene for 90 days survived the experiment and there were no significant differences in final body weight. Statistically significant increases were observed in total cell and macrophage numbers in BAL of all treated groups after 90 days in comparison to controls. Significant increases were also observed in total protein, lactate dehydrogenase (LDH) and acid phosphatase (AP) in BAL fluid of all treated groups. However, the observed increases in these parameters were either at or near their highest observed response at the lowest exposure concentration, and there was no indication of further concentration-related increases. For the observed effects, the lowest exposure level used (123 mg/m^3) would be a LOAEL; however, the toxicological significance of these effects is not clear.

In a study by IBT (1981), groups of 5 male and 5 female COBS rats were exposed to 49 or 480 mg/m^3 MCS-1809 6 hours/day, 5 days/week for 4 weeks (IBT, 1981). MCS-1809 was identified as a compound containing 75% 1,2,4-trimethylbenzene and 25% C9 aromatics (Monsanto, 1992). The test atmosphere was generated by passing the MCS-1809 through a nebulizer; no information on the particle size distribution was reported. Based on the vapor pressure of 1,2,4-trimethylbenzene, it is likely that the animals were predominantly exposed to 1,2,4-trimethylbenzene vapors rather than a mist. The following parameters were used to assess toxicity: daily observations, weekly body-weight measurements, organ weights (adrenal glands, brain, gonads, heart, kidneys, liver, lungs, spleen and thyroid gland), gross necropsy and histopathological examination of adrenal glands, brain, bronchi, gonads, heart, kidneys, liver, lungs, pancreas, pituitary glands, lymph nodes, spleen, trachea and thyroid gland of the control and 480 mg/m^3 groups (tissues from the 49 mg/m^3 group were examined if significant findings were found in the 480 mg/m^3 group).

Exposure to MCS-1809 did not result in deaths. Clinical signs of toxicity in the 480 mg/m^3 group included ataxia and hypoactivity that persisted between exposures, ptosis, red ocular discharge, and ruffed fur. Less pronounced hypoactivity and ruffed fur were observed in the 49 mg/m^3 group. In the 480 mg/m^3 group, significant decreases in body weight gain (35% lower in the males; no significant alteration in females), increases in absolute (females only) and relative liver weights and decreases in absolute and relative spleen weights (females only) were observed. A significant increase in absolute liver weight was also observed in the 49 mg/m^3 female rats. Histological alterations were limited to focal or diffuse testicular atrophy in 3/5 male rats exposed to 480 mg/m^3 in the absence of statistically significant changes in testis weight; no testicular effects were observed in the 49 mg/m^3 (testes examined in four rats from this group) or control groups. This study identified a NOAEL of 49 mg/m^3 and LOAEL of 480 mg/m^3 (6 hours/day, 5 days/week) for clinical signs of toxicity (persistent ataxia and hypoactivity, ptosis, ocular discharge), decreased body weight gain, and histopathological evidence of testicular atrophy. The increased absolute liver weight observed in the 49 mg/m^3 female rats was not considered adverse because no histological alterations were observed at the 49 or 480 mg/m^3 concentrations.

Bättig et al. (1958) exposed 8 male rats (strain not reported) to air concentrations of 1700 ppm of Fleet-X DV 99 solvent for 4 months (8 hours/day, 5 days/week). Other rats (sex, strain,

and number not specified) were exposed to 500 ppm for 70 days (8 hours/day, 5 days/week). As described earlier, Fleet-X DV 99 is a solvent containing 97.5% aromatic hydrocarbons (>50% 1,2,4-trimethylbenzene and >30% 1,3,5-trimethylbenzene) and 2.5% of paraffinic and naphthenic hydrocarbons. The 500- and 1700-ppm concentrations would be approximately 2523 and 8155 mg/m³, respectively, if the aromatic hydrocarbon fraction of the vapors were comprised exclusively of trimethylbenzenes. Within 2 weeks of exposure, 4 of the 8 rats exposed to 1700 ppm died and were replaced, while none of the animals in the 500-ppm group died. Histopathologic examinations, performed only on 1700-ppm animals that died, revealed cloudy swelling and fatty infiltration in the kidneys, peripheral fatty infiltration in the liver, an increase in secondary nodules in the spleen and marked congestion of the pulmonary capillaries with alveolar wall thickening. Alterations in differential white blood cell counts (increase in the percentage of segmented neutrophilic granulocytes and a decrease in the percentage of lymphocytes) were reported at 500 ppm. Increases in drinking water consumption (43-45% higher than in the control group) were observed in the 1700-ppm group. The authors reported that during the exposure period, the 1700-ppm animals were initially “highly excited and aggressive” followed by a period of narcosis and ataxia. Because histopathology was only performed on the animals that died, no histopathology data are available on the 500-ppm rats. Due to the limited scope of the study, a NOAEL or LOAEL cannot be identified. The high concentration of 1700 ppm (8155 mg/m³) is a FEL for mortality.

Bernshtein (1972) exposed rats (number, sex and strain not specified) to 1000 mg/m³ (200 ppm) of a mixture of trimethylbenzenes for 6 months (4 hours/day, 6 days/week). An inhibition of phagocytic activity of the leukocytes was reported. This study was summarized by Sandmeyer (1981) and further experimental details were not provided.

Korsak et al. (1997) examined the effect of acute exposures (6 min) to the trimethylbenzene isomers, 1,2,3-trimethylbenzene (90-95% pure), 1,3,5-trimethylbenzene (99% pure) and 1,2,4-trimethylbenzene (97% pure) on the respiratory rate of Balb/C male mice (8-10/group) at concentrations ranging from 253 to 1591 ppm (1926-9453 mg/m³). The concentration depressing mouse respiratory rate by 50% (RD₅₀) was calculated by least squares regression and the Kruskal-Wallis test was applied for evaluation of protein and enzyme levels in the BAL fluid. All three trimethylbenzene isomers showed irritating effects on the respiratory tract and caused concentration-dependent decreases in respiratory rate. The concentration depressing the respiratory rate in mice to 50% was 519 ppm (2547 mg/m³) for 1,2,4-trimethylbenzene.

The developmental toxicity of 1,2,4-trimethylbenzene was assessed by Saillenfait et al. (2005). Groups of mated (sperm-positive) Sprague-Dawley rats (24/group) were exposed (whole body) to 1,2,4-trimethylbenzene (97% pure) at vapor concentrations of 0, 100, 300, 600 or 900 ppm (0, 491, 1475, 2950 or 4425 mg/m³) for 6 hours/day on gestation days 6 through 20. Maternal food consumption was recorded for the intervals of gestation days 6-13 and 13-21. Maternal body weights were recorded weekly during gestation. At necropsy on gestation day 21, the uterus was weighed and numbers of corpora lutea, implantation sites, resorptions and dead and live fetuses were recorded. Live fetuses were weighed, sexed and examined for external anomalies. Half of the live fetuses from each litter were prepared for visceral examination, the others were subjected to skeletal examination.

All dams survived to scheduled necropsy (Saillenfait et al., 2005). No clinical signs of 1,2,4-trimethylbenzene-induced toxicity were observed. Maternal food consumption was significantly ($p < 0.01$) depressed in the 600- and 900-ppm groups (approximately 12-14% and 15-19%, respectively, relative to controls). The 900-ppm dams exhibited significantly reduced mean body weight gain (22-52% lower than controls) throughout the exposure period. Significantly ($p < 0.01$) reduced body weight gain (30% lower than controls) was observed in the 600-ppm group, but only during the first week of 1,2,4-trimethylbenzene exposure. At necropsy on gestation day 21, mean body weight gain (corrected for gravid uterine weight) was significantly depressed in both 600- and 900-ppm dams (approximately 50% lower than controls). Mean fetal body weight was significantly lower in both 600- and 900-ppm exposure groups (approximately 5 and 11% lower, respectively, than controls). There were no other significant indications of maternal or fetal toxicity. This study identified a NOAEL of 300 ppm (1475 mg/m^3) and a LOAEL of 600 ppm (2950 mg/m^3 , 6 hours/day on gestation days 6 through 20) for maternal and fetal body weight effects. However, the observed fetal toxicity was likely secondary to maternal toxicity because the decreased fetal body weight was noted only at exposure levels resulting in significantly depressed maternal body weight gain.

Other Studies

Limited genotoxicity data suggest that 1,2,4-trimethylbenzene is not mutagenic. 1,2,4-trimethylbenzene produced negative results in the Ames test with *Salmonella typhimurium* strains TA97a, TA98, TA100 and TA102 both in the presence and absence of rat liver S9 metabolic activation (Janik-Spiechowicz et al., 1998). 1,2,4-Trimethylbenzene was not cytogenic in the mouse micronucleus test, but elicited a positive response in sister chromatid exchange (SCE) tests with bone marrow cells of Imp:Balb/c mice treated *in vivo* (Janik-Spiechowicz et al., 1998).

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfDs FOR 1,2,4-TRIMETHYLBENZENE

Limited information is available regarding the oral toxicity of 1,2,4-trimethylbenzene in humans and animals. The nephrotoxicity study by Borriston Laboratories (1984) is too limited in scope to be used to identify a NOAEL or LOAEL for 1,2,4-trimethylbenzene, although the 2000 mg/kg dose (1429 mg/kg-day) is clearly a FEL for increased mortality. The study of Maltoni et al. (1997) is also unsuitable for derivation of an RfD, as only one dose level was employed, decreased survival occurred at this dose level and reporting of the results was inadequate. Thus, the database for 1,2,4-trimethylbenzene is inadequate to derive a provisional RfD.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfCs FOR 1,2,4-TRIMETHYLBENZENE

Several studies have examined the inhalation toxicity of mixtures predominantly containing one or more trimethylbenzene isomers, or pure 1,2,4-trimethylbenzene. Significant

increases in the incidence of CNS toxicity (vertigo, dizziness, headaches) and non-significant increases in the incidences of respiratory effects (bronchitis) and hematological effects (hyperchromic anemia and blood clotting alterations) have been observed in workers exposed to 10-60 ppm (49-295 mg/m³) of a solvent containing >80% trimethylbenzene isomers (Bättig et al., 1958). Many of these effects reported in humans have been observed in experimental animals repeatedly exposed to 1,2,4-trimethylbenzene or other trimethylbenzene isomers or trimethylbenzene mixtures. For example, hematological effects have been reported in experimental animals exposed to a trimethylbenzene mixture (Bernshtein, 1972) or 1,2,4-trimethylbenzene (Korsak et al., 2000). Signs of adverse CNS effects have been observed in animals exposed to mixtures containing 1,2,4-trimethylbenzene (Bättig et al., 1958; IBT, 1981) or 1,2,4-trimethylbenzene alone (Gralewicz and Wiaderna, 2001; Gralewicz et al., 1997a, 1997b; Korsak and Rydzyński, 1996). Results of other animal studies provide evidence of 1,2,4-trimethylbenzene-induced respiratory effects (Korsak et al., 1997, 2000). Other effects observed in animal studies include testicular atrophy in rats exposed to 480 mg/m³ (98 ppm) of a mixture containing 75% 1,2,4-trimethylbenzene for 4 weeks (6 hours/day, 5 days/week) (IBT, 1981) and lung, liver, kidney and spleen effects in rats exposed to 1700 ppm (8155 mg/m³) of a solvent containing >80% trimethylbenzene isomers for 4 months (8 hours/day, 5 days/week) (Bättig et al., 1958).

The lowest estimated level of occupational exposure to the solvent Fleet-X DV 99 (>80% 1,2,4- and 1,3,5-trimethylbenzene) in the study of Bättig et al. (1958) was 10 ppm (49 mg/m³). Assuming that the solvent exclusively contained trimethylbenzene isomers, the 49 mg/m³ exposure concentration can be considered to represent a LOAEL. Although the Bättig et al. (1958) report provides the lowest inhalation LOAEL (49 mg/m³) of any study, it may be an inappropriate study for consideration as the principal study for a number of reasons. Importantly, Bättig et al. (1958) identified spectrophotographically the presence of various aromatic hydrocarbons, to include naphthenic and paraffenic compounds, in addition to 1,2,4-trimethylbenzene in the solvent mixture. While 1,2,4-trimethylbenzene comprised up to 50% of the Fleet-X DV 99 mixture, it is virtually impossible to confidently attribute human toxicities solely to 1,2,4-trimethylbenzene (i.e. the study LOAEL is for the mixture not the individual compound). Additional concerns that warrant exclusion of the Bättig et al. (1958) human study from consideration include inappropriate selection of a human control population [e.g. nutritional status (Vit. C deficient)], and the fact that average Fleet-X DV 99 solvent exposure duration, for the 27 exposed workers examined, was not reported.

An advantage of some of the animal models of 1,2,4-trimethylbenzene inhalation exposure over the Bättig et al. (1958) human study is that controlled atmospheres involved the compound of interest at relatively high purities (e.g. 97% 1,2,4-trimethylbenzene in the Korsak et al., 2000 study). However, available repeated exposure inhalation studies in animals are limited to subchronic exposure duration (4 weeks to 3 months) in which the lowest identified LOAEL for 1,2,4-trimethylbenzene was 491 mg/m³ (Gralewicz and Wiaderna, 2001; Gralewicz et al., 1997a; Korsak and Rydzyński, 1996); furthermore, many of the effects observed in these rodent studies are of unclear toxicological significance and/or have concentration-responses that are difficult to interpret.

Provisional RfCs may be derived based on adverse pulmonary or hematological effects reported in male or female rats, respectively, exposed to 1,2,4-trimethylbenzene (97% pure) for 3 months (Korsak et al., 2000). The selection of the Korsak et al. (2000) study as the basis for deriving RfCs is supported by previous observations in rats (Korsak et al., 1997) and humans (Bättig et al., 1958) exposed to pure 1,2,4-trimethylbenzene or a mixture of trimethylbenzenes, respectively, for ≥ 90 days. Indeed, pulmonary lesions and hematological abnormalities in rats exposed to pure 1,2,4-trimethylbenzene for 3 months (Korsak et al., 2000) are consistent with observations in humans following presumably longer duration exposure to a mixture containing 1,2,4-trimethylbenzene (Bättig et al., 1958).

Subchronic p-RfC

The subchronic p-RfC for 1,2,4-trimethylbenzene is derived from the NOAEL of 25 ppm (123 mg/m^3) identified in the Korsak et al. (2000) rat subchronic inhalation study. Two different toxic effects (pulmonary or hematological) were identified in male or female rats, respectively, in this study at the same LOAEL/NOAEL. As such, two separate subchronic p-RfC derivations are presented below to identify the most sensitive endpoint. Under an assumption of category 3 for decreased clotting time in female Imp:WIST rats, an adjusted experimental NOAEL can be derived using the NOAEL of 123 mg/m^3 and the exposure duration data from Korsak et al. (2000) as follows:

$$\begin{aligned}\text{NOAEL}_{[\text{ADJ}]} (\text{mg/m}^3) &= \text{rat NOAEL} (\text{mg/m}^3) \times 6\text{hr}/24\text{hr} \times 5 \text{ days}/7 \text{ days} \\ &= 123 \text{ mg/m}^3 \times 0.25 \times 0.71 \\ &= 21.8 \text{ mg/m}^3\end{aligned}$$

According to equation (4-48) for extrapulmonary effects [Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994)], a human equivalent concentration ($\text{NOAEL}_{[\text{HEC}]}$) can be calculated as follows:

$$\text{NOAEL}_{[\text{HEC}]} (\text{mg/m}^3) = \text{NOAEL}_{[\text{ADJ}]} (\text{mg/m}^3) \times (\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$$

*blood:gas (b/g) partition coefficients for 1,2,4-trimethylbenzene could not be located, therefore a default value of 1 is used for the term $(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$. The human $\text{NOAEL}_{[\text{HEC}]}$ is equivalent to the duration adjusted rat NOAEL of 21.8 mg/m^3 . A **subchronic p-RfC of $7\text{E}-2 \text{ mg/m}^3$** based on a hematological effect is derived by dividing the $\text{NOAEL}_{[\text{HEC}]}$ of 21.8 mg/m^3 by a composite UF of 300, as follows:

$$\begin{aligned}\text{UF (animal to human)} &= 3 \\ \text{UF (interindividual variability)} &= 10 \\ \text{UF (database deficiencies)} &= 10\end{aligned}$$

$$\begin{aligned}\text{Subchronic p-RfC} &= \text{NOAEL}_{[\text{HEC}]} / \text{UF} \\ &= 21.8 \text{ mg/m}^3 / 300 \\ &= 0.07 \text{ mg/m}^3 \text{ or } 7\text{E}-2 \text{ mg/m}^3\end{aligned}$$

Under an assumption of category 1 for pulmonary toxicity in male rats, the same duration adjusted rat NOAEL of 21.8 mg/m³ is obtained as shown above. Histopathological observations in lung tissue of male Imp:WIST rats exposed to 1,2,4-trimethylbenzene for 3 months indicated inflammatory lesions primarily in the bronchiolar region. Therefore, according to equation 4-22 for Category 1 tracheobronchial effects [Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994)], a NOAEL_[HEC] can be calculated as follows:

$$\begin{aligned}\text{NOAEL}_{[\text{HEC}]} (\text{mg}/\text{m}^3) &= \text{NOAEL}_{[\text{ADJ}]} (\text{mg}/\text{m}^3) \times \text{RGDR}_{\text{TB}}^{\dagger} \\ &= 21.8 \text{ mg}/\text{m}^3 \times 1.6 \\ &= 34.9 \text{ mg}/\text{m}^3\end{aligned}$$

[†] Derivation of the RGDR_{TB} can be found in Appendix 1

A **subchronic p-RfC of 1E-1 mg/m³** based on pulmonary effects is derived by dividing the NOAEL_[HEC] of 34.9 mg/m³ by the same composite UF of 300 identified above:

$$\begin{aligned}\text{Subchronic p-RfC} &= \text{NOAEL}_{[\text{HEC}]} / \text{UF} \\ &= 34.9 \text{ mg}/\text{m}^3 / 300 \\ &= 0.1 \text{ mg}/\text{m}^3 \text{ or } 1\text{E}-1 \text{ mg}/\text{m}^3\end{aligned}$$

The composite UF includes a factor of 3 for extrapolation from animal to human, 10 for interindividual variability, and 10 for database deficiencies. The reduced uncertainty of 3 for animal to human extrapolation is due in part to both the conversion of the rat NOAEL to a human equivalent concentration as well as the consistency of hematological and pulmonary toxicity between rats and humans exposed to 1,2,4-trimethylbenzene. The database deficiencies include lack of developmental toxicity studies in a second species, multigeneration reproductive toxicity studies, and a lack of confidence in the large majority of available animal studies reporting effects of undetermined toxicological significance with concentration-responses that are difficult to interpret. The derivations shown above clearly indicate that decreased clotting time in female rats due to subchronic inhalation exposure to 1,2,4-trimethylbenzene is the more sensitive or health protective endpoint under consideration.

Chronic p-RfC

The **chronic p-RfC of 7E-3 mg/m³** based on decreased clotting time in female rats (Korsak et al., 2000) is derived by dividing the NOAEL_[HEC] of 21.8 mg/m³ by a composite UF of 3000, as follows:

$$\begin{aligned}\text{Chronic p-RfC} &= \text{NOAEL}_{[\text{HEC}]} / \text{UF} \\ &= 21.8 \text{ mg}/\text{m}^3 / 3000 \\ &= 0.007 \text{ or } 7\text{E}-3 \text{ mg}/\text{m}^3\end{aligned}$$

As for the chronic RfC, the composite UF includes a factor of 10 for extrapolation from subchronic to chronic exposure, 3 for extrapolation from animal to human, 10 for interindividual variability, and 10 for database deficiencies.

Confidence in the principal study (Korsak et al., 2000) is low. While it is remarkable that hematological and pulmonary effects are apparently conserved from rats (Korsak et al., 2000) to humans (Bättig et al., 1958), the concentration-response for either compartment in rats (particularly male rats) is difficult to interpret. Specifically, the low inhalation concentration (123 mg/m^3) in female rats from the Korsak et al. (2000) study was clearly a NOAEL for decreased clotting time (hematological compartment); this NOAEL was also identified for pulmonary effects (e.g. proliferation of peribronchial lymphatic tissue, interstitial lymphocytic infiltration of parenchyma, bronchitis and bronchopneumonia) in male rats. Interestingly, female rats seemed slightly more resistant to these pulmonary effects. However, the overall commutative score, following statistical trend analysis, of all pulmonary lesions suggested that the lungs of male and female Imp:WIST rats are significantly affected by inhalation exposure to 1,2,4-trimethylbenzene at the mid dose of 492 mg/m^3 (Korsak et al., 2000). However, paradoxically, in male rats of the high dose group the pulmonary effects decreased compared to animals in the mid dose group. This counter-intuitive concentration-response relationship might suggest a concentration-dependent transition in mode of action for pulmonary toxicity (note the increase in absolute lung weight of male Imp:WIST rats at the mid concentration of 492 mg/m^3 , which is the concentration at which inflammatory foci were identified in lung tissue, and yet in the high concentration group lung weight decreased back to control levels (Korsak et al., 2000); more work would be required to verify.).

According to the derivations provided above it appears that the hematological endpoint (i.e. decreased clotting time in female rats) may be a more appropriate endpoint to consider for inhalation exposure to 1,2,4-trimethylbenzene. Further work in this area is certainly warranted. The human occupational report from Bättig et al. (1958) identified a lower inhalation effect level (e.g. LOAEL = 49 mg/m^3) compared to any of the available animal data. However, the utility of this study in derivation of RfCs is limited by poor reporting of results, undetermined exposure levels, the lack of statistical analysis of results, the lack of information on the exposed and control groups (e.g., age, education level, length of employment), small group sizes and possibly a poorly matched control group (as evidenced by increased incidence of vitamin C deficiency in controls). Also, the controls worked in adjacent rooms and the possibility that they also may have been exposed to trimethylbenzene cannot be excluded. Confidence in the database is low because the database is lacking developmental toxicity data in a second species and reproductive toxicity studies. Reflecting low confidence in the principal study and database, confidence in the provisional subchronic and chronic RfC values is low.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 1,2,4-TRIMETHYLBENZENE

Weight-of-evidence Classification

No information was located regarding the carcinogenicity of 1,2,4-trimethylbenzene or mixtures of trimethylbenzene isomers in humans. The database of information regarding the carcinogenicity of trimethylbenzene in animals is limited to a single carcinogenicity study in which male and female Sprague-Dawley rats (50/sex/group) were administered 1,2,4-trimethylbenzene via oral gavage at doses of 0 or 800 mg/kg for 104 weeks (Maltoni et al.,

1997). Although quantitative survival data were not included in the study report, the authors noted “intermediate” and “slight” reduction in the survival of 1,2,4-trimethylbenzene treated male and female rats, respectively. Under the conditions of the study, oral exposure to 1,2,4-trimethylbenzene did not cause a statistically significant increase in the incidence of animals bearing either malignant tumors or benign and malignant tumors (combined) or in the incidence of neuroesthesioepitheliomas. The study of Maltoni et al. (1997) included a single animal species (rat) and a single 1,2,4-trimethylbenzene dose level (800 mg/kg). Based on limitations in study design and reporting of results and the lack of additional carcinogenicity data in animals, the database of information for 1,2,4-trimethylbenzene is inadequate to establish the potential carcinogenicity of 1,2,4-trimethylbenzene. Limited genotoxicity data demonstrated that 1,2,4-trimethylbenzene was not mutagenic in several strains of *Salmonella typhimurium*, and did not elicit cytogenicity in the mouse micronucleus test, but did elicit a positive response in sister chromatid exchange (SCE) tests with bone marrow cells of Imp:Balb/c mice treated *in vivo* (Janik-Spiechowicz et al., 1998). These data provide inadequate evidence for genotoxic activity.

Collectively, the available carcinogenicity and genotoxicity data do not adequately assess the carcinogenic potential of 1,2,4-trimethylbenzene in humans or animals. Under the current U.S. EPA (2005) cancer guidelines, the human and animal data are inadequate for a determination of the human carcinogenic potential of 1,2,4-trimethylbenzene.

Quantitative Estimates of Carcinogenic Risk

There are no appropriate human or animal data from which to derive an oral slope factor or inhalation unit risk for 1,2,4-trimethylbenzene.

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APPENDIX 1

According to equation 4-22 for Category 1 tracheobronchial effects [Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994)], a $NOAEL_{[HEC]}$ is calculated using the duration adjusted animal NOAEL and a dosimetric adjustment factor (DAF). In this case the DAF is the RGDR for the tracheobronchial region of the lung ($RGDR_{TB}$). The $RGDR_{TB}$ is calculated as follows:

$$RGDR_{TB} = (RGD_{TB})_A / (RGD_{TB})_H = \frac{(V_E/SA_{TB})_A}{(V_E/SA_{TB})_H} \frac{(e^{-SA_{ET}/V_E})_A}{(e^{-SA_{ET}/V_E})_H}$$

where,

Rat

$V_E = 160.07$ ml/min or 0.16 L/min (derived using equation 4-4, default body wt. for Wistar rats, and the default intercept and coefficient values provided in Table 4-6)

$SA_{TB} = 22.5$ cm² (Table 4-4)

$SA_{ET} = 15.0$ cm² (Table 4-4)

Human

$V_E = 13.8$ L/min (default value based on human body weight of 70 kg)

$SA_{TB} = 3,200$ cm² (Table 4-4)

$SA_{ET} = 200.0$ cm² (Table 4-4)

$$= \frac{(0.16 \text{ L/min} / 22.5 \text{ cm}^2)_A}{(13.8 \text{ L/min} / 3,200 \text{ cm}^2)_H} \frac{(e^{-15.0 \text{ cm}^2 / 0.16 \text{ L/min}})_A}{(e^{-200.0 \text{ cm}^2 / 13.8 \text{ L/min}})_H}^*$$

* the exponential portion of the equation is much smaller than 1; thus this half of the equation is negligible.

$$= 0.007 / 0.0043$$

$$RGDR_{TB} = 1.6$$

Provisional Peer Reviewed Toxicity Values for

1,2-Dichloroethane (Ethylene dichloride)

(CASRN 107-06-2)

Derivation of a Chronic Oral RfD

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit

NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
1,2-DICHLOROETHANE (ETHYLENE DICHLORIDE) (CASRN 107-06-2)
Derivation of a Chronic Oral RfD**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

IRIS (U.S. EPA, 2002) reports that an RfD for 1,2-dichloroethane (1,2-DCA) is not available at this time. In addition, no RfD for 1,2-DCA was listed in the HEAST (U.S. EPA, 1997). U.S. EPA (1984, 1985a) reviewed chronic and subchronic oral studies (Alumot et al., 1976; Lane et al., 1982; Munson et al., 1982; NCI, 1978) but did not attempt to derive an oral RfD because 1,2-DCA is carcinogenic. U.S. EPA (1985b) presented interim RfD derivations based on the oral multigeneration study by Lane et al. (1982) and on inhalation data, but U.S. EPA (1987) concluded that no appropriate data were available for determining an RfD, and therefore, did not estimate a lifetime health advisory for this chemical. However, the NCI (1978) study was considered only for carcinogenic effects in the analysis by U.S. EPA (1987). Therefore, lacking any further update, no RfD for 1,2-DCA appears in the Drinking Water and Health Advisories List (U.S. EPA, 2000). No other EPA documents pertinent to derivation of an RfD for 1,2-DCA were located in the CARA lists (U.S. EPA, 1991, 1994).

ATSDR (1994, 2001) derived an intermediate-duration oral MRL of 0.2 mg/kg-day from a 13-week dosed-water study in rats (NTP, 1991) in which a LOAEL of 58 mg/kg-day for increased kidney weight was identified. Uncertainty factors used in the MRL derivation included 3 for use of a minimal LOAEL, 10 for interspecies extrapolation, and 10 for human variability.

Other resources consulted included the NTP (2001) Management Status Report, the IARC monograph series (IARC, 1979, 1999), WHO (1995), and Patty's Toxicology (Reid, 2001). Literature searches of the following databases were conducted from 1993 to April 2001 for relevant studies: TOXLINE, MEDLINE, TSCATS, GENETOX, HSDB, CANCERLIT, CCRIS, EMIC/EMICBACK, DART/ETICBACK, and RTECS.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

The most recent reviews (ATSDR, 1994, 2001; U.S. EPA, 1984, 1985a,b; IARC, 1999; WHO, 1995) reported that information concerning the toxic effects of ingested 1,2-DCA in humans comes primarily from case reports of individuals who accidentally or intentionally ingested 1,2-DCA. Only crude estimates of ingested dose are available, limiting the value of the data. Symptoms of 1,2-DCA intoxication include cardiac arrhythmia, bronchitis, hemorrhagic gastritis and colitis, hepatocellular damage, renal tubular necrosis and calcification, and central nervous system depression. Although an association between 1,2-DCA in drinking water and major birth defects was found in epidemiological studies, concurrent mixed chemical exposures indicate that the results are only suggestive, do not establish a cause-and-effect relationship, and should be interpreted with caution. Primary routes of exposure in these epidemiological studies may have been both oral and inhalation (volatilized from household water). No studies were located regarding immunological, reproductive, or developmental effects in humans exposed solely to 1,2-DCA by ingestion. The literature search identified no new studies examining the toxicity of 1,2-DCA in humans following oral exposure.

Animal Studies

In an NCI (1978) carcinogenicity study, Osborne-Mendel rats (50/sex/group) were treated with 1,2-DCA in corn oil by gavage at TWA doses of 47 or 95 mg/kg-day, 5 days/week for 78 weeks. B6C3F1 mice (50/sex/group) were also treated for 78 weeks with 97 or 195 mg/kg-day (male mice) and 149 or 299 mg/kg-day (female mice), 5 days/week. Untreated and vehicle controls (20/sex/group) were maintained concurrently. Signs of toxicity, body weight, and food consumption were evaluated throughout the study. Observation continued for 13 weeks after the dosing period. Comprehensive gross and histological examinations were performed upon moribund condition, death, or sacrifice at the end of the bioassay. Hematological and clinical chemistry determinations were not conducted.

In rats, there was no effect on body weight gain, but mortality was significantly ($p < 0.001$) increased in 95 mg/kg-day treated male and female rats compared with controls. Survival of male and female rats treated with 95 mg/kg-day was 50% at weeks 55 and 57, respectively. For rats treated with 47 mg/kg-day, survival was reported as 52% at 82 weeks for male rats and 50% at 85 weeks in female rats. Of the vehicle controls, 50% of male and female rats survived at least 72 and 88 weeks, respectively, while 50 and 60% of untreated male and female control rats survived until the end of the study. The study authors attributed the high mortality in the rats to toxic effects and bronchopneumonia, rather than to cancer. Several rats (number not reported) in both the 47 and 95 mg/kg-day dose groups had a hunched appearance and transient labored breathing beginning during the 6th week of treatment. Although one or two control rats (untreated or vehicle not specified) started to show these signs, the incidence was substantially higher in the treated groups than in the control groups. The only treatment-related nonneoplastic lesion found upon microscopic examination was spleen hematopoiesis in female rats which occurred in 1/20 (5%) vehicle controls, 0/50 low dose, and 16/50 (32%) of 95 mg/kg-day dosed animals. A LOAEL of 47 mg/kg-day (TWA = 34 mg/kg-day), the lowest dose tested, for serious clinical signs of labored breathing and hunched appearance is identified in both sexes of rats. However, this study was limited by dosage adjustments and poor survival.

Female mice treated with 195 mg/kg-day also had significantly increased mortality, possibly related to cancer, but mortality was not affected in the other groups of mice. For female mice, mortality (36/50, 72%) was significant in the 195 mg/kg-day group; these deaths may have been tumor-related as 25/36 (69%) had one or more tumors. For male mice, there was no statistically significant association between 1,2-DCA dosage and mortality. Clinical signs in treated groups were unremarkable compared with controls. Body weight was not affected by treatment in male mice or low-dose female mice. Body weight in high-dose female mice became significantly depressed around 30 weeks and was reduced by >45% of control weight at 90 weeks. The incidence of chronic murine pneumonia was dose-related in mice; present in 0/17 untreated control, 0/19 vehicle control, 5/46 (11%) low dose, and 11/47 (23%) high dose males, and in 0/19 untreated control, 0/20 vehicle control, 1/50 (2%) low dose and 6/48 (13%) high dose females. However, only the incidence in high dose males is statistically significant (Fisher's Exact test by SRC $p < 0.05$). No other treatment-related nonneoplastic lesions were found in mice. A NOAEL and LOAEL of 149 (TWA = 106 mg/kg-day) and 299 mg/kg-day (TWA = 214 mg/kg-day), respectively, the only two doses tested in female mice, are identified for reduced body weight in female mice. A NOAEL and LOAEL of 97 (TWA = 69 mg/kg-day) and 195 mg/kg-day (TWA = 139 mg/kg-day), respectively, the only two doses tested in male mice, are identified for significant dose-related incidence of chronic murine pneumonia in male mice.

_____ Groups of F344/N rats, Sprague-Dawley rats, Osborne-Mendel rats, and B6C3F1 mice (10 animals/sex) were exposed to drinking water containing 0, 500, 1000, 2000, 4000, or 8000 ppm of 1,2-DCA for 13 weeks (NTP, 1991). The high concentration was close to the solubility limit for 1,2-DCA in water. The authors estimated daily doses based on drinking water consumption and average body weights as follows:

Concentration in Water (ppm)	Doses in mg/kg-day in Rats					
	F344/N		Sprague-Dawley		Osborne-Mendel	
	Male	Female	Male	Female	Male	Female
500	49	58	60	76	54	82
1000	86	102	99	106	88	126
2000	147	182	165	172	146	213
4000	259	320	276	311	266	428
8000	515	601	518	531	492	727

Concentration in Water (ppm)	Doses in mg/kg-day Mice	
	Male	Female
500	249	244
1000	448	647
2000	781	1182
4000	2710	2478
8000	4207	4926

Additional groups of F344/N rats (10/sex) were administered 1,2-DCA by gavage on 5 days/week for 13 weeks to compare toxicity resulting from bolus administration with that of the continuous exposure in drinking water. Gavage doses were 0, 30, 60, 120, 240, or 480 mg/kg-day in the male rats and 0, 18, 37, 75, 150, or 300 mg/kg-day in the female rats. Signs of toxicity, body weight, food and water consumption, hematology and serum chemistry (in control and ≥ 2000 ppm male rat groups only) were evaluated throughout the study, and comprehensive gross and histological examinations were performed at the end of the exposure period.

Rat drinking water studies: None of the rats given 1,2-DCA in drinking water died. No treatment-related clinical signs were observed. Significantly (Dunn's test or Shirley's test) decreased mean body weights were seen in male F344/N rats at 4000 ($p < 0.01$) and 8000 ppm ($p < 0.01$) (259 and 515 mg/kg-day), in male Sprague-Dawley rats at 8000 ppm (518 mg/kg-day) ($p < 0.05$), and in male Osborne-Mendel rats at 8000 ppm (492 mg/kg-day) ($p < 0.05$). There were no significant reductions in body weight gain in female rats of any strain. Dose-related decreased water consumption occurred in all treated groups; 4-44% in F344/N males, 5-42% in F344/N females, 14-56% in male Sprague-Dawley males, 25-70% in Sprague-Dawley females, 17-60% in Osborne-Mendel male rats, and 21-58% in Osborne-Mendel female rats. Increases in erythrocyte counts and mild decreases in mean cell volume, measured in males, were attributed by the authors to dehydration. Alkaline phosphatase activity was significantly decreased on

selected test days at ≥ 2000 ppm in F344/N (≥ 147 mg/kg-day) and Sprague-Dawley (≥ 165 mg/kg-day) males, but was unaffected in Osborne-Mendel males. Alanine aminotransferase activity was significantly decreased on selected test days at ≥ 4000 ppm in F344/N (≥ 259 mg/kg-day) and Osborne-Mendel (≥ 266 mg/kg-day) males, at 8000 ppm (492 mg/kg-day) in Sprague-Dawley males. Blood urea nitrogen (BUN) levels were significantly increased in F344/N males at ≥ 2000 ppm (≥ 147 mg/kg-day) on days 7 and 14, and on day 3 at 4000 ppm. In Sprague-Dawley males, BUN levels were significantly increased at 2000 ppm (165 mg/kg-day) on days 7 and 45, at 4000 ppm (276 mg/kg-day) on days 7 and 45, and at 8000 ppm (518 mg/kg-day) on days 3, 7, and 45. BUN levels were significantly increased in male Osborne-Mendel rats at ≥ 4000 ppm (≥ 266 mg/kg-day) on day 3. Creatine kinase activity was unaffected in male rats of all three strains. Sorbitol dehydrogenase activity was significantly increased at 8000 ppm (515 mg/kg-day) on days 14 and 45 in F344/N males and at 8000 ppm (518 mg/kg-day) on day 14 in Sprague-Dawley males. Sorbitol dehydrogenase activity were unaffected in Osborne-Mendel males. Absolute kidney weights were significantly (Dunn's test or Shirley's test $p < 0.05$ or < 0.01) increased at > 500 ppm in female F344/N (≥ 58 mg/kg-day), Sprague-Dawley (≥ 76 mg/kg-day) and Osborne-Mendel rats (≥ 82 mg/kg-day), at ≥ 1000 ppm (≥ 86 mg/kg-day) in male F344/N rats, and at 1000 ppm (≥ 88 mg/kg-day) in male Osborne-Mendel rats. Absolute kidney weight changes were not significant in male Sprague-Dawley rats. Relative kidney weights were significantly increased at ≥ 500 ppm in Sprague-Dawley (≥ 76 mg/kg-day) and Osborne-Mendel (≥ 82 mg/kg-day) females, and at ≥ 1000 ppm (≥ 102 mg/kg-day) F344/N females. Relative kidney weights were significantly increased at ≥ 1000 ppm (≥ 86 mg/kg-day) in F344/N males, at ≥ 4000 ppm (≥ 266 mg/kg-day) in Osborne-Mendel males, and at 1000, 4000, and 8000 ppm (99, 276, and 518 mg/kg-day) in Sprague-Dawley males. Absolute liver weights were significantly increased at 2000 ppm (147 mg/kg-day) in male F344/N rats, at 1000 (102 mg/kg-day) and 4000 ppm (320 mg/kg-day) in female F344/N rats and at 1000 ppm (88 mg/kg-day) in male Osborne-Mendel rats. Absolute liver weight changes were not significant in male and female Sprague-Dawley rats and in female Osborne-Mendel rats. Relative liver weights were significantly increased at ≥ 2000 ppm (≥ 147 mg/kg-day) in F344/N males, at ≥ 4000 ppm (≥ 320 mg/kg-day) in F344/N female rats, at ≥ 500 ppm (≥ 60 mg/kg-day) in Sprague-Dawley male rats, at 8000 ppm (531 mg/kg-day) in Sprague-Dawley female rats, at 1000 and 2000 ppm (88 and 146 mg/kg-day) in Osborne-Mendel male rats. Relative liver weight changes were not significant in Osborne-Mendel females. No lesions attributable to 1,2-DCA were observed in the liver. The only histopathological finding was minimal to mild renal tubular regeneration, which was found in all groups of treated and control F344/N male rats, Sprague-Dawley male and female rats, and Osborne-Mendel male and female rats at similar incidence and severity. Minimal to mild renal tubular regeneration at similar incidence and severity was found in all groups of treated and control F344/N male and female rats, Osborne-Mendel male and female rats, and Sprague-Dawley male and female rats. Kidney and liver weights were increased in dosed rats of all three strains. No compound-related lesions were observed except for a dose-related incidence of renal tubular regeneration in female F344/N rats: 0/10 in controls, 0/10 at 58 mg/kg-day, 1/10 at 102 mg/kg-day, 2/10 at 182 mg/kg-day, 3/10 at 320 mg/kg-day and 9/10 at 601 mg/kg-day. Statistical analyses of the lesion incidence were not reported, but only the incidence at the high dose (601 mg/kg-day) is statistically significant according to Fisher's Exact Test performed for this assessment. However,

support for potentially associated renal functional deficits from serum chemistry measurements was lacking, as serum chemistry analyses were only performed in males. This lesion was of minimal severity in all affected rats. A minimal LOAEL of 58 mg/kg-day, the lowest dose tested, is identified for increased kidney weight in female F344/N rats. The increased kidney weight, without changes in body weight at this dose, is considered to be an early stage adverse effect because dose-related incidence of renal histopathology (tubular regeneration, indicative of previous tubular injury with subsequent repair) also developed at higher doses in the same strain of rats.

Mouse drinking water study: Before the end of the study deaths occurred only in 8000 ppm treated females (90%). No treatment-related clinical signs were observed. Body weight gain was significantly reduced (Dunn's test or Shirley's test $p < 0.01$) in high-dose males. Hematological and serum chemical analyses were not performed. Increased liver weight and/or liver:body weight ratio was significant at ≥ 500 ppm (≥ 249 mg/kg-day) in males and ≥ 1000 ppm (≥ 647 mg/kg-day) in females. Increased kidney weight and/or kidney:body weight ratio was significant at ≥ 1000 ppm (≥ 448 mg/kg-day) in males and ≥ 500 ppm (≥ 244 mg/kg-day) in females. A dose-related increased incidence of tubular regeneration (minimal to moderate) occurred in males; 0/10 in controls, 1/10 at 500 ppm (249 mg/kg-day), 2/10 at 1000 ppm (448 mg/kg-day), 2/10 at 2000 ppm (781 mg/kg-day), 8/10 ($p < 0.01$) at 4000 ppm (2710 mg/kg-day) and 9/10 ($p < 0.01$) at 8000 ppm (4207 mg/kg-day). Other renal lesions, including karyomegaly (10/10), dilatation (5/10), protein casts (8/10), and mineralization (5/10), occurred significantly in males at the highest dose. Tubular regeneration was observed in 1/10 females at 4000 ppm (2478 mg/kg-day); no other renal lesions were reported in females. Treatment-related lesions were not reported in other organs. Significantly increased absolute and relative kidney weights occurred in male mice at ≥ 249 mg/kg-day. In addition, a dose-related increased incidence of tubular regeneration, achieving statistical significance at ≥ 2710 mg/kg-day, occurred in males. A NOAEL and minimal LOAEL of 249 and 448 mg/kg-day, respectively, are identified for increased kidney weight in male mice. The increased kidney weight is considered to be an early stage adverse effect because dose-related incidence of renal histopathology developed at higher doses in male mice.

Rat gavage study: Additional groups of F344/N rats (10/sex) were administered 1,2-DCA by gavage on 5 days/week for 13 weeks to compare toxicity resulting from bolus administration with that of the continuous exposure in drinking water. Gavage doses were 0, 30, 60, 120, 240, or 480 mg/kg-day in the male rats and 0, 18, 37, 75, 150, or 300 mg/kg-day in the female rats. Signs of toxicity, body weight, food and water consumption, hematology and serum chemistry (in control, 120 and 240 mg/kg-day male rat groups only) were evaluated throughout the study, and comprehensive gross and histological examinations were performed at the end of the exposure period. Deaths occurred in all males before the end of the study at ≥ 240 mg/kg-day and 90% of females at 300 mg/kg-day; clinical signs preceding death included tremors, salivation, and emaciation. Pathology in moribund/dead animals included necrosis in the thymus and cerebellum. No deaths occurred at other doses. Small but significant changes in various hematological parameters occurred in higher dose groups and were considered to be indicative of

dehydration and attributed to significantly reduced water consumption (60% compared to controls). There were no effects on growth at sublethal doses. Other effects included minimal to mild hyperplasia and inflammation of the forestomach epithelium (sometimes with foci of necrosis and mineralization); 0/10 in controls of each sex, 1/10 males at 120 mg/kg-day, 5/10 males at 240 mg/kg-day, 3/10 males at 480 mg/kg-day, and 3/10 females at 300 mg/kg-day (histological examinations were not performed on low dose animals). Liver weight and liver:body weight ratio significantly increased in males at 120 mg/kg-day (no data from higher doses due to mortality) and females at all doses. Kidney weight and/or kidney:body weight ratio significantly increased in males at ≥ 30 mg/kg-day and ≥ 75 mg/kg-day in females. No liver lesions were reported and incidence of renal tubular regeneration in dosed groups was comparable to vehicle control. Small, but significant, changes in various hematological and serum chemistry parameters, compared with controls, were measured in males dosed at 120 and 240 mg/kg-day; however, these measurements were not conducted in other male treatment groups. These changes were considered to be indicative of dehydration and attributed to significantly reduced water consumption (60% compared with controls). These experiments identify an FEL of 240 mg/kg-day for significant mortality in male F344/N rats. It is difficult to discern a LOAEL in this study, as organ weight changes were not accompanied by histological alterations. The NOAEL is 120 mg/kg-day. Based on significant organ weight changes in rats treated by gavage or drinking water, the liver and kidney appear to be target organs for 1,2-dichloroethane.

WHO (1995) briefly reviewed a study by Van Esch et al. (1977), whereby rats of both sexes (number and strain not specified) were treated by gavage with 1,2-DCA at 0, 10, 30 or 90 mg/kg-day for 5 days/week for 90 days. Six rats were dosed with 300 mg/kg-day for 5 days and all rats died. Decreased weight gain was observed at the two highest doses. Increased relative kidney weight occurred in both sexes at 90 mg/kg-day, but increased relative liver and brain weight was seen only in the females at 90 mg/kg-day. Clinical chemistry parameters were normal, and there were no treatment-related histopathological lesions. Sporadic hematological changes were seen, but not in a dose-related manner. The histology on the six rats that died after receiving 300 mg/kg-day 1,2-DCA for 5 days revealed fatty degeneration of the liver and an increase in liver triglycerides. A NOAEL and LOAEL of 90 and 300 mg/kg-day, respectively, are identified for fatty liver with increased triglycerides.

Sprague-Dawley rats (10/sex/dose) received 0 (vehicle control), 37.5, 75, or 150 mg/kg-day of 1,2-DCA in corn oil by gavage for 90 consecutive days (Daniel et al., 1994). Signs of toxicity, body weight, and food consumption were evaluated throughout the study. Ophthalmoscopic examinations were performed prior to treatment and during the last week of the study. Blood and urine chemistry analyses were performed during the last week of the study. Comprehensive gross examinations were performed at necropsy. There were no treatment-related deaths or clinical signs of toxicity. Body weight gain and food consumption were significantly decreased at 150 mg/kg-day in males, but comparable to control in all other groups. In males, relative brain, kidney, and liver weights were significantly increased at ≥ 75 mg/kg-day. In females, relative kidney weight was increased at ≥ 75 mg/kg-day and relative liver weight at

150 mg/kg-day. No significant ocular changes were observed. In females, red blood cells, lymphocytes, hemoglobin, and hematocrit were significantly decreased while platelets, white blood cells, neutrophils and monocytes were significantly increased at the highest dose. In males, hemoglobin and hematocrit were significantly decreased at the two highest doses while platelets were increased only in the high dose group. In females, potassium levels were increased and albumin levels decreased in the two highest dose groups, while in males, alkaline phosphatase was increased at these same doses (data not presented). Urinalysis data were unremarkable. None of the few gross or microscopic lesions observed were considered to be of toxicological significance (no further details reported). A NOAEL and LOAEL of 37.5 and 75 mg/kg-day, respectively, for hematological changes are identified.

Bred female Sprague Dawley rats (25-26/group) were given 0 (vehicle control), 1.2, 1.6, 2.0 or 2.4 mmol/kg-day 1,2-DCA dissolved in corn oil (0, 119, 158, 198 or 238 mg/kg-day) by oral gavage on gestation days 6-20 (15 days) (Payan et al., 1995). No maternal deaths occurred. A dose-related reduction in maternal weight body gain during pregnancy occurred, with statistical significance achieved at the two highest doses (>30% reduction). Pregnancy rates were similar in all groups. No significant effect of 1,2-DCA was noted on the mean number of implantation sites and live fetuses, fetal sex ratio, and male and female fetal weights. There was a slight but significant trend for increase in the mean percentages of nonsurviving implants and resorptions relative to control animals with statistically significant differences only at 198 mg/kg-day. Incidences of visceral and skeletal variations and malformations of the fetus were similar in all groups. A NOAEL and LOAEL of 158 and 198 mg/kg-day, respectively, are identified for increases in nonsurviving implants and resorptions.

In a study to determine the possible immunotoxicity of 1,2-DCA, male CD-1 mice (32/group) were treated with drinking water containing 0, 20, 200 or 2000 ppm 1,2-DCA (0, 3, 24 or 189 mg/kg-day, as calculated by the authors) for 90 days (Munson et al., 1982). Reduced water consumption was seen at 24 and 189 mg/kg-day, and an appreciable decrease in growth was seen in the high-dose group. No significant effects were seen on organ weights, hematological parameters, or immunological function. Since this study was mainly concerned with immunological end points, histological examination of organs and tissues was not conducted. This study identifies a NOAEL of 189 mg/kg-day, the highest dose tested, for immunological effects in mice.

In a 2-year study, groups of rats (18/sex/dose) of unspecified strain were fed a feed mash fumigated with 1,2-DCA that resulted in concentrations of 0, 250, or 500 ppm (equivalent to \approx 0, 12, or 25 mg/kg-day) (Alumot et al., 1976). No effects were found on mortality, growth, food consumption, or liver or kidney function as determined by analysis of serum chemistry indices, but histological examinations were not performed. No differences were found in the percentage of females bearing litters, litter size, mortality, and body weights of pups. In a preliminary study, in which rats were fed a dietary level of 1600 ppm (\approx 80 mg/kg-day) for 7 weeks, hepatic biochemical changes consisting of a significant 15% increase in fat accumulation and 75% increase in total triglycerides were observed, although liver weight was unchanged. Histological

examinations were not performed. A NOAEL of 25 mg/kg-day for changes in serum chemistry is identified. A NOAEL and LOAEL of 25 and 80 mg/kg-day, respectively, are identified for biochemical changes in the liver indicating an increase in fat and triglyceride storage. However, this study is limited by inadequacies in the conduct and reporting (U.S. EPA, 1985b).

In a multigeneration reproduction study, male and female ICR Swiss mice were given drinking water containing 0, 30, 90, or 290 ppm of 1,2-DCA, giving nominal daily doses of 0, 5, 15, or 50 mg/kg-day (Lane et al., 1982). No parental treatment-related effects were observed in F0 and F1B generations as judged by mortality rates, fluid intake, body weight gain, and gross pathology. No significant increase in gross, visceral or skeletal anomalies or any fetotoxic effects were observed. Furthermore, there were no significant differences between treated and control groups for number of pregnant females, weight gain, implants/litter, resorptions, live fetuses and 4- and 21-day survival. There was also no evidence of dominant lethality in treated males mated to untreated females. This study identifies a NOAEL of 50 mg/kg-day, the highest dose tested, for parental and fetal toxicity.

DERIVATION OF A PROVISIONAL ORAL RfD FOR 1,2-DICHLOROETHANE

Cardiac arrhythmia, bronchitis, central nervous system depression, and injury to the liver, kidneys, and gastrointestinal tract have occurred in humans following ingestion of 1,2-DCA (ATSDR, 1994, 2001; IARC, 1999; U.S. EPA, 1984, 1985a,b; WHO, 1995); however, no human studies are suitable for derivation of an RfD for 1,2-DCA. One- and two-generation reproduction studies found no chemical-related effects on fertility indices in long-term feed or drinking water studies in mice and rats (Alumot et al., 1976; Lane et al., 1982), but exposure to a higher oral gavage dose of 198 mg/kg-day caused increases in nonsurviving implants and resorptions in rats that also experienced maternal toxicity (>30% decreased body weight gain) (Payan et al., 1995). While reproductive performance was not evaluated, histological examinations showed no changes in male or female reproductive tissues in rats administered ≤ 480 mg/kg-day by gavage for ≤ 90 days (Daniel et al., 1994; NTP, 1991; Van Esch et al., 1977), in rats and mice exposed to ≤ 492 and $\leq 4,210$ mg/kg-day, respectively, in drinking water for ≤ 13 weeks (NTP, 1991), or in rats and mice exposed to ≤ 95 and ≤ 299 mg/kg-day, respectively, by gavage for ≤ 78 weeks (NCI, 1978).

Clinical signs of toxicity have only been observed in the gavage studies; tremors, salivation, and emaciation at ≥ 240 mg/kg-day (NTP, 1991) and hunched appearance and labored breathing at ≥ 47 mg/kg-day (NCI, 1978) in rats. However, in another gavage study no clinical signs of toxicity were observed at ≤ 150 mg/kg-day (Daniel et al., 1994). In drinking water studies, clinical signs of toxicity were not observed at ≤ 727 mg/kg-day in rats and $\leq 4,926$ mg/kg-day in mice (NTP, 1991).

Increased organ weights in animal oral studies (Daniel et al., 1994; NTP, 1991; Van Esch et al., 1977) at ≥ 18 mg/kg-day, and clinical chemistry results in an oral study (Daniel et al., 1994) at ≥ 75 mg/kg-day, support the kidney and liver as target organs for 1,2-DCA toxicity. While animals studies point to the kidney and liver as target organs of 1,2-DCA toxicity, the database indicates that the dose-relation for toxic effects, including kidney and liver effects, following gavage or dosed drinking water exposure differ. Dose-related organ histopathology was observed in drinking water studies, but not in gavage studies. While kidney and liver weights increased following dosed water administration of 1,2-DCA, accompanying dose-related histopathology was only observed in the kidney. Therefore, the kidney is selected as the target organ for chronic toxicity effects of 1,2-DCA.

Renal effects reported in animals include increases in kidney weight and minimal-to-moderate histopathological changes after longer-term exposures. Relative kidney weight was increased without altered histology in rats that were treated with 75-90 mg/kg-day by gavage for 90 days (Daniel et al., 1994; Van Esch et al., 1977). An NTP (1991) 13-week gavage study in rats found significant dose-related increases in kidney weight and kidney-to-body-weight ratio at 30-120 mg/kg-day in males and 75-150 mg/kg-day in females (kidney weight was not measured in higher-dose animals because of mortality). Exposure to 1,2-DCA in the drinking water for 13 weeks caused significant dose-related increases in kidney weight and kidney-to-body-weight ratio in rats at ≥ 58 mg/kg-day and mice at ≥ 244 mg/kg-day (NTP, 1991). Histopathological examination of the animals in drinking water studies showed dose-related increased incidences of minimal-to-moderate renal regeneration in female rats at ≥ 102 mg/kg-day and in male mice at ≥ 249 mg/kg-day (NTP, 1991). These changes are indicative of previous tubular injury with subsequent repair. More severe renal effects including karyomegaly, dilation, protein casts, and mineralization occurred in male mice exposed to 4,207 mg/kg-day of 1,2-DCA (NTP, 1991). However, there were no changes in kidney weight in mice after exposure to 189 mg/kg-day in drinking water for 90 days (Munson et al., 1982), and kidney function, as measured by changes in serum levels of urea and uric acid, was normal in rats exposed to 25 mg/kg-day in food for 2 years (Alumot et al., 1976). Histological examination of the kidney was not performed in either of these studies. No histological changes were observed in the kidneys of rats and mice that were administered ≤ 95 and ≤ 299 mg/kg-day, respectively, by gavage for ≤ 78 weeks (NCI, 1978), or in the kidneys of rats that were administered ≤ 480 mg/kg-day by gavage for 13 weeks (NTP, 1991).

It is more appropriate to base an RfD on an effect level from a drinking water study rather than from a gavage study due to toxicokinetic considerations. Bolus administration may cause saturation of the detoxification/ excretion mechanism resulting in higher blood levels and apparent adverse effects at lower gavage doses. The lowest dose in female rats, 58 mg/kg-day, a minimal LOAEL for increased kidney weight (NTP, 1991) is selected as the basis of the RfD. The increased kidney weight is considered to be an early-stage adverse effect because a dose-related increase in the incidence of renal tubular regeneration developed at doses greater than the minimal LOAEL in the same strain (F344/N) of rats. Increased kidney weights at

≥448 mg/kg-day, accompanied by renal tubular regeneration at higher doses, were also observed in another drinking water study in male mice (NTP, 1991).

With a modifying factor of unity, dividing the minimal LOAEL of 58 mg/kg-day by an uncertainty factor of 3,000 (3 for use of a LOAEL, 10 for extrapolation to chronic duration, 10 for interspecies extrapolation, and 10 for human variability) results in the provisional RfD of **0.02 mg/kg-day**.

$$\begin{aligned}\text{p-RfD} &= \text{LOAEL} \div (\text{UF} \times \text{MF}) \\ &= 58 \text{ mg/kg-day} \div (3,000 \times 1) \\ &= \mathbf{0.02 \text{ mg/kg-day}}\end{aligned}$$

STATEMENT OF CONFIDENCE

Confidence in the principal study (NTP, 1991) is medium. The study used adequate numbers of mice and three strains of rats and administered the test chemical in an extensive range of doses by gavage or dosed-drinking water for comparison of bolus and continuous exposure. Sufficient detail was provided for both methods and results. Confidence in the database is medium. Although it describes a range of dose-related effects in subchronic and developmental toxicity studies, the single chronic study indicates serious effects at the lowest level, which have not been confirmed in other studies, and the test chemical was administered by gavage. Medium confidence in the RfD for 1,2-DCA results.

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11-30-2003

Provisional Peer Reviewed Toxicity Values for

1,2-Dichloropropane (CASRN 78-87-5)

Derivation of a Carcinogenicity Assessment

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
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Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
1,2-DICHLOROPROPANE (CASRN 78-87-5)
Derivation of a Carcinogenicity Assessment**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

An inhalation unit risk for 1,2-dichloropropane is not available on IRIS (U.S. EPA, 2003) or in the HEAST (U.S. EPA, 1997). Relevant documents in the CARA list (U.S. EPA, 1991, 1994) include a Health Effects Assessment (U.S. EPA, 1987a), a Drinking Water Criteria Document (U.S. EPA, 1987b), and a Health and Environmental Effects Profile (U.S. EPA, 1985). An inhalation unit risk was not derived in any of these documents due to the absence of adequate inhalation cancer data. CAL/EPA (1997, 2001) derived an inhalation unit risk of $1.8\text{E-}05$ (g/m^3)⁻¹ by route-to-route extrapolation from an oral slope factor of $3.6\text{E-}02$ ($\text{mg}/\text{kg}\cdot\text{day}$)⁻¹. Available reviews by IARC (1986, 1999), ATSDR (1989), IPCS (1993) and Reid (2001), and the NTP (2001) status report, were consulted to identify relevant studies. Literature searches of the following databases were conducted from 1984 to October 2001: TOXLINE, MEDLINE,

TSCATS, GENETOX, HSDB, CANCERLIT, CCRIS, EMIC/EMICBACK, DART/ETICBACK and RTECS.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

An epidemiological study of 71 Italian florists, who were found to have used an average of 162 kg/year of 1,2-dichloropropane (as well as other fumigants, insecticides and fungicides), showed that the mean frequency of peripheral lymphocyte micronuclei for the florists was higher than the mean for 75 controls (Bolognesi et al., 1995). However, due to the confounding exposure to other chemicals, it is not clear that this finding was attributable to 1,2-dichloropropane exposure.

Animal Studies

The available reviews identified a single study regarding the carcinogenicity of 1,2-dichloropropane in animals by inhalation exposure (U.S. EPA, 1987a,b). Heppel et al. (1948) briefly described an experiment in which 80 C3H mice were exposed a total of 37 times to 400 ppm of 1,2-dichloropropane, 4 to 7 hours per exposure, and observed for 7 months. Only 3/80 mice survived; multiple hepatomas were seen in all 3 survivors. The early mortality was associated with severe necrotic liver lesions. No additional inhalation cancer studies were located in the literature search.

An oral cancer bioassay conducted in rats and mice by NTP (1986) concluded that there was *some evidence of carcinogenicity* in both male and female mice based on increased incidences of liver tumors (primarily adenomas), *equivocal evidence of carcinogenicity* in female rats based on mammary gland adenocarcinomas, and *no evidence of carcinogenicity* in male rats.

Other Studies

1,2-Dichloropropane has produced generally positive results in a variety of genotoxicity assays (ATSDR, 1989; IARC, 1999; U.S. EPA, 1987a,b). In bacteria, 1,2-dichloropropane induced reverse mutations in *Salmonella* with and without activation in a number of studies, but was not mutagenic in *Streptomyces*. 1,2-Dichloropropane weakly induced mutations in the fungus *Aspergillus*, but did not produce chromosomal effects in this species. In mammalian cells, results were positive for mutation in mouse lymphoma cells when tested with activation, but not without. With or without activation, results were positive for sister chromatid exchange and chromosomal aberrations in Chinese hamster ovary cells. *In vivo* studies found that 1,2-

dichloropropane did not induce sex-linked recessive lethal mutations in *Drosophila* or dominant lethal mutations in mice, but did produce chromosomal aberrations in rat bone marrow.

FEASIBILITY OF DERIVING A PROVISIONAL INHALATION UNIT RISK FOR 1,2-DICHLOROPROPANE

The only relevant inhalation study (Heppel et al., 1948) was inadequate to evaluate the carcinogenicity of 1,2-dichloropropane. On the basis of the available information, it is not feasible to derive a provisional inhalation unit risk for 1,2-dichloropropane.

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7-30-2007

Provisional Peer Reviewed Toxicity Values for
2,4-Dichlorophenol
(CASRN 120-83-2)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor

p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2,4-DICHLOROPHENOL (CASRN 120-83-2)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A chronic reference dose (RfD) for 2,4-dichlorophenol (2,4-DCP) is available on IRIS (U.S. EPA, 2007). The RfD of 0.003 mg/kg-day is based on decreased delayed hypersensitivity response in a rat study (Exon and Koller, 1985). Rats were exposed during gestation, through lactation and in drinking water for 15 weeks and a NOEL of 0.3 mg/kg-day was identified (Exon and Koller, 1985). Uncertainty factors of 10 each for interspecies extrapolation and protection of sensitive humans were applied to the NOEL to derive the RfD. The source document was a U.S. EPA (1985) Drinking Water Criteria Document (DWCD). The Drinking Water Standards and Health Advisories list (U.S. EPA, 2006) includes the same chronic RfD of 0.003 mg/kg-day as reported on IRIS. The HEAST (U.S. EPA, 1997) reports a subchronic RfD of 0.003 mg/kg-day for 2,4-DCP, adopting the chronic RfD from IRIS as the subchronic RfD. ATSDR (1999) has prepared a toxicological profile for chlorophenols. An intermediate-duration oral Minimal Risk Level (MRL) of 0.003 mg/kg-day was derived for 2,4-DCP by ATSDR (1999). The MRL is based on the same study, endpoint, and uncertainty factors as the IRIS chronic RfD. The World Health Organization (WHO, 1989, 2003) evaluated the toxicity of 2,4-DCP. WHO (2003) declined to derive health-based guideline values, citing limitations in the toxicity database. Because a chronic RfD is available on IRIS, the present document does not include a chronic provisional-RfD (p-RfD); however, a subchronic p-RfD is included.

An RfC for 2,4-DCP is not available on IRIS (U.S. EPA, 2007) or in the HEAST (U.S. EPA, 1997). ATSDR (1999) did not derive any inhalation MRLs for 2,4-DCP, as there were no inhalation toxicity studies of this compound. The CARA list (U.S. EPA, 1991, 1994) includes a Health and Environmental Assessment (HEA) for 2,4-DCP (U.S. EPA, 1987a) and a Health and

Environmental Effects Document (HEED) for Chlorinated Phenols (U.S. EPA, 1987b), in addition to the DWCD (U.S. EPA, 1985) cited above, but none of these documents reported pertinent data regarding subchronic or chronic inhalation toxicity. The American Conference of Governmental Industrial Hygienist (ACGIH, 2006), Occupational Safety and Health Administration (OSHA, 2006) and National Institute for Occupational Safety and Health (NIOSH, 2006) have not established occupational health standards for 2,4-DCP.

A carcinogenicity assessment for 2,4-DCP is not available on IRIS (U.S. EPA, 2007) or in the HEAST (U.S. EPA, 1997). The Drinking Water Standards and Health Advisories document classifies the carcinogenicity of 2,4-DCP in category E, evidence of noncarcinogenicity for humans (U.S. EPA, 2006). The International Agency for Research on Cancer (IARC, 1999) concluded that there was *evidence suggesting lack of carcinogenicity* for 2,4-DCP in experimental animals based on an oral study in mice and two oral studies in rats (NTP, 1989; Exon and Koller, 1985). 2,4-DCP is not included in the National Toxicology Program's (NTP) 11th Report on Carcinogens (NTP, 2006). Patty's Toxicology (Gingell et al., 2001) was also consulted for relevant information.

To identify toxicological information pertinent to the derivation of provisional toxicity values for 2,4-DCP, searches were conducted in August, 2006 for literature dating from the 1960s to 2006 using the following databases: MEDLINE, TOXLINE, BIOSIS, TSCATS, CCRIS, Current Contents, DART/ETIC, GENETOX, HSDB and RTECS.

REVIEW OF PERTINENT DATA

Human Studies

Studies exist regarding the toxicity of chlorophenol mixtures in humans, but neither the literature search nor available reviews (U.S. EPA, 1987a,b; ATSDR, 1999; IARC, 1986, 1999; WHO, 1989) identified any studies regarding the toxicity of 2,4-DCP as a single agent in humans. Among workers in a 2,4-DCP and 2,4,5-trichlorophenol manufacturing plant, chloracne and porphyria were detected (Bleiberg et al., 1964). Elevated serum transaminase levels and evidence of liver damage (regeneration and hemofuscin deposition) were detected by liver biopsy in two cases. Poland et al. (1971) examined employees from the same plant 6 years after the report of Bleiberg et al. (1964). Of the 73 male workers examined, 48 (66%) had some degree of acne, and chloracne was found in 13 workers (18%); no cases of clinical porphyria were documented and only one worker had uroporphyrinuria. The severity of the chloracne was not correlated with job location within the plant or duration of employment. A causal relationship between these effects and exposure to 2,4-DCP in these workers cannot be assumed due to concurrent exposure to a variety of chlorinated compounds.

The literature search did not identify any studies regarding carcinogenicity of 2,4-DCP as a single agent in humans. There are several case-control (Eriksson et al., 1981; Hardell, 1981; Hardell et al., 1981, 1982; Hardell and Sandstrom, 1979) and cohort studies (Axelson et al., 1980; Bueno de Mesquita et al., 1993; Hogsted and Westerlund, 1980; Kogevinas et al., 1992, 1993; Saracci et al., 1991; Lynge, 1987; Riihimaki et al., 1982, 1983; U.S. Air Force, 1983;

Vena et al., 1998) of workers involved in the manufacture of phenoxy herbicides based on 2,4-DCP. In these studies, workers were exposed to a mixture of chemicals including 2,4-DCP and it was not possible to link mortality or tumor incidence to any particular chemical exposure in any of these studies. A follow up of a cohort study to investigate the potential carcinogenic effect of phenoxy herbicides (e.g., 2,4-dichlorophenol) in Danish workers (Lyng, 1985) reported soft tissue sarcomas in male workers. However, the total cancer risk among persons employed in the manufacturing and packaging of phenoxy herbicides was equivalent to the cancer risk in the Danish population. Furthermore, this study had several potential biases, such as exposure to mixtures of other chemicals. Therefore, observed cancer incidences cannot be linked to 2,4-dichlorophenol.

Morbidity and Mortality Weekly Report (MMWR, 2000) reported 5 human fatalities associated with acute exposure to 2,4-DCP in occupational settings. In each of these cases, dermal exposure was significant and chemical burns were common. Inhalation exposure was possible in several of the cases, but the proportion of dose attributable to inhalation exposure could not be determined (MMWR, 2000). Kintz et al. (1992) also reported a fatality associated with dermal exposure to 2,4-DCP. After spilling the pure compound on his right thigh and arm, a 33-year old man experienced seizures and died. His blood level of 2,4-DCP was measured to be 24.3 mg/L.

Animal Studies

Oral Exposure

Subchronic Exposure — In a subchronic study using CD-1 mice, Borzelleca et al. (1985a) administered 2,4-DCP (99% pure) in drinking water to groups of 20 male and 20 female mice for 90 days. Concentrations of 0.2, 0.6 or 2.0 mg/L in 10% Emulphor were added to the drinking water of treated animals; two control groups received either vehicle or deionized water. Water was provided *ad libitum* and intake was measured twice weekly. Clinical observations were made twice daily and body weights were measured weekly. At study termination, surviving mice were sacrificed and necropsied. Blood was collected for hematology (erythrocyte count, leukocyte count [total and differential], platelet count, hematocrit, hemoglobin [Hb], prothrombin and thromboplastin times, plasma fibrinogen) and clinical chemistry (aspartate aminotransferase [AST], alanine aminotransferase [ALT], lactate dehydrogenase [LDH], alkaline phosphatase [ALP], blood urea nitrogen [BUN], glucose, bilirubin, albumin, total protein, cholesterol, creatinine, phosphorus, calcium, globulin, albumin/globulin ratio and electrolytes). Hepatic microsomal enzyme activities (ethoxycoumarin O-deethylase, testosterone hydroxylation, cytochrome p-450 reductase) were also measured. Brain, liver, spleen, lung, thymus and kidney weights were recorded. No tissues were examined histologically.

Based on measured water consumption and body weights, the authors estimated doses of 0, 40, 114 and 383 mg/kg-day for male mice and 0, 50, 143 and 491 mg/kg-day for female mice (Borzelleca et al., 1985a). Treatment with 2,4-DCP did not result in significant differences in body weight, absolute or relative organ weights or microsomal enzyme activity when compared with the vehicle control; however, the vehicle itself (Emulphor) apparently increased body weight and altered some organ weights compared with the naïve controls. Among male rats

treated at the high dose, there was a significant increase in leukocytes (35% higher than vehicle control, $p \leq 0.05$). In females at the high dose, a significant increase in ALP was observed (1.7-fold higher, $p \leq 0.05$). The significance of these changes is uncertain in the absence of toxicological correlates. No other changes in hematology or clinical chemistry were dose-related. Effect levels cannot be identified from these data due to the confounding effect of the vehicle and the absence of histopathological evaluation.

The NTP sponsored a 13-week study to evaluate the toxic effects of subchronic oral exposure to 2,4-DCP and to determine the appropriate doses to be used in a 2-year study (NTP, 1989). F344/N rats and B6C3F1 mice (10/sex) were given 2,4-DCP (>99% pure) in the diet at concentrations of 0, 2500, 5000, 10,000, 20,000 or 40,000 ppm. Clinical observations were performed twice daily; body weight and food consumption were measured twice during the study. After 13 weeks, all animals were subjected to necropsy and histological examination of a comprehensive list of tissues (>40) was conducted on the control and high-dose animals. In the 10,000 ppm and 20,000 ppm rats, the bone marrow, colon, heart, jejunum, stomach and urinary bladder were evaluated histologically; the femoral bone marrow was also examined in 2500 ppm and 5000 ppm female rats. Histologic examination of the liver was performed in 2500, 5000 and 10,000 ppm mice of both sexes.

Using limited data on food consumption and body weight from the report (NTP, 1989), doses in rats can be estimated as 160, 310, 675, 1373 and 2703 mg/kg-day in males and 182, 338, 750, 1376 and 2795 mg/kg-day in females. All rats survived to study termination. Body weights decreased in a dose-related fashion in both sexes. Terminal body weight was significantly lower than controls in males exposed to 10,000 ppm and higher ($p < 0.01$ in t-test performed for this review). Terminal body weight was lower than controls by 5%, 20% and 40% at 10,000, 20,000, and 40,000 ppm, respectively. In females, terminal body weight was significantly below control values at concentrations of 20,000 and 40,000 ppm (11% and 21% lower). Average food consumption (measured on weeks 7 and 13) was decreased to 77% and 81% of control values in males and females exposed to 40,000 ppm. Statistical comparison of the food consumption rates was not reported. Rats exposed to 40,000 ppm exhibited hunched posture and rough hair coats. At necropsy, histopathology evaluations revealed bone marrow atrophy in all animals of both sexes exposed to the two highest concentrations and in females (6/10) of the 10,000 ppm group. The incidence of bone marrow atrophy in controls and lower dose groups, if any, was not reported. No other histopathology findings were reported. This study identifies a LOAEL of 10,000 ppm (750 mg/kg-day) for bone marrow atrophy in females. Male rats treated at this concentration also had slight reductions in body weight (5%). The 5000 ppm concentration (338 mg/kg-day) represents a NOAEL.

Based on limited data on food consumption and body weight from the report (NTP, 1989), doses in mice¹ can be estimated as 782, 1533, 1627, 2960 and 6805 mg/kg-day in males and 973, 2438, 3305, 3913 and 8911 mg/kg-day in females. Among high-dose mice, mortality was 100% within the first 3 weeks on study; survival was not different from controls in other

¹ Two measurements of body weight and food consumption were available for all but the high-dose group; due to the mortality in this group, only the initial body weight was available. Food consumption for this group was assumed to be equal to the intake measured in the next lower dose group (20,000 ppm group) for the purpose of these calculations.

dose groups of either sex. There was some evidence for an effect of 2,4-DCP treatment on body weight. In male mice exposed to 20,000 ppm, terminal body weight was 12% below that of controls ($p < 0.01$ based on t-test conducted for this review). In females at this concentration, body weights were reduced by about 10-15% from control values during most of the study, but there was no difference from controls in terminal body weight due to a 10% decline in control weights during the final week; the authors did not suggest a cause for this decrease in control weights. Average food consumption (measured at weeks 7 and 13) was 67 to 77% of control values in mice of both sexes exposed to 10,000 ppm, and 44 to 57% of controls at 20,000 ppm; statistical comparison of the consumption rates was not presented. At 10,000 ppm and higher concentrations, mice of both sexes exhibited rough hair coats; the incidences were not reported. Histologic examination of the liver showed hepatocellular necrosis in male mice at all dose levels (0/10, 4/10, 4/10, 6/10, 10/10 at 0, 2500, 5000, 10,000, and 20,000 ppm). The increase was statistically significant ($p < 0.05$) at all treatment levels; however, the severity was characterized as minimal at concentrations below 20,000 ppm. Syncytial alterations (multinucleated hepatocytes) were observed in all male mice exposed to 10,000 and 20,000 ppm 2,4-DCP, but not in exposed females, controls of either sex, or males exposed to 2500 or 5000 ppm. NTP (1989) noted renal tubular epithelial necrosis in eight male and three female mice at 40,000 ppm; all of these animals died in the first three weeks on study. The 2500 ppm concentration (782 mg/kg-day) is considered a minimal LOAEL based on mild hepatocellular necrosis in male mice; no NOAEL can be identified.

Exon et al. (1984; Exon and Koller, 1985) evaluated the immunotoxicity of 2,4-DCP in male and female Sprague-Dawley rats exposed prenatally or both pre- and postnatally. All animals were offspring (10/group) of dams exposed via drinking water to concentrations of 0, 3, 30 or 300 ppm 2,4-DCP (99% pure) from 3 weeks of age through breeding (at 90 days) and parturition (these were the same rats used in the reproductive study reported in the same publication; see below). Upon weaning, the prenatal-only groups were maintained untreated until 6 weeks of age, when immunocompetence was assessed. Dams of the pre- and postnatal groups were treated throughout the lactation period and, after weaning, offspring were given the treatment compound in the drinking water at the same concentrations until 13 weeks of age. Based on reported body weight and default water consumption values (U.S. EPA, 1988), the postnatal exposure concentrations correspond to doses of approximately 0.3, 3.0 and 30 mg/kg-day. The animals were weighed biweekly and observed daily for clinical signs. At 6 weeks and 13 weeks of age, respectively, the immunocompetence of groups treated prenatally and both pre- and postnatally was evaluated. Humoral and cell-mediated immune responses and macrophage function were assessed. Humoral immunity was quantified using ELISA assays for IgG antibodies to bovine serum albumin (BSA) or keyhole limpet hemocyanin (KLH). Cell-mediated immunity was measured using delayed-type hypersensitivity response to oxazolone (ear application) or BSA (footpad injection). Finally, macrophage function was assessed *in vitro*. Body and organ weights (liver, spleen and thymus) were recorded at sacrifice; complete necropsies were performed, as well as histopathologic evaluation of the liver, spleen and thymus.

Among the rats treated prenatally (and not postnatally), there was no effect on body weight when measured at 6 weeks of age; however, absolute spleen weights were significantly increased (29%, $p < 0.05$) in rats treated at 300 ppm. Prenatal exposure alone did not significantly

alter any of the immune parameters assessed, and there were no significant histopathology findings.

Among rats treated at 300 ppm both pre- and postnatally, there was a significant increase (35%, $p \leq 0.05$) in anti-KLH antibody production compared with controls. Delayed-type hypersensitivity response, measured as the mean footpad swelling, was significantly reduced at both 30 and 300 ppm (40% and 43%, $p \leq 0.05$) (See Table 1). Absolute spleen and liver weights were significantly increased at the highest concentration (almost 2-fold higher spleen weight and 19% higher liver weight; $p \leq 0.05$); body and thymus weights were not affected. There were no histopathological differences among the groups. The LOAEL from this study is 30 ppm (3 mg/kg-day) based on decreased cell-mediated immunity (delayed-type hypersensitivity response); the NOAEL is 3 ppm or 0.3 mg/kg-day.

Table 1. Significant effects of prenatal and postnatal exposure to 2,4-DCP on immune parameters		
2,4-DCP (ppm)	Anti-KLH antibody production (mean \pm SE absorbance at 405 nm)	Delayed-type hypersensitivity (mean \pm SE mm footpad swelling)
0	1.24 \pm 0.10	1.10 \pm 0.13
3	1.30 \pm 0.10	0.85 \pm 0.11
30	1.39 \pm 0.10	0.67 \pm 0.11 ^a
300	1.68 \pm 0.08 ^a	0.63 \pm 0.11 ^a
^a Significantly different by analysis of variance and least-square means comparison.		

Source: Exon et al., 1984; Exon and Koller, 1985.

Chronic Exposure — In the chronic NTP studies, F344/N rats and B6C3F1 mice (50 animals/sex/dose) were administered 2,4-DCP (>99% pure) in feed for 103 weeks (NTP, 1989). Dietary concentrations of 0, 5000 or 10,000 ppm were given to male rats and mice of both sexes; female rats were given concentrations of 0, 2500 or 5000 ppm. The authors estimated the doses of 2,4-DCP to be 210 or 440 mg/kg-day for male rats, 120 or 250 mg/kg-day for female rats, 800 or 1300 mg/kg-day for male mice and 430 or 820 mg/kg-day for female mice. Clinical observations were conducted twice daily, while body weight was measured weekly through week 13 and then monthly thereafter. Food consumption was measured monthly. After 103 weeks of dosing, all animals were sacrificed and necropsied. Histopathology of a comprehensive set of tissues (>40) was evaluated in control and high-dose animals; histopathology of low-dose animals was limited to the liver, nose, pituitary and thyroid of male rats; adrenal glands, lymph nodes, pancreas and spleen of female rats; liver, prostate, spleen and tarsal joints for male mice; and uterus for female mice.

Survival was not affected by treatment in rats of either sex, and there were no clinical signs of toxicity (NTP, 1989). Mean body weights in high-dose male and female rats were generally lower than those of controls (5-12%) beginning in week 3 (males) or week 31 (females), but statistical comparisons were not reported, nor were estimates of variability or individual body weight data that would permit statistical comparisons. Food consumption was significantly lower than controls (5-6%, $p < 0.05$ based on t-tests conducted for this review) in males of both treatment groups and in high-dose females. Reductions in body weight predated differences in food consumption, indicating a toxic, rather than an organoleptic effect. The only nonneoplastic lesion that was significantly increased over controls was multifocal degeneration

of the nasal epithelium in male rats (25/45, 38/48, 42/46); the increases were significant at both doses ($p < 0.05$). This study identifies a LOAEL of 210 mg/kg-day for nasal lesions in male rats; no NOAEL was identified.

There were no compound-related increases in the incidence of any neoplastic lesions in rats (NTP, 1989). The incidence of mononuclear cell leukemia was significantly decreased in dosed male rats relative to that in controls (control, 31/50; low dose, 17/50; high dose, 17/50).

In mice, treatment did not affect survival, nor were there any clinical signs of toxicity (NTP, 1989). Mean body weights of high-dose male mice and both dosed groups of female mice were generally lower than those of controls, although statistical comparisons were not provided. In high-dose males, body weight decrements occurred between weeks 25 and 86 and ranged from 3-9% in magnitude. In low-dose females, body weight decrements began in week 34 and ranged from 5-11%; in high-dose females, the reduction from control values increased over the course of the study and the mean terminal body weight was 17% below controls. Average food consumption was significantly lower than controls in high dose mice of both sexes (22% and 15%, for males and females, respectively; $p < 0.05$ based on t-tests conducted for this review). However, body weight decrements preceded reductions in food consumption temporally, indicating that palatability of the diet was not an issue. A dose-related increase in the incidence of syncytial alteration of hepatocytes was observed in dosed male mice (11/50; 33/49; 42/48; $p < 0.01$ at both doses based on Fisher's exact tests conducted for this review). This effect was also observed in the subchronic toxicity study with male mice, in which other evidence of liver toxicity (hepatocellular necrosis) was also observed. A LOAEL of 800 mg/kg-day was identified based on liver lesions (syncytial alteration of hepatocytes) in male mice and no NOAEL was identified.

As with rats, treatment did not result in a significantly increased incidence of any neoplastic lesion in mice (NTP, 1989). There was a marginally significant dose-related trend ($p = 0.037$) in the incidence of squamous papilloma or carcinoma of the forestomach in male mice (0/50, 0/50, 3/50), but pairwise comparison did not indicate a significant increase at the high dose ($p = 0.121$). The authors did not consider this increase to be treatment-related, as there was a negative trend for female mice and 2,4-DCP treatment did not increase the incidence of forestomach hyperplasia in the male mice. The incidence of malignant lymphomas was decreased in high-dose female mice (4/50) relative to controls (12/50). Under the conditions of these 2-year feeding studies, there was no evidence of carcinogenic activity for rats and mice fed 2,4-DCP.

In an oral carcinogenicity study, groups of Sprague-Dawley rats received both pre- and postnatal exposure to 2,4-DCP (99% pure) at concentrations of 0, 3, 30 or 300 ppm in drinking water (0, 0.45, 4.5 or 45 mg/kg-day²) for up to 24 months (Exon and Koller, 1985). Groups of 13 female rats were exposed from weaning through breeding at 90 days of age and until parturition. Offspring (groups of about 24/sex) were then given the test compound in drinking water from weaning until death or 24 months of age. Daily clinical observations were conducted, body weights of offspring were measured monthly and blood samples for hematology

² Based on default values for body weight and water consumption (U.S. EPA, 1988).

(erythrocyte and leukocyte counts, Hb, mean corpuscular volume [MCV] and packed-cell volume) were collected bimonthly. Necropsies were performed on tumor-bearing or moribund animals (and, presumably, survivors sacrificed at study termination), including histologic examination of the lung, heart, liver, spleen, kidney, adrenal, intestine, stomach, urinary bladder, brain, spinal cord, muscle and any tumors. The authors did not report any information on body weights or clinical signs of toxicity for the carcinogenicity study. When data from males and females exposed to 300 ppm 2,4-DCP for 14 months were combined, both erythrocyte count and Hb content were significantly increased (9% and 16%, respectively; $p \leq 0.05$). No other hematology data were reported or discussed. The toxicological significance of this finding is uncertain in the absence of data from other exposure levels and/or time periods; however, more pronounced hematological effects (bone marrow atrophy) were observed in female rats exposed to higher doses (750 mg/kg-day, NTP, 1989). Because this study was aimed at assessing the carcinogenicity of 2,4-DCP and no data on nonneoplastic findings (other than the single hematology measurements) were reported, effect levels were not derived for this study.

2,4-DCP administration had no effect on the incidence, latency or types of tumors relative to untreated controls (Exon and Koller, 1985). In a cocarcinogenicity study conducted simultaneously, tumor incidences in rats treated prenatally with the carcinogen ethylnitrosourea (ENU) and exposed to 2,4-DCP (either prenatally, postnatally or both) were not different from ENU-only treated rats. However, the authors noted that the group treated only with ENU had an unusually low incidence of tumors, potentially confounding the results of the cocarcinogenicity study with 2,4-DCP.

Kobayashi et al. (1972) evaluated the toxicity of 2,4-DCP in ICR mice fed the compound in the diet for 6 months. The study was published in Japanese and was not translated for this review; the summary contained herein is based on the English abstract and tables. The purity of the compound was not specified in the abstract. Groups of seven mice were fed concentrations of 0, 0.02%, 0.05%, 0.1% or 0.2% 2,4-DCP in the diet. From the available data, it appears that the high concentration group began treatment approximately 3 weeks later than the other groups; this is not discussed in the abstract. While the abstract does not specify the toxicological endpoints examined, the tables indicate that hematology (erythrocyte and leukocyte count), liver function (AST or ALT) and organ weights (liver, kidney, spleen and heart) were assessed in all treatment groups and histopathology evaluations (liver, kidney, spleen, heart and adrenal glands) were performed on controls and animals in the 0.1% and 0.2% groups.

Reuber (1983) reviewed all available studies related to the carcinogenicity of the phenoxy herbicide 2,4-dichlorophenoxy acetic acid (2,4-D) and its primary metabolite, 2,4-dichlorophenol (2,4-DCP). This report indicated that while 2,4-D was carcinogenic to both male and female rats, 2,4-DCP only demonstrated promoter activity in mouse skin cancer studies.

Based on measured body weights and food consumption rates, the authors estimated the doses to be 0, 45, 100 and 230 mg/kg-day. The authors reported that there were no effects on behavior. Both the abstract and the tabulated data on body weight, food consumption, hematology, liver function and organ weights (liver, kidney) indicated no effect of 2,4-DCP treatment. The authors reported that the histologic examinations showed "slight unfavorable" effects on the liver (the table reports these as "small round cell infiltration, swelling of hepatic

cells, unequal size of hepatic cells and dark cells”) at the highest dose; however, the numbers of affected animals were small (1 or 2 in a group of 7), so the toxicological significance is difficult to assess. The authors considered the 100 mg/kg-day dose to be a NOEL in mice. However, due to the small numbers of animals used in this study and the lack of a full translation, it was not considered appropriate to assign effect levels based on these data.

Reproductive/Developmental Studies — Aoyama et al. (2005) conducted a two-generation reproductive toxicity study in Wistar-Hannover rats exposed to 2,4-DCP via the diet. Groups of 24 rats/sex/group (aged 5 weeks) were given 2,4-DCP (99.7% pure) at concentrations of 0, 500, 2000 or 8000 ppm for 10 pre-mating weeks and during mating, gestation and lactation. Pregnant dams were allowed to give birth and on postnatal day (PND) 4, the litters were culled to 8 pups (4/sex when possible). On PND 21, groups of 24 male and female weanlings (1 male and 1 female from each litter) were selected to become F1 parents. F0 parents and any weanlings not selected to be F1 parents were necropsied at this time. F1 parents were treated in the same manner as the F0 parents with dietary 2,4-DCP during 10 pre-mating weeks and through lactation of the F2 pups. Upon weaning of the F2 generation, F1 parents and F2 pups were sacrificed and necropsied.

Clinical observations of parental animals were conducted daily and body weights and food consumption measured weekly (Aoyama et al., 2005). Female estrous cyclicity was evaluated by vaginal smear for 2 weeks prior to mating. Upon parturition, fertility and gestation parameters were recorded and the number and sex of live pups noted. Pups were weighed on PND 0, 4, 7, 14 and 21. Developmental milestones (pinna unfolding, tooth eruption, eye opening) were recorded in both generations. In addition, age at preputial or vaginal opening was evaluated in F1 pups selected to be parents, while anogenital distance on PND 4 was recorded in F2 pups. At necropsy (after weaning of pups for parental animals and at weaning for F2 pups), the number of uterine implantation sites was noted in female parents and sperm count and motility were recorded in male parents. The following organs were examined histologically in parental animals: liver, kidneys, pituitary, reproductive organs of both sexes and mammary glands. In F2 pups, the brain, thymus, spleen and uterus were weighed, but not examined histologically. Levels of pituitary and ovarian hormones (FSH, LH, prolactin, 17 β -estradiol and progesterone) were measured in F1 parent females upon necropsy.

Using mean body weight and food consumption values, the authors estimated doses of 33.4, 134 and 543 mg/kg-day for males and 49.1, 194 and 768 mg/kg for females (Aoyama et al., 2005). Clinical signs of toxicity (soiled fur in the abdominal/genital regions) were apparent in the highest dose group. At the highest dose, food consumption and body weight were significantly ($p \leq 0.01$) lower in both males and females of the F0 generation and in parental females of the F1 generation throughout most of pre-mating, gestation and lactation (data presented graphically). Females exposed at 2000 ppm also exhibited occasional statistically significant (p -value and data not reported) reductions in food consumption and/or body weight (during pre-mating in the F0 generation and during gestation in the F1 generation).

The mean number of implantation sites was slightly decreased in a dose-dependent fashion in both F0 and F1 parents, but was statistically significant ($p \leq 0.05$) only at the high dose in the F1 generation (10.2 vs. 12.7 in controls; Aoyama et al., 2005). In addition, the number of

live pups appeared to decrease with dose in both generations, but there were no statistically significant reductions. The age at preputial separation was significantly ($p \leq 0.05$) longer (42.2 days vs. 41.2 days in controls) in high-dose parental males of the F1 generation; however, the authors attributed this effect to reduced body weight. In contrast, despite lower body weight in high-dose females (F1), the age at vaginal opening was accelerated, albeit nonsignificantly (31.5 days vs. 32.2 days in controls). Neither sperm parameters nor female hormone concentrations were affected by treatment. Among high-dose parents, significant increases in relative organ weight (kidneys, testes) occurred in the absence of absolute organ weight changes and were attributable to decreased body weight. No other organ weight changes occurred in a dose-dependent manner and/or across generations.

Birth weight of pups was not affected by treatment (Aoyama et al., 2005). Pups of high-dose parents of both generations had reduced body weights (compared with controls) beginning on PND 7 and continuing through weaning. As dams treated at this dose (both generations) had significantly reduced food consumption and body weight, the reductions in pup weight are not unexpected. At the highest dose, eye opening was significantly ($p \leq 0.01$) delayed in pups of both generations and both sexes (50.5 to 65.3% of pups with eyes open on PND 14 vs. 89.1 to 94.6% of controls). Other developmental parameters were not affected by treatment. Upon necropsy of weanlings, there were significant reductions in absolute weights of brain, thymus and spleen in the high-dose group; however, these were attributable to reduced body weight. In contrast, the uterus weight of both F1 and F2 females was significantly increased at the high dose (42% and 20%, respectively; $p \leq 0.01$) and at the mid-dose in F1 females (25%, $p \leq 0.05$). Histologic examination of selected uteri of high-dose weanlings in the F2 generation indicated increased epithelial cell height (7/10 vs. 1/10 controls).

The authors indicated that the NOAEL for parental toxicity was 500 ppm and called the 2000 ppm concentration the “minimum toxic dose” based on reduced food consumption and body weight in parental females. Palatability of the treatment diet was not likely the cause of reduced weight. There were no reductions in food consumption or body weight in F0 males, indicating that this group consumed the treatment compound readily. Further, body weight reductions in high-dose F1 females preceded reductions in food consumption, indicating a toxic effect rather than an organoleptic effect. Body weight reductions have been reported in other studies (NTP, 1989), including a gavage study (Rodwell et al., 1989). Thus, the 2000 ppm concentration (194 mg/kg-day in females) is considered a minimal LOAEL based on reduced body weight and the 500 ppm concentration (49.1 mg/kg-day in females) is the NOAEL for parental toxicity.

For reproductive endpoints, the authors identified the 8000 ppm concentration (768 mg/kg-day in females) as a toxic dose based on reduced number of implantation sites in F1 parental females, increased uterine weight in F1 and F2 weanlings and accelerated sexual maturation of F1 females. In addition, eye opening was significantly delayed at this dose. The 2000 ppm concentration (194 mg/kg-day in females) is a NOAEL for reproductive effects.

Exon et al. (1984; Exon and Koller, 1985) evaluated a limited number of reproductive parameters in a study using female Sprague-Dawley rats exposed to 2,4-DCP (99% pure) from 3 weeks of age, through breeding with untreated males and until parturition. This information was

collected in conjunction with the immunotoxicity study on the progeny of exposed rats (see above). Groups of 13 rats were exposed via drinking water to concentrations of 3, 30 or 300 ppm 2,4-DCP (estimated to result in doses of 0.45, 4.5 and 45 mg/kg-day³). Percent conception, litter size, percent stillborn, birth and weaning weight and survival to weaning were evaluated. The authors reported that there were no treatment-related effects on the dams. Although the text indicated that the percent stillborn tended to be greater in the treatment groups, the increases were not statistically significant (2% in the high- and mid-concentration groups, 1% in the low concentration group and 0 in controls). The average litter size was smaller in the high-concentration group (6.3 ± 1.6) compared with controls (9.8 ± 1.3), but the difference was not statistically significant⁴. Other parameters were not affected by treatment. Due to the limited number of parameters assessed in this study, effect levels were not identified.

A two-generation rat reproductive study was conducted to investigate potential endocrine-mediated effects. One of the herbicides, 2,4-dichlorophenol, was administered at 0, 500, 2000 and 8000 ppm in the diet. Increased uterine weights were observed in both F1 and F2 females. A reduced number of implantation sites and reduced live births in F1 parental animals were observed at 2000 ppm and higher exposure levels (Yamasaki et al., 2005).

To understand the structural basis for estrogenic activity, Tarasaka et al. (2006) performed DNA-micro array assay of several structurally similar chemicals, including 2,4-dichlorophenol. This assay demonstrated the estrogenic activity of 2,4-dichlorophenol by down regulating enzymes and signaling pathway compared to chemicals with high levels of estrogenic activity.

Using prostate cancer cell lines, Kim et al. (2005) evaluated the endocrine disrupting activity of 2,4-dichlorophenoxyacetic acid and 2,4-dichlorophenol. These chemicals did not show any androgenic activity. However, co-exposure with 5alpha-dihydroxytestosterone synergistic androgenic activity was demonstrated in this assay.

Rodwell et al. (1989; Dow Chemical Co., 1983) evaluated the developmental toxicity of 2,4-DCP in F344 rats. Groups of 34 sperm-positive female rats were given 2,4-DCP (99.2% pure, in corn oil) via gavage on gestation days (GD) 6-15. Doses of 0, 200, 375 or 750 mg/kg-day were administered. Maternal body weights were recorded on GD 0, 6, 10, 12, 15 and 20. On GD 20, dams were sacrificed by carbon dioxide and subjected to caesarean section. The uterus was weighed and examined for number and location of viable and nonviable fetuses and early and late resorptions. Number and location of *corpora lutea* were recorded. Fetuses were weighed, measured, sexed and examined externally; half were then prepared for soft tissue examination and the remainder prepared for skeletal examination.

Four dams in the high-dose group died during treatment (Rodwell et al., 1989; Dow Chemical Co., 1983). Clinical observations in the high-dose group included red staining around the eyes, nares and mouth; abdominal alopecia; and respiratory rales. While incidences were not

³ Based on default values for body weight and water consumption (U.S. EPA, 1988).

⁴ Exon and Koller (1985) reported that the decrease in litter size was statistically significant at $p \leq 0.10$; Exon et al. (1984) reported that it was not statistically significant. The latter interpretation was accepted here given the relatively high critical value (0.10) used to assess significance in the 1985 paper.

reported, the authors indicated that these were observed in a majority of animals at this dose and in some animals exposed to 375 mg/kg-day. A few high-dose rats also displayed ataxia, prostration and reduced activity. In addition, yellow staining of fur in the urogenital area occurred in all treated groups (incidence not provided, but reported to increase with dose). Maternal body weight gain during treatment was significantly ($p<0.05$) lower than control weight gain in all dose groups (82%, 77%, and 32% of controls during GD 6-15 at the low, mid-, and high doses); further, the body weight decrements persisted after exposure ceased, although the decrease was statistically significant only at the high dose. There were no significant differences in reproductive or teratogenic parameters. Significantly ($p<0.05$) increased incidences of unossified sternebrae (4/22 litters vs. 0/27 litters in controls) and delayed ossification of vertebral arches (6/22 litters vs. 0/27 litters in controls) were observed in the high-dose group. The high dose in this study was a Frank Effect Level (FEL) due to 4 maternal deaths. The LOAEL for maternal toxicity was 200 mg/kg-day, based on clinical signs and decreased body weight gain during treatment; no NOAEL can be identified for maternal toxicity. Developmental effects (increased skeletal variations) were observed at the maternal FEL of 750 mg/kg-day, but not at lower doses; thus, the NOAEL for developmental toxicity was 375 mg/kg-day.

Inhalation Exposure

The literature search identified no studies regarding toxicity of 2,4-DCP in animals following inhalation exposure. Similarly, the available reviews (U.S. EPA, 1987a,b; ATSDR, 1999; IARC, 1986, 1999; WHO, 1989) did not identify any inhalation toxicity studies of this compound.

Other Studies

Acute Toxicity — Borzelleca et al. (1985a,b) reported oral LD50 values of 1276 mg/kg and 1352 mg/kg for male and female CD1-ICR mice observed for up to 14 days. Kobiyashi et al. (1972) calculated oral LD50 values of 1600 mg/kg in ICR mice (same value for males and females) and 3670 and 4500 mg/kg in male and female Sprague-Dawley rats (respectively) observed for up to 10 days.

Dermal Carcinogenicity Studies — Boutwell and Bosch (1959) examined the ability of 2,4-DCP to act as a complete carcinogen on the skin of mice and to promote skin tumors following a single initiating dose of dimethylbenzanthracene (DMBA). In the study for complete carcinogenesis, a group of 23 female Sutter mice (2-3 months of age) was treated with a topical application of 25 μ L of 20% 2,4-DCP in benzene applied twice weekly to the back of each mouse for 24 weeks. Of the 23 mice, 16 (70%) survived to 24 weeks, at which time 75% of the survivors (12 mice) had papillomas and 6% (1 mouse) had a carcinoma. The average number of papillomas per mouse was 1.62. At 39 weeks, 62% of surviving mice (number not reported) had carcinomas. There was no control group maintained concurrently in this experiment. The absence of a similarly-treated concurrent control group, along with the significant mortality (30%) in this short-duration study, limits the usefulness of these data.

In the promotion experiment, a group of 33 female Sutter mice was treated with an initial topical application of 75 μ L of 0.3% DMBA in benzene, followed by 25 μ L of 20% 2,4-DCP in benzene applied twice weekly to the back of each mouse for 15 weeks (Boutwell and Bosch, 1959). At 15 weeks, 27 of 33 (82%) 2,4-DCP-treated mice survived, compared to 15 of 20 (75%) in the control group. Based on information in the publication, the control group was treated with an initiating dose of DMBA; it is not clear whether the controls received applications of vehicle (benzene) on the promotion schedule⁵. At the end of treatment, the incidence of papillomas was significantly increased ($p < 0.01$) in 2,4-DCP-treated surviving mice (13/27), compared with controls (1/15). The average number of papillomas per mouse was 1.07 vs. 0.07 in controls. Three of 27 (11%) 2,4-DCP-treated survivors had carcinomas, compared to no carcinomas in the initiator only group; however this difference was not statistically significant ($p > 0.05$).

U.S. EPA (1980) criticized several aspects of this study, including the failure to histologically confirm tumor types and the use of creosote-coated wooden cages to house the animals. The use of creosote-treated cages could not be confirmed; the publication indicates that “screen-bottomed metal cages” were used. In addition, U.S. EPA (1980) noted that the high concentration of 2,4-DCP (20% in benzene) applied to the skin may have caused physical abrasion of the skin. 2,4-DCP is known to be corrosive to the skin (HSDB, 2006) and this irritant property may have enhanced the papillomatous response in both studies. It is also important to note that there was significant mortality in the control group of the promotion study (25%) despite the short duration of the study (15 weeks). The reason for this high rate of mortality or that of the 2,4-DCP treated group in the complete carcinogenicity study (30%), was not discussed by the authors.

Genotoxicity — Genotoxicity testing of 2,4-DCP has generally given negative results. Positive results in clastogenicity testing have often been associated with cell toxicity. 2,4-DCP produced no increases in revertant colonies in *Salmonella typhimurium* strains TA98, TA100 or TA1537 with or without exogenous metabolic activation (Haworth et al., 1983; Rasanen et al., 1977). The mutagenic effect of 2,4-DCP in *Salmonella typhimurium* strain TA1535 was initially considered to be equivocal in the presence of hamster liver S9 metabolic activation (Haworth et al., 1983). However, a reevaluation of the data resulted in a determination that the response was negative (Zeiger, 1990). 2,4-DCP significantly increased trifluorothymidine (Tft) resistance in the mouse L5178Y assay at concentrations of 30-60 μ g/mL when tested without metabolic activation (Myhr et al., 1990). 2,4-DCP was cytotoxic to V79 Chinese hamster cells, but did not induce 6-thioguanine-resistant mutants when tested at concentrations up to 50 μ g/mL without exogenous metabolic activation (Jansson and Jansson, 1986). In a cell-mediated test (wherein metabolic activation was provided by co-cultured cells), 2,4-DCP was weakly mutagenic at concentrations that were also cytotoxic to V79 cells (cell survival 41-54% of controls; Fiskesjo, 1988).

In cultured CHO cells, 2,4-DCP did not induce chromosomal aberrations at concentrations up to 75 μ g/mL (0.51 mM) without S9 (8-hour treatment) and at up to 150 μ g/mL

⁵ In other experiments using DMBA followed by promotion testing of agents dissolved in benzene, the control is reported as a “benzene control”; in this experiment, it is not.

(1.02 mM) with S9 (2-hour treatment) (Anderson et al., 1990). In another CHO cell assay, chromosomal aberrations developed in a significant percentage of cells both with and without activation (Hilliard et al., 1998). Aberrations were observed in 14% of cells following a 3-hour treatment at 1.4 mM without S9 and in 14.5% cells treated at 0.6 mM with S9, compared with 1.5% of control cells (Hilliard et al., 1998). Cell survival was reduced in both of these cases (27% and 54% of controls, respectively), raising the possibility that the aberrations were related to toxicity (Hilliard et al., 1998). Testing of human TK6 lymphoblasts for chromosomal aberrations resulted in an equivocal (nonsignificant) increase (5% of cells with aberrations, compared with 0% in controls) for 2,4-DCP at 0.8 mM, a concentration that gave 59% survival compared with controls. 2,4-DCP increased the frequency of sister chromatid exchanges (SCEs) both in the presence and absence of S9 (Anderson et al., 1990).

The cytogenic effect of 2,4-dichlorophenol was studied in bone marrow, germ cells and spermhead abnormalities in mice treated intraperitoneally at 1/10, 1/5, 1/2 dose levels (Amer and Aly, 2001). This report demonstrated weaker genotoxic effects as indicated by lower percentage of induced chromosomal aberrations and spermhead abnormalities.

2,4-DCP induced error-prone DNA repair (*umu*-test) in *S. typhimurium* cells when tested without metabolic activation (Ono et al., 1992). In an *in vitro* alkaline elution/rat hepatocyte genotoxicity assay, 2,4-DCP produced evidence of DNA damage; however, cytotoxicity tests showed significant toxicity at concentrations resulting in DNA damage and the authors suggested that DNA effects likely resulted from activation of degradative endonucleases in dead or dying cells (Storer et al., 1996). 2,4-DCP did not induce unscheduled DNA synthesis in primary cultures of rat hepatocytes at a concentration of 50 nmol/mL (Probst et al., 1981). In a prophage-induction assay, 2,4-DCP did not induce DNA damage in *E. coli* at doses up to 480 μ mol (DeMarini et al., 1990). 2,4-DCP produced negative results in an *in vivo-in vitro* mouse hepatocyte replicative DNA synthesis (RDS) test (Miyagawa et al., 1995).

Genotoxicity testing of 2,4-DCP is complicated by the fact that this compound uncouples oxidative phosphorylation, leading to depletion of cellular energy supplies. Mitsuda et al. (1963) reported that a concentration of 42 μ M 2,4-DCP caused 50% inhibition of ATP production in rat liver mitochondria *in vitro*; in fact, 2,4-DCP was the most potent of the mono- and dichlorophenols tested in this study. Disturbances in energy production may be responsible for the cellular toxicity observed in genotoxicity assays, many of which were conducted at high concentrations. Cell toxicity can lead to false-positive findings, especially in assays for clastogenicity, because DNA damage commonly occurs in apoptotic and necrotic cells (Storer et al., 1996; Hilliard et al., 1998).

DERIVATION OF A PROVISIONAL SUBCHRONIC ORAL RfD VALUE FOR 2,4-DICHLOROPHENOL

A chronic oral RfD of 0.003 mg/kg-day based on an immunotoxicity study (Exon et al., 1984) is available on IRIS. Several oral toxicity studies, including the immunotoxicity study, are available for use in deriving a provisional subchronic oral RfD for 2,4-DCP. Table 2 summarizes the findings of those studies in which a NOAEL and/or LOAEL was identified. As

Table 2. Summary of Oral Noncancer Dose-Response Information

Species	Sex	Doses (mg/kg-day)	Exposure Duration	NOAEL (mg/kg-day)	LOAEL (mg/kg-day)	Responses	Comments	Reference
Chronic Studies								
Rats	M/F	0, 210, 440 (M) 0, 120, 250 (F)	103 weeks	NA	210	Multifocal degeneration of nasal epithelium		NTP, 1989
Mice	M/F	0, 800, 1300 (M) 0, 430, 820 (F)	103 weeks	NA	800	Syncytial alteration of hepatocytes in males		NTP, 1989
Subchronic Studies								
Rats	M/F	0, 160, 310, 675, 1373 (M) 0, 182, 338, 750, 1376, 2795 (F)	13 weeks	338	750	Bone marrow atrophy in females		NTP, 1989
Mice	M/F	0, 782, 1533, 1627, 2960, 6805 (M) 0, 973, 2438, 3305, 3913, 8911 (F)	13 weeks	NA	782	Mild hepatocellular necrosis		NTP, 1989
Rats	M/F	0, 0.3, 3, 30	Pre- and postnatal	0.3	3	Decreased cell-mediated immunity	Immunotoxicity study. Exposure prenatally, through lactation and via drinking water from weaning until 13 weeks of age	Exon and Koller, 1985
Reproductive/Developmental Studies								
Rats	F	0, 200, 375, 750	GD 6-15	NA (maternal) 375 (developmental)	200 (maternal) 750 (developmental)	Maternal toxicity: clinical signs, reduced body weight gain Developmental effects: increased incidence of skeletal variations		Rodwell et al., 1989; Dow Chemical Co., 1983

Table 2. Summary of Oral Noncancer Dose-Response Information

Species	Sex	Doses (mg/kg-day)	Exposure Duration	NOAEL (mg/kg-day)	LOAEL (mg/kg-day)	Responses	Comments	Reference
Rats	M/F	0, 33.4, 134, 543 (M) 0, 49.1, 194, 768 (F)	10 weeks premating , through gestation and lactation	49.1 (parental) 194 (reproductive)	194 (parental) 768 (reproductive)	Parental toxicity: transient reductions in body weight of dams Reproductive effects: reduced number implantation sites (F1 dams), delayed eye opening and effects on uterus in offspring	Two-generation reproductive toxicity study	Aoyama et al., 2005

the table indicates, the LOAEL for immunotoxicity (3 mg/kg-day; Exon et al., 1984) is much lower (almost 2 orders of magnitude) than LOAELs for other endpoints. This study was thus selected as the basis for the subchronic p-RfD. The critical effect in this study is a decrease in cell-mediated immunity, as measured by decreased footpad swelling. The data selected for modeling are shown in Table 3. Models for continuous variables in U.S. EPA's Benchmark Dose Software (BMDS) were fit to the cell-mediated immunity data in accordance with U.S. EPA (2000) methodology. A default benchmark response (BMR) of one standard deviation from the control mean was used. Appendix A contains details of the modeling and a plot of the best fitting model.

Table 3. Cell-mediated Immunity Data as Modeled					
2,4-DCP (ppm)	2,4-DCP (mg/kg-day)	No. of rats	Delayed-type hypersensitivity (mm footpad swelling)		
			Mean	SE	SD
0	0	10	1.10	0.13	0.41
3	0.3	10	0.85	0.11	0.35
30	3.0	10	0.67	0.11	0.35
300	30	10	0.63	0.11	0.35

Source: Exon et al. (1984; Exon and Koller, 1985)

The test for homogenous variance indicated that the homogenous variance model provided adequate fit to the variance data. Using the homogenous variance model, the linear model did not provide adequate fit to the means, so the remaining models were applied; however, none provided adequate fit. In order to try to achieve model fit, the high dose group was dropped from the analysis. Using the reduced data set, the homogenous variance model again provided adequate fit to the variance data, and the linear model provided adequate fit to the means. BMD and BMDL predictions from the modeling of the reduced data set were 3.21 and 1.84 mg/kg-day, respectively. The BMDL from this study (2 mg/kg-day) was thus selected as the point-of-departure (POD) for derivation of the subchronic p-RfD.

The **subchronic p-RfD of 0.02 mg/kg-day** is calculated as the BMDL of 2 mg/kg-day divided by an uncertainty factor of 100, as shown below:

$$\begin{aligned}
 \text{Subchronic p-RfD} &= \text{BMDL/UF} \\
 &= 2 \text{ mg/kg-d} / 100 \\
 &= \mathbf{0.02 \text{ mg/kg-day}}
 \end{aligned}$$

An interspecies uncertainty factor of 10 was applied and another 10-fold uncertainty factor was used for protection of sensitive individuals. A factor of 1 for duration was applied, as the exposure included prenatal and lactational exposure, followed by exposure via drinking water to 13 weeks of age. Since a BMDL was used as the POD, no adjustment for use of a LOAEL was necessary. No database uncertainty factor was used; the toxicological database for 2,4-DCP contains chronic studies in two species, several subchronic studies in rats and mice, two developmental toxicity studies in rats, and a multigeneration reproductive toxicity study in rats. The database lacks a neurotoxicity study; however, existing studies suggest that neurotoxic effects occur only at high doses.

Confidence in the principal study (Exon et al., 1984) is medium because, despite the investigation of sensitive endpoints, the sample sizes were relatively small (10 per dose). Confidence in the database is high because the database includes well-conducted chronic studies in two species, several subchronic studies in rats and mice, two developmental toxicity studies in rats, and a multigeneration reproductive toxicity study in rats. Medium to high confidence in the subchronic p-RfD follows.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION p-RfC VALUES FOR 2,4-DICHLOROPHENOL

There are no inhalation studies available for use in developing subchronic and/or chronic provisional RfCs (p-RfC) for 2,4-DCP.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2,4-DICHLOROPHENOL

Weight-of-Evidence Classification

Under the 2005 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), 2,4-DCP is *not likely to be carcinogenic to humans via oral exposure*. There is *inadequate information to assess the carcinogenic potential* of 2,4-DCP to humans via inhalation exposure. There are no human data addressing the potential carcinogenicity of 2,4-DCP alone, either via oral or inhalation exposure. 2,4-DCP tested negative in adequate 2-year NTP dietary bioassays using both rats and mice. In addition, a second adequate chronic study in rats found no increase in tumor formation with chronic 2,4-DCP exposure (Exon and Koller, 1985). 2,4-DCP has not been tested for carcinogenicity via inhalation exposure. In an old publication where dermally-applied 2,4-DCP was tested both for complete carcinogenicity and as a promoter, an increased incidence of papillomas was observed (Boutwell and Bosch, 1959); however, there are a number of limitations that call into question these results, including: lack of control in the complete carcinogenicity study, high mortality in the control group for the promotion study and use of a potentially corrosive concentration of 2,4-DCP in the skin applications. Genotoxicity testing of 2,4-DCP has largely given negative responses; instances where positive responses were reported have often been associated with cytotoxicity.

Quantitative Estimates of Carcinogenic Risk

There are no appropriate human or animal data from which to derive an oral slope factor or inhalation unit risk for 2,4-DCP.

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**APPENDIX A. BENCHMARK DOSE MODELING OF CELL-MEDIATED IMMUNITY
(MEAN FOOTPAD SWELLING)
(EXON ET AL., 1984; EXON AND KOLLER, 1985)**

The model fitting procedure for continuous data is as follows. The simplest model (linear) is first applied to the data while assuming constant variance. If the data are consistent with the assumption of constant variance ($p \geq 0.1$), then the fit of the linear model to the means is evaluated. If the linear model adequately fits the means ($p \geq 0.1$), then it is selected as the model for BMD derivation. If the linear model does not adequately fit the means, then the more complex models are fit to the data while assuming constant variance. Among the models providing adequate fit to the means ($p \geq 0.1$), the one with the lowest AIC for the fitted model is selected for BMD derivation. If the test for constant variance is negative, the linear model is run again while applying the power model integrated into the BMDS to account for nonhomogenous variance. If the nonhomogenous variance model provides an adequate fit ($p \geq 0.1$) to the variance data, then the fit of the linear model to the means is evaluated. If the linear model does not provide adequate fit to the means while the nonhomogenous variance model is applied, then the polynomial, power and Hill models are fit to the data and evaluated while the variance model is applied. Among those providing adequate fit to the means ($p \geq 0.1$), the one with the lowest AIC for the fitted model is selected for BMD derivation. If the test for constant variance is negative and the nonhomogenous variance model does not provide an adequate fit to the variance data, then the data set is considered unsuitable for modeling.

Following the above procedure, continuous-variable models in the EPA BMDS (version 1.3.2) were fit to the data shown in Table 3 (page 23) for decreased cell-mediated immunity (as measured by mean footpad swelling) in rats. Using these data, the constant variance model provided adequate fit to the variance data. With the homogeneous variance model applied, the linear model did not provide an adequate fit to the means, as shown in Table A-1. Further, none of the remaining models provided adequate fit to the data (there were not enough dose groups to apply the Hill model). In order to achieve model fit, the high dose group was dropped from the analysis. With the reduced data set, the homogenous variance model again fit the variance data adequately. With the homogenous variance model applied, the linear model provided adequate fit to the means (Figure A-1). The BMDs and the 95% lower confidence limits (BMDLs) associated with a change of 1 standard deviation (SD) from the control were calculated using the linear model with homogenous variance model applied.

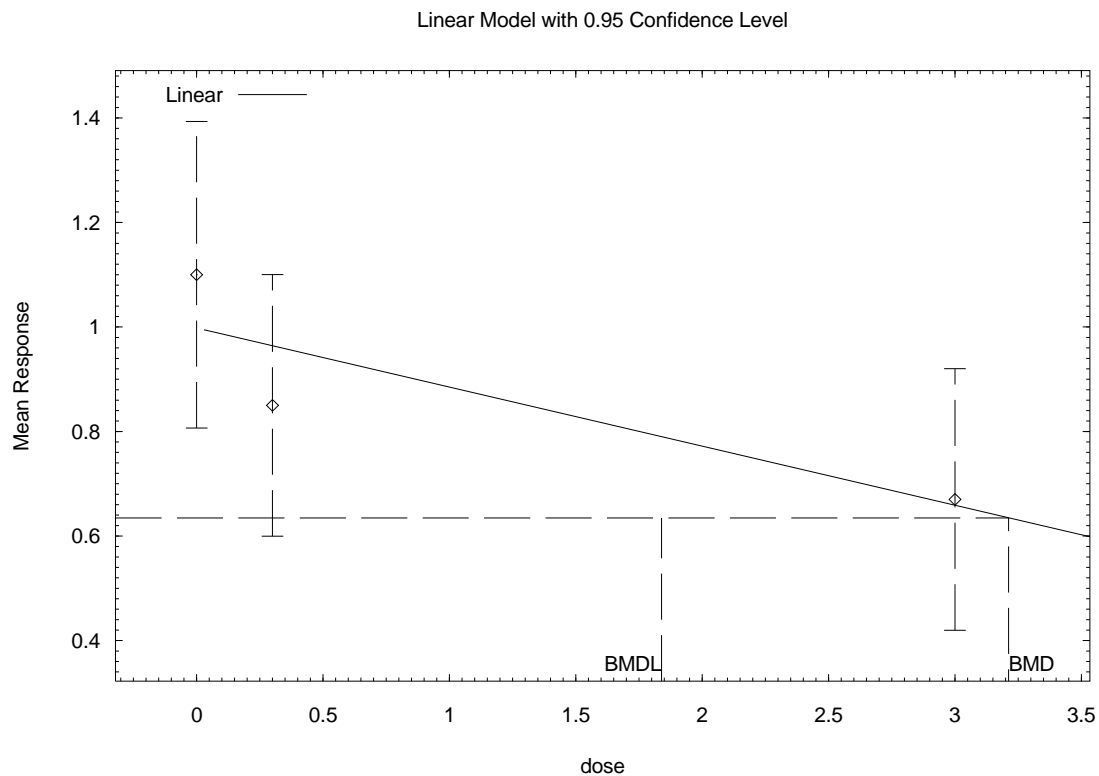
Table A-1. Model Predictions for Footpad Swelling in Rats Exposed to 2,4-DCP (Exon et al. 1984; Exon and Koller, 1985)				
Model	Variance <i>p</i>-value^a	Means <i>p</i>-value^a	BMD_{1sd} (mg/kg-day)	BMDL_{1sd} (mg/kg-day)
All dose groups				
Linear (constant variance)	0.9413	0.04207	40.30	21.74
Polynomial (constant variance) ^b	0.9413	0.01183	40.30	21.74
Power (constant variance) ^c	0.9413	0.01183	40.30	21.74
Hill (constant variance) ^c	NA ^d			
Without high dose group				
Linear (constant variance)	0.8418	0.1747	3.21	1.84

^aValues <0.10 fail to meet conventional goodness-of-fit criteria

^bCoefficients restricted to be negative; no adequate fit with any degree polynomial; 2-degree polynomial shown

^cPower restricted to ≥ 1

^dNA = not applicable (insufficient degrees of freedom available to fit this model)



12:36 01/09 2007

Figure A-1. Mean Footpad Swelling (mm) in Rats Exposed to 2,4-DCP (Reduced dataset) (Exon et al., 1984; Exon and Koller, 1985)

BMDs and BMDLs indicated are associated with a change of 1 SD from the control, and are in units of mg/kg-day.

From: <Nunes.Robert@epamail.epa.gov>
To: <txsmith@gw.dec.state.ny.us>
CC: <Allen.Burton@earthtech.com>, "Mike Spera"
<Michael.Spera@earthtech.com>...
Date: 3/17/2008 9:43 AM
Subject: Fw: Toxicity Value Request for the Onondaga Lake Site in NY
Attachments: pic01523.jpg; Onondaga Lake 1 - Sivak.pdf

Tracy - Can you please pass this on to OB&G? They can contact us if they have any questions.

Bob Nunes
New York Remediation Branch
Emergency and Remedial Response Division
US EPA Region II
290 Broadway, 20th Floor
New York, NY 10007-1866
Tel: (212) 637-4254
Fax: (212) 637-3966
Email: nunes.robert@epa.gov

----- Forwarded by Robert Nunes/R2/USEPA/US on 03/17/2008 09:36 AM -----

Michael
Sivak/R2/USEPA/U
S To
Robert Nunes/R2/USEPA/US@EPA
03/14/2008 07:02 cc
PM Chloe Metz/R2/USEPA/US@EPA
Subject
Fw: Toxicity Value Request for
the Onondaga Lake Site in NY

Bob,

Here's the first installment. I also sent a note to STSC to ask specifically about naphthalene. I will also ask for an estimate of when the PPRTVs that are in development will be completed. I'll let you know what I find out.

Michael Sivak
Technical Support Team
Program Support Branch

EPA Region 2 Superfund Program
sivak.michael@epa.gov
tel: 212.637.4310
fax: 212.637.3083

----- Forwarded by Michael Sivak/R2/USEPA/US on 03/14/2008 07:00 PM

SUPERFUND STSC

Sent by: Teresa
Shannon

To
Michael Sivak/R2/USEPA/US@EPA
cc

03/14/2008 10:52
AM

Subject
Re: Toxicity Value Request for
the Onondaga Lake Site in NY
(Document link: Michael Sivak)

(Embedded image moved to file: pic01523.jpg)

Hey Michael,

I am sending your response in pieces like you requested. Here is the first piece:

1,1,2,2-tetrachloroethane - PPRTV in development, can be sent upon completion
1,1,2-trichloroethane - Current PPRTV attached below, contains no values
1,1'-biphenyl - No information available
1,2,3-trichlorobenzene - PPRTV in development, can be sent upon completion
1,2,4-trichlorobenzene - OSF on Cal EPA; PPRTV in development, can be sent upon completion
1,2,4-trimethylbenzene - Current PPRTV attached below, contains no values
1,2-dichlorobenzene - RfC in '97 HEAST; PPRTV in development, can be sent upon completion
1,2-dichloroethane - RfD in Current PPRTV attached below; ATSDR has RfC
1,2-dichloropropane - ATSDR has RfD; Cal EPA and HEAST have OSF; Cal EPA

has IUR and we have a current PPRTV that contains no values
1,3,5-trimethylbenzene - PPRTV in development, can be sent upon completion
1,3-dichlorobenzene - PPRTV in development, can be sent upon completion
1,4-dichlorobenzene - no RfD info found; Cal EPA has OSF and IUR
1-phenyl-1-(2,4-dimethylphenyl)ethane - No information available
1-phenyl-1-(4-methylphenyl)ethane - No information available
2,4,6-trichlorophenol - IUR on IRIS; no RfC info found
2,4-dichlorophenol - Current PPRTV attached below, contains no values

If you have any questions about the first portion of our response,
please feel free to contact the Center. I'll keep the pieces coming!

Thanks and have a good weekend!

Teresa Shannon
STSC

(See attached file: Onondaga Lake 1 - Sivak.pdf)

Michael	
Sivak/R2/USEPA/	
US	To
	SUPERFUND STSC@EPA
03/06/2008	cc
12:05 PM	Robert Nunes/R2/USEPA/US@EPA
	Subject
	Toxicity Value Request for the
	Onondaga Lake Site in NY

Hi there!

I'm back! And with a super long list of chemicals for you. I didn't really know how to organize them, so I grouped them by the type of toxicity factor. I hope that's not too horrible. Please let me know if you'd like me to reorganize the list.

Thanks in advance for your help...

RfD (chronic unless otherwise noted): cobalt, mercury (subchronic), 4,4'-DDD, a-BHC, d-BHC, toxaphene, 1-phenyl-1-(2,4-dimethylphenyl)ethane, 1-phenyl-1-(4-methylphenyl)ethane, 2,6-dinitrotoluene, 2-nitroaniline, 2-nitrophenol, 3&4 methylphenol, 3,3'-dichlorobenzidine, 3-nitroaniline, 4,6-dinitro-2-methylphenol, 4-bromophenyl phenyl ether, 4-chloro-3-methylphenol, 4-chlorophenyl phenol ether, 4-methylphenol, 4-nitroaniline, 4-nitrophenol, acenaphthylene, benzo(ghi)perylene, carbazole, dibenzofuran, hexachlorobutadiene, N-nitro-di-N-propylamine, phenanthrene, 1,1,2,2-tetrachloroethane, 1,2,3-trichlorobenzene, 1,2,4-trimethylbenzene, 1,2-dichloroethane, 1,2-dichloropropane, 1,3,5-trimethylbenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene, 2-hexanone, chloroethane, tetrachloroethene, trichloroethene, p-isopropyltoluene, t-1,3-dichloropropene, ammonia, chloride, dodecane, sec-butylbenzene, sulfate.

RfC: antimony, barium, cadmium, cobalt, copper, cyanide, iron, nickel, selenium, thallium, vanadium, zinc, PCBs, 4,4'-DDD, 4,4'-DDT, aldrin, a-BHC, d-BHC, atrazine, dieldrin, endosulfan, endrin, heptachlor epoxide, toxaphene, 1-phenyl-1-(2,4-dimethylphenyl)ethane, 1-phenyl-1-(4-methylphenyl)ethane, 2,4,6-trichlorophenol, 2,4-dichlorophenol, 2,4-dimethylphenol, 2,4-dinitrophenol, 2,4-dinitrotoluene, 2,6-dinitrotoluene, 2-chlorophenol, 2-methylnaphthalene, 2-nitroaniline, 2-nitrophenol, 3&4 methylphenol, 3,3'-dichlorobenzidine, 3-nitroaniline, 4,6-dinitro-2-methylphenol, 4-bromophenyl phenyl ether, 4-chloro-3-methylphenol, 4-chlorophenyl phenol ether, 4-methylphenol, 4-nitroaniline, 4-nitrophenol, acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(ghi)perylene, bis(2-chloroethoxy)methane, bis(2-chloroethyl)ether, bis(2-ethylhexyl)phthalate, carbazole, chrysene, dibenz(ah)anthracene, dibenzofuran, fluoranthene, fluorene, hexachlorobenzene, hexachlorobutadiene, hexachloroethane, indeno(123-cd)pyrene, nitrobenzene, N-nitro-di-N-propylamine, pentachlorophenol, phenanthrene, phenol, pyrene, 1,1,2,2-tetrachloroethane, 1,1,2-trichloroethane, 1,2,3-trichlorobenzene, 1,2,4-trichlorobenzene, 1,2-dichlorobenzene, 1,2-dichloroethane, 1,3,5-trimethylbenzene, 1,3-dichlorobenzene, 2-hexanone, acetone, bromodichloromethane, carbon tetrachloride, chlorobenzene, chlorodibromomethane, chloroform, methylene chloride, p-isopropyltoluene, tetrachloroethene, trichloroethene, t-1,3-dichloropropene, 1,1'-biphenyl, ammonia, chloride, chlorine, dodecane, sec-butylbenzene, sulfate.

SForal: aluminum, cobalt, iron, nickel, vanadium, atrazine, endosulfan, endrin aldehyde, endrin ketone, 1-phenyl-1-(2,4-dimethylphenyl)ethane, 1-phenyl-1-(4-methylphenyl)ethane, 2,4-dinitrotoluene, 2,6-dinitrotoluene, 2-nitroaniline, 2-nitrophenol, 3-nitroaniline, 4,6-dinitro-2-methylphenol, 4-chloro-3-methylphenol, 4-chlorophenyl phenyl ether, 4-nitroaniline, carbazole, 1,2,3-trichlorobenzene, 1,2,4-trichlorobenzene, 1,2,4-trimethylbenzene, 1,2-dichloropropane,

1,3,5-trimethylbenzene, 1,4-dichlorobenzene, p-isopropyltoluene, chlorine, dodecane, nitrogen, sec-butylbenzene, sulfate

SFinhalation: aluminum, beryllium, cobalt, iron, nickel, vanadium, atrazine, endosulfan, endrin aldehyde, endrin ketone, 1-phenyl-1-(2,4-dimethylphenyl)ethane, 1-phenyl-1-(4-methylphenyl)ethane, 2,4,6-trichlorophenol, 2,4-dichlorophenol, 2,4-dimethylphenol, 2,4-dinitrophenol, 2,6-dinitrotoluene, 2-chlorophenol, 2-nitrophenol, 3&4 methylphenol, 3,3'-dichlorobenzidine, 3-nitroaniline, 4,6-dinitro-2-methylphenol, 4-chloro-3-methylphenol, 4-chlorophenyl phenol ether, 4-methylphenol, 4-nitroaniline, 4-nitrophenol, acenaphthene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, bis(2-chloroethoxy)methane, bis(2-chloroethyl)ether, bis(2-ethylhexyl)phthalate, carbazole, chrysene, dibenz(ah)anthracene, indeno(123-cd)pyrene, naphthalene, 1,2,3-trichlorobenzene, 1,2,4-trimethylbenzene, 1,2-dichloropropane, 1,3,5-trimethylbenzene, 1,4-dichlorobenzene, 2-hexanone, bromodichloromethane, carbon disulfide, chlorodibromomethane, xylenes, p-isopropyltoluene, toluene, t-1,3-dichloropropene, 1,1'-biphenyl, ammonia, chloride, chlorine, dodecane, sec-butylbenzene, sulfate

Michael Sivak
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fax: 212.637.3083



To: Robert Nunes/R2/USEPA/US@EPA
From: Michael Sivak/R2/USEPA/US
Date: 03/19/2008 12:00PM
cc: Chloe Metz/R2/USEPA/US@EPA
Subject: Fw: Toxicity Value Request for the Onondaga Lake Site in NY

Bob,

Please see the note below. The CalEPA IUR is recommended for naphthalene, which will influence how inhalation exposures are screening and evaluated. Please forward this to DEC and along to OBG and Honeywell. They will need to include this in the HHRA for HB/WBB and other sites.

Please let me know if you or anyone has any questions. Thanks!

Michael Sivak
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fax: 212.637.3083

Hi Michael,

This is the response that the chemical manager provided regarding the naphthalene IUR:

Inform the client that the best value available now is the CalEPA IUR. The client should use that value until the IRIS file is on line Thanks.

If you need anything else, please let me know. I am working on the second set of files to send to you. Thanks for your patience!

Teresa Shannon
STSC

Michael Sivak/R2/USEPA/US

SUPERFUND STSC

Hey Michael!

I received your request and have started researching your list of chemicals. I will be in touch as soon as I have a response put together for you.

Thanks!



Teresa Shannon
STSC

Michael Sivak/R2/USEPA/US

Hi there!

I'm back! And with a super long list of chemicals for you. I didn't really know how to organize them, so I grouped them by the type of toxicity factor. I hope that's not too horrible. Please let me know if you'd like me to reorganize the list.

Thanks in advance for your help...

RfD (chronic unless otherwise noted): cobalt, mercury (subchronic), 4,4'-DDD, a-BHC, d-BHC, toxaphene, 1-phenyl-1-(2,4-dimethylphenyl)ethane, 1-phenyl-1-(4-methylphenyl)ethane, 2,6-dinitrotoluene, 2-nitroaniline, 2-nitrophenol, 3&4 methylphenol, 3,3'-dichlorobenzidine, 3-nitroaniline, 4,6-dinitro-2-methylphenol, 4-bromophenyl phenyl ether, 4-chloro-3-methylphenol, 4-chlorophenyl phenol ether, 4-methylphenol, 4-nitroaniline, 4-nitrophenol, acenaphthylene, benzo(ghi)perylene, carbazole, dibenzofuran, hexachlorobutadiene, N-nitro-di-N-propylamine, phenanthrene, 1,1,2,2-tetrachloroethane, 1,2,3-trichlorobenzene, 1,2,4-trimethylbenzene, 1,2-dichloroethane, 1,2-dichloropropane, 1,3,5-trimethylbenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene, 2-hexanone, chloroethane, tetrachloroethene, trichloroethene, p-isopropyltoluene, t-1,3-dichloropropene, ammonia, chloride, dodecane, sec-butylbenzene, sulfate.

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SFinhalation : aluminum, beryllium, cobalt, iron, nickel, vanadium, atrazine, endosulfan, endrin aldehyde, endrin ketone, 1-phenyl-1-(2,4-dimethylphenyl)ethane, 1-phenyl-1-(4-methylphenyl)ethane, 2,4,6-trichlorophenol, 2,4-dichlorophenol, 2,4-dimethylphenol, 2,4-dinitrophenol, 2,6-dinitrotoluene, 2-chlorophenol, 2-nitrophenol, 3&4 methylphenol, 3,3'-dichlorobenzidine, 3-nitroaniline, 4,6-dinitro-2-methylphenol, 4-chloro-3-methylphenol, 4-chlorophenyl phenol ether, 4-methylphenol, 4-nitroaniline, 4-nitrophenol, acenaphthene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, bis(2-chloroethoxy)methane, bis(2-chloroethyl)ether, bis(2-ethylhexyl)phthalate, carbazole, chrysene, dibenz(ah)anthracene, indeno(123-cd)pyrene, naphthalene, 1,2,3-trichlorobenzene, 1,2,4-trimethylbenzene, 1,2-dichloropropane, 1,3,5-trimethylbenzene, 1,4-dichlorobenzene, 2-hexanone, bromodichloromethane, carbon disulfide, chlorodibromomethane, xylenes, p-isopropyltoluene, toluene, t-1,3-dichloropropene, 1,1'-biphenyl, ammonia, chloride, chlorine, dodecane, sec-butylbenzene, sulfate

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March 24, 2008

Chloe Metz, Region 2

Onondaga Lake

ASSISTANCE REQUESTED: Toxicity values for approximately 50 chemicals

ENCLOSED INFORMATION: Attachment 1: PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR 1,2,4-TRIMETHYLBENZENE (CASRN 95-63-6)

Attachment 2: PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR 2-CHLOROPHENOL (CASRN 95-57-8)

Attachment 3: PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR 2-NITROPHENOL (CASRN 88-75-5)

Attachment 4: PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR ALUMINUM (CASRN 7429-90-5)

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (4)

cc: STSC files

6-11-2007

Provisional Peer Reviewed Toxicity Values for
1,2,4-Trimethylbenzene
(CASRN 95-63-6)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 1,2,4-TRIMETHYLBENZENE (CASRN 95-63-6)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Neither a reference dose (RfD) nor a reference concentration (RfC) are available for 1,2,4-trimethylbenzene in the Integrated Risk Information System (IRIS) database (U.S. EPA, 2007) or the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997). There is no Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile on 1,2,4-trimethylbenzene, other trimethylbenzene isomers, or mixtures of trimethylbenzene isomers (ATSDR, 2006). The Chemical Assessments and Related Activities (CARA) list (U.S. EPA, 1991, 1994a) and the HEAST (U.S. EPA, 1997) list a Health and Environmental Assessment (HEA) for trimethylbenzenes (U.S. EPA, 1987a); however, the available toxicity data were considered inadequate for quantitative risk assessment (U.S. EPA, 1997). The CARA (U.S. EPA, 1991, 1994a) lists a Drinking Water Health Advisory for 1,2,4-trimethylbenzene (U.S. EPA, 1987b). Because available human and animal toxicity data were considered inadequate for longer-term and lifetime quantitative risk assessment, the U.S. EPA (1987b) derived an RfD of 0.64 mg/kg-day for 1,2,4-trimethylbenzene based on assumptions that the Threshold Limit Value (TLV) of 25 ppm (125 mg/cu.m) for mixed trimethylbenzenes recommended by the American Conference of Governmental Industrial Hygienists (ACGIH, 2001, 2005) represents a NOAEL for 1,2,4-trimethylbenzene and that exposure results in 50% absorption. The National Institute of Occupational Safety and Health (NIOSH) adopted a recommended exposure limit (REL) time-weighted average (TWA) of 25 ppm (123 mg/m³) for 1,2,4-trimethylbenzene (NIOSH, 2006). The Occupational Safety and Health Administration (OSHA) has not adopted a permissible exposure limit (PEL) for 1,2,4-trimethylbenzene (OSHA, 2006). Health assessments for 1,2,4-trimethylbenzene are not available from other major sources, including CalEPA (2006), the

National Toxicology Program (NTP, 2006), the World Health Organization (WHO, 2006), and the International Agency for Research on Cancer (IARC, 2006).

A Group D (not classifiable as to human carcinogenicity) cancer classification is included in the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). A cancer assessment for 1,2,4-trimethylbenzene is not available on IRIS (U.S. EPA, 2007) or the HEAST (U.S. EPA, 1997). A cancer assessment for 1,2,4-trimethylbenzene is not available from CalEPA (2006), the National Toxicology Program (NTP, 2006), the World Health Organization (WHO, 2006), or the International Agency for Research on Cancer (IARC, 2006). Occupational exposure limits for 1,2,4-trimethylbenzene listed by NIOSH (2006) include no cancer notation.

Literature searches were performed to identify relevant information for 1,2,4-trimethylbenzene for the years 1986-1998 in the databases HSDB, RTECS, TSCATS, MEDLINE, and TOXLINE. Update literature searches were conducted in TOXLINE, MEDLINE (plus PubMed cancer subset), and DART/ETICBACK for the time period including January, 1998 to December, 2005. Update search of the TOXCENTER database was performed for the time period of August, 2000 to December, 2005. Databases searched without date limitations in December, 2005, included TSCATS, RTECS, GENETOX, HSDB and CCRIS. Search of Current Contents encompassed July to December, 2005.

REVIEW OF PERTINENT DATA

Human Studies

Oral Exposure

No data were located regarding the oral toxicity or carcinogenicity of 1,2,4-trimethylbenzene in humans.

Inhalation Exposure

Data regarding the inhalation toxicity of 1,2,4-trimethylbenzene in humans come from an occupational exposure study in which workers were exposed to a mixture of trimethylbenzene isomers. Bättig et al. (1958) examined 27 workers exposed to Fleet-X DV 99 solvent in the painting shop of a Swiss transportation plant. The solvent was analyzed spectrographically and was found to consist primarily of aromatic hydrocarbons (97.5%) and paraffinic and naphthenic hydrocarbons (2.5%). The aromatic hydrocarbon portion was composed of 1,2,4-trimethylbenzene (>50%), 1,3,5-trimethylbenzene (>30%), and possibly included 1,2,3-trimethylbenzene, 1-methyl-2-ethyl benzene, 1-methyl-3-ethyl benzene, and 1-methyl-4-ethyl benzene. Based on analysis of air samples collected from the plant, the concentration of the solvent was roughly estimated at 10-60 ppm (49-295 mg/m³). The control group consisted of 10 unskilled workers employed in a different section of the plant. Although the authors stated that the Fleet-X DV 99 solvent was used for "a period of some ten years," the average exposure duration of the workers was not reported. The workers reported CNS symptoms (vertigo, headaches and drowsiness) more often than the control group (70% versus 30% in the controls).

Chronic asthma-like bronchitis (30% of workers versus 10% of controls), anemia [defined as < 4.5 million erythrocytes/mm³ and usually combined with normal hemoglobin] (52% versus 20%) and alterations in blood clotting (30% versus 10%) were also observed in the exposed workers. The incidence of CNS symptoms was statistically significantly higher in the exposed workers than in the control group (Fisher's exact test conducted for this assessment; $p < 0.05$). For the other effects, the incidences did not significantly differ between the groups. A higher incidence of vitamin C deficiency was observed in the control group, suggesting that the two groups may not have been matched for socioeconomic status. If the assumption is made that the solvent exclusively contained trimethylbenzene isomers, then this study identifies a LOAEL in the range of 10-60 ppm (49-295 mg/m³) for signs of neurotoxicity.

Animal Studies

Oral Exposure

The database of repeated oral exposure studies in animals for 1,2,4-trimethylbenzene is limited to a 4-week study (Borrison Laboratories, 1984) and a chronic exposure carcinogenicity study (Maltoni et al., 1997). No oral developmental or reproductive toxicity studies were located for 1,2,4-trimethylbenzene.

The primary focus of the Borrison Laboratories (1984) study was the ability of 1,2,4-trimethylbenzene to induce nephrotoxicity. In this study, groups of 10 male Fischer-344 rats were administered doses of 0.5 or 2.0 g/kg neat 1,2,4-trimethylbenzene by gavage 5 days/week for 4 weeks; the duration-adjusted doses were 357 and 1429 mg/kg-day, respectively. A group of rats serving as controls were gavaged with saline. Gross necropsy was conducted in all rats, but only the kidneys underwent histopathologic examination. Mortality rates during treatment in the control, low-, and high-dose groups were 0/10, 1/10, and 10/10, respectively. Deaths in the high-dose group occurred as early as the third day of treatment. Final body weight and absolute kidney weight of low-dose rats were not significantly different than controls. Gross necropsy findings in low-dose animals included speckled cortical surfaces in the kidneys and white gelatinous material inside the urinary bladders. High-dose rats exhibited mottled and red thymus, spotty kidney and liver surfaces, enlarged adrenals, gas filled and yellow intestines and lung congestion. The presence or absence of hydrocarbon nephropathy was determined by examining the incidence of hyaline droplet changes, regenerative epithelium and tubular dilation with granular material. Treatment with 1,2,4-trimethylbenzene did not significantly increase the incidence or severity of nephropathy relative to controls; however, according to the authors, it is possible that high-dose rats died before nephropathy could develop. A NOAEL or LOAEL could not be determined due to the limited scope of the study, although the high dose of 1429 mg/kg-day was clearly a frank effects level (FEL) for mortality.

Maltoni et al. (1997) investigated the carcinogenicity of 1,2,4-trimethylbenzene (99% pure) in a long-term oral exposure experiment. Male and female Sprague-Dawley rats (50/sex/group) received doses of either 0 or 800 mg/kg (4 days/week) of 1,2,4-trimethylbenzene by gavage in 1 ml olive oil for 104 weeks. Food and water consumption and body weights were recorded throughout the experiment. Upon death or terminal sacrifice at 123 weeks, the animals were subjected to systemic necropsy. Histopathology was performed on brain, pituitary gland,

Zymbal glands, salivary glands, Harderian glands, head, tongue, thymus, mediastinal lymph nodes, lung, heart, diaphragm, liver, spleen, pancreas, kidneys, adrenal glands, esophagus, stomach, intestine (four levels), bladder, prostate, uterus, vagina, gonads, interscapular fat pad, subcutaneous and mesenteric lymph nodes, sternum, femur, spinal cord and any other organs and tissues with pathological lesions. No statistical analysis of the data was presented.

“Slight” reduction in the survival of the female Sprague-Dawley rats and an “intermediate” reduction in the survival of male rats were reported (Maltoni et al., 1997). However, quantitative survival data were not presented in the report and no statistical analysis of the decreases in survival were presented. Although the study report indicated that food and water consumption and body weight data were recorded, these data were not included in the report. There was no significant increase in the incidence of animals bearing either malignant or benign + malignant tumors (Table 1). Fisher’s exact tests conducted for EPA indicated that the differences in total tumors between the exposed and treated animals were not statistically significant ($p < 0.05$). Neuroesthesioepitheliomas were observed in the nasal cavity of 3/100 exposed animals (M + F). This tumor was not seen in concurrent controls, and a Fisher’s exact test of the data showed that the increase in incidence of neuroesthesioepitheliomas was not statistically significant ($p < 0.05$). The authors, however, indicated that these tumors are quite rare in the colony of Sprague-Dawley rats used for these experiments and suggested that this finding presents some evidence for carcinogenicity of 1,2,4-trimethylbenzene.

Table 1. Incidences of Benign and Malignant Tumors in Male and Female Sprague-Dawley Rats after a Long-term (104 week) Oral Exposure to 1,2,4-Trimethylbenzene.^a

Dose (mg/kg bw) ^b	Animals		Percent of animals with tumors	
	Sex	Number	Benign + Malignant	Malignant
800	M	50	62	26
	F	50	66	24
	M + F	100	64	25
0	M	50	54	24
	F	50	70	22
	M + F	100	62	23

^aSource: Maltoni et al., 1997

^bGavage dose administered 4 days/week for 104 weeks and animals were terminated after 123 weeks.

Inhalation Exposure

Korsak and Rydzyński (1996) examined the neurotoxic effects of acute exposure of male Wistar rats (10/group) to 1,2,4-trimethylbenzene (>97% pure) and other trimethylbenzene isomers, and also examined the neurotoxic effects of subchronic exposure to 1,2,4-trimethylbenzene and 1,2,3-trimethylbenzene. In the acute experiment, rats were exposed to concentrations of 250-2000 ppm (1227-9816 mg/m³) for 4 hours. Acute exposure to 1,2,4-trimethylbenzene caused concentration-related impairment in a rotarod performance test (EC₅₀ = 4693 mg/m³) and concentration-related decreased pain sensitivity (as measured by increased paw-lick response latency; EC₅₀ = 5682 mg/m³).

In the subchronic experiment, rats were exposed to 1,2,4-trimethylbenzene at concentrations of 0, 25, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³), 6 hours/day, 5 days/week for 3 months and observed for exposure-related clinical signs and body weight effects (Korsak and Rydzyński, 1996). Rotarod performance and hot-plate behavior were measured as indices of the neurotoxicity of trimethylbenzene isomers. Rotarod performance was tested prior to start of the study, weekly during exposure, and 2 weeks after the termination of the exposure. Hot-plate behavior was tested immediately after termination of the exposure. Fisher's exact test was used for analysis of rotarod performance and the Kruskal-Wallis test used for changes in pain sensitivity (hot plate behavior). Exposures to 1,2,4-trimethylbenzene did not result in any apparent body weight effects or clinical signs of toxicity. However, exposure-related indicators of neurotoxicity were noted. Rotarod performance failure increased in a concentration-related manner in the groups exposed to 1,2,4-trimethylbenzene, but reached the level of statistical significance (40% failure; p<0.05) only in the highest (1227 mg/m³) exposure group following 8 or 13 weeks of exposure. The incidence of rotarod performance failure in control rats was 0% throughout the study period. Although the mean rotarod performance failure rate in the highest exposure group remained at 30% after a 2-week recovery period, the rate was not significantly different from controls. Pain-sensitivity was also decreased in a concentration dependent manner (evidenced by increased latency of the paw-lick response). As shown in Table 2, the increased latency reached the level of statistical significance in the 491- and 1227-mg/m³ groups. After a 2-week recovery period, the highest (1227 mg/m³) exposure group no longer exhibited a significant difference in pain sensitivity, relative to controls. This study identified a NOAEL of 123 mg/m³ and a LOAEL of 491 mg/m³ (6 hours/day, 5 days/week) for significantly decreased pain sensitivity.

Table 2. Exposure-Related Effect on Latency of the Paw-Lick Response in Rats Exposed to 1,2,4-Trimethylbenzene Vapors 6 Hours/Day, 5 Days/Week for 3 Months.^a

Number of rats	Exposure level (mg/m ³)	Mean latency of paw-lick response (seconds)
9	0	15.4 ± 5.8 ^b
10	123	18.2 ± 5.7
9	491	27.6 ± 3.2 ^c
10	1227	30.1 ± 7.9 ^c

^a Source: Korsak and Rydzyński, 1996

^b The authors did not specify whether standard deviation or standard error of the mean is presented

^c Statistically significantly different from controls (p≤0.01)

Gralewicz et al. (1997a) investigated 1,2,4-trimethylbenzene-induced behavioral effects on groups of male Wistar rats (15/group) exposed to vapor concentrations of 0, 50, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³) for 6 hours/day, 5 days/week for 4 weeks. To assess the effect of exposure on short-term working memory, choice accuracy in a radial arm maze was tested. Effects on spontaneous activity were evaluated with an open field test. Effects on long-term memory and learning ability were assessed on the basis of conditioned passive and active avoidance tests. The hot-plate test was performed to compare the groups with respect to the decrease in responsiveness to a thermal stimulus following a brief intermittent foot shock. Animals were subjected to the following sequence of behavioral testing:

1. radial maze: 2 weeks before exposure and on days 14-18 after exposure,
2. open field activity: day 25 after exposure,
3. passive avoidance: days 35-45 after exposure,
4. hot-plate test: days 50 and 51 after exposure,
5. active avoidance: day 54 after exposure.

The data were analyzed by ANOVA and comparisons among treatments were made using Sheffe's test, or Tukey's test for 2-way ANOVA.

There was no significant effect of 1,2,4-trimethylbenzene exposure on body weight gain during the 4-week exposure. Passive-avoidance learning was significantly ($p < 0.001$) retarded in groups exposed to 491 or 1227 mg/m³ of 1,2,4-trimethylbenzene and tested 35-45 days after the end of the exposure period. Retardation of passive-avoidance learning was more pronounced in the 491 mg/m³ exposure group than in the 1227 mg/m³ group. In the hot-plate test following foot shock, evaluation of rats 50 days following termination of exposures to 491 or 1227 mg/m³ of 1,2,4-trimethylbenzene revealed significantly ($p < 0.01$) increased paw-lick latency times, in comparison to unexposed controls. There was no significant change in the active avoidance test, although there was a trend toward decreased avoidance responses with increasing 1,2,4-trimethylbenzene exposure concentration. Short-term working memory did not appear to be adversely affected by 1,2,4-trimethylbenzene exposure. In the open field test there was no significant effect on spontaneous movement or on rearing behavior; however, there was a significant ($p < 0.05$) increase in grooming behavior of animals exposed to 1,2,4-trimethylbenzene at 491 mg/m³. Although grooming behavior also was increased above controls in the 123 and 1227 mg/m³ groups, the difference was not statistically significant. The results of these experiments suggest that 4-week exposures at concentrations that produced no overt clinical signs of toxicity can produce long-term effects on the functional state of the rat central nervous system. Based on findings of significantly retarded passive avoidance learning and increased paw-lick latency in rats of the 491 and 1227 mg/m³ exposure groups, the 123 mg/m³ group represented a NOAEL and the 491 mg/m³ group represented a LOAEL (6 hours/day, 5 days/week) for persistent neurotoxic effects.

Gralewicz and Wiaderna (2001) employed the same general protocol used by Gralewicz et al. (1997a) in a comparative study of the behavioral effects of repeated inhalation exposure to individual trimethylbenzene isomers or *m*-xylene. The study included a group of 11 adult male Wistar rats exposed to 100 ppm (491 mg/m³) of 1,2,4-trimethylbenzene (purity not stated) and a control (air only) group of 10 male rats. Exposures were for 6 hours/day, 5 days/week for 4

weeks. The sequence of behavioral testing varied slightly from that employed by Gralewicz et al. (1997a) and included:

1. radial maze: 1 week before exposure and on days 14-18 after exposure,
2. open field activity: day 8 before exposure and day 25 after exposure,
3. passive avoidance: days 39-48 after exposure,
4. hot-plate test: days 50 and 51 after exposure,
5. active avoidance: days 54 and 60 after exposure.

No significant exposure-related effects were seen regarding body weights or short-term working memory (as determined in the radial arm maze test) for any of the trimethylbenzene isomers or *m*-xylene. Acquisition, but not retention, of the two-way active avoidance response was significantly impaired in all solvent-exposed groups. Results of other behavioral tests demonstrated exposure-related effects for each of the solvents. In the case of 1,2,4-trimethylbenzene, significantly increased spontaneous locomotor activity in the open field, impaired passive avoidance learning and significantly longer paw-lick latencies in the hot-plate test 24 hours after foot shock were observed. These results support the findings of the earlier study (Gralewicz et al., 1997a) in which the 491 mg/m³ (6 hours/day, 5 days/week) exposure level represented a LOAEL for neurotoxic effects in 1,2,4-trimethylbenzene-exposed rats.

Gralewicz et al. (1997b) investigated the effect of a 4-week (6 hours/day, 5 days /week) inhalation exposure to 1,2,4-trimethylbenzene (purity not stated) at concentrations of 0, 25, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³) on the occurrence of spike-wave discharges (SWD) in the neurocortex of male Wistar rats (9-10/group). Bursts of SWD increase in number and/or duration with advancing age and it was hypothesized that exposure to neurotoxic solvents may accelerate the aging process in the brain. Electrodes were implanted into the fronto-parietal cortex and into the dorsal hippocampus. One-hour EEG recordings were performed immediately before initiation of exposure, at the end of the exposure period, 1 month later and 3 months later. The occurrence of SWD bursts is limited to the state of awake immobility. The number and total duration of SWD bursts were determined from each EEG. The data were analyzed by ANOVA and multiple comparisons among treatments was performed with Tukey's test. The study results included information regarding mean body weights, but the study report did not provide details of body weight data collection.

The study authors (Gralewicz et al., 1997b) indicated that 1,2,4-trimethylbenzene exposure resulted in no statistically significant body weight effects. In the control and lowest (123 mg/m³) exposure groups, the total duration of SWD showed an increasing trend with time, in comparison to pre-exposure SWD and reached statistical significance ($p < 0.05$) at 3 months after exposure. In contrast, the total duration of SWD tended to decline with time in the mid- and high-exposure groups after exposure. The decrease in SWD occurrence, however, was statistically significant only for the measurements performed 1 month after the end of exposure in the mid-exposure (491 mg/m³) group. A similar trend was seen when the number of SWD bursts per hour was determined. The frequency of SWD bursts increased with age in the control and lowest exposure groups and tended to decline with time in the mid- and high-exposure groups. The data, however, were highly variable and were statistically significantly different from pre-exposure SWD only for the highest exposure level at 3 months after exposure. Thus,

there were no clear 1,2,4-trimethylbenzene induced concentration-related effects on SWD, although the results are suggestive that long-term effects on brain activity may have occurred.

Korsak et al. (2000) exposed groups of male and female rats (10/sex/group; 20/sex/group at the highest exposure concentration) of outbred Imp:WIST to 1,2,4-trimethylbenzene (97% pure) vapors at target concentrations of 0, 123, 492 or 1230 mg/m³ for 6 hours/day, 5 days/week for 3 months. Animals were observed twice daily for clinical signs of toxicity. Body weights were recorded prior to initiation of exposures and weekly thereafter and food consumption was measured weekly. Blood was drawn for hematological examination prior to initiation of exposures and 1 week prior to exposure termination. Clinical chemistry testing was performed at the end of the 3-month exposure period. Organ weights were determined for lungs, liver, spleen, kidneys, adrenals, heart and gonads. Histopathological examinations were performed on tissues from brain, nose, larynx, trachea, thymus, lungs, heart, liver, spleen, kidney, adrenals, thyroid, pancreas, gonads, urinary bladder, stomach, duodenum, small and large intestines and salivary glands.

Clinical findings were unremarkable (Korsak et al., 2000). No significant exposure-related effects were seen regarding food consumption or body weights. The few differences in some relative or absolute organ weights did not appear to be of toxicological relevance. Significant concentration-related trends ($p < 0.01$) for decreased numbers of red blood cells and increased numbers of white blood cells were noted for male (but not female) rats. In the male rats of the highest 1,2,4-trimethylbenzene exposure group (1230 mg/m³), both red and white blood cell counts were significantly different ($p < 0.01$) from those of control males. A significant trend ($p < 0.01$) for concentration-related decreased reticulocyte counts was observed in female rats and the difference was significant ($p < 0.05$) in the 1230 mg/m³ group. Hematological testing also revealed a significant trend ($p < 0.01$) for decreased clotting time in female (but not male) rats; the decrease reached the level of statistical significance ($p < 0.05$) in the 492 and 1230 mg/m³ groups. Clinical chemistry results were unremarkable, with the exception of significantly increased serum sorbitol dehydrogenase in all 1,2,4-trimethylbenzene-exposed groups of male rats (not concentration related). Histopathological examinations revealed exposure-related significantly increased severity of pulmonary lesions, which included increased proliferation of peribronchial lymphatic tissue in males of the mid- (but not highest) exposure level, increased alveolar macrophages in males of the highest exposure level and increases in interstitial lymphocytic infiltrations in males of the mid- (but not highest) exposure level and females of the highest exposure level. No significant exposure-related changes were seen in the other examined organs and tissues. The mid exposure level of 492 mg/m³ can be considered a LOAEL for hematological and respiratory effects in this study. The low exposure level of 123 mg/m³ is a NOAEL.

Korsak et al. (1997) exposed male Wistar rats of IMP:DAK outbred stock (10/group) to 1,2,4-trimethylbenzene ($\geq 97\%$ pure) at concentrations of 0, 25, 100 or 250 ppm (0, 123, 491 or 1227 mg/m³) for 90 days (6 hours/day, 5 days/week). Lung lavage fluid was collected 24 hours after termination of the subchronic exposure and centrifuged at 400 g for 10 minutes. Differential counts of bronchoalveolar lavage (BAL) cell smears were determined by light microscopy after staining and the trypan blue test was used to determine cell viability. Total

protein concentration, mucoprotein concentration, lactate dehydrogenase and acid phosphatase activity were determined in the BAL supernatant.

All rats exposed to 1,2,4-trimethylbenzene for 90 days survived the experiment and there were no significant differences in final body weight. Statistically significant increases were observed in total cell and macrophage numbers in BAL of all treated groups after 90 days in comparison to controls. Significant increases were also observed in total protein, lactate dehydrogenase (LDH) and acid phosphatase (AP) in BAL fluid of all treated groups. However, the observed increases in these parameters were either at or near their highest observed response at the lowest exposure concentration, and there was no indication of further concentration-related increases. For the observed effects, the lowest exposure level used (123 mg/m^3) would be a LOAEL; however, the toxicological significance of these effects is not clear.

In a study by IBT (1981), groups of 5 male and 5 female COBS rats were exposed to 49 or 480 mg/m^3 MCS-1809 6 hours/day, 5 days/week for 4 weeks (IBT, 1981). MCS-1809 was identified as a compound containing 75% 1,2,4-trimethylbenzene and 25% C9 aromatics (Monsanto, 1992). The test atmosphere was generated by passing the MCS-1809 through a nebulizer; no information on the particle size distribution was reported. Based on the vapor pressure of 1,2,4-trimethylbenzene, it is likely that the animals were predominantly exposed to 1,2,4-trimethylbenzene vapors rather than a mist. The following parameters were used to assess toxicity: daily observations, weekly body-weight measurements, organ weights (adrenal glands, brain, gonads, heart, kidneys, liver, lungs, spleen and thyroid gland), gross necropsy and histopathological examination of adrenal glands, brain, bronchi, gonads, heart, kidneys, liver, lungs, pancreas, pituitary glands, lymph nodes, spleen, trachea and thyroid gland of the control and 480 mg/m^3 groups (tissues from the 49 mg/m^3 group were examined if significant findings were found in the 480 mg/m^3 group).

Exposure to MCS-1809 did not result in deaths. Clinical signs of toxicity in the 480 mg/m^3 group included ataxia and hypoactivity that persisted between exposures, ptosis, red ocular discharge, and ruffed fur. Less pronounced hypoactivity and ruffed fur were observed in the 49 mg/m^3 group. In the 480 mg/m^3 group, significant decreases in body weight gain (35% lower in the males; no significant alteration in females), increases in absolute (females only) and relative liver weights and decreases in absolute and relative spleen weights (females only) were observed. A significant increase in absolute liver weight was also observed in the 49 mg/m^3 female rats. Histological alterations were limited to focal or diffuse testicular atrophy in 3/5 male rats exposed to 480 mg/m^3 in the absence of statistically significant changes in testis weight; no testicular effects were observed in the 49 mg/m^3 (testes examined in four rats from this group) or control groups. This study identified a NOAEL of 49 mg/m^3 and LOAEL of 480 mg/m^3 (6 hours/day, 5 days/week) for clinical signs of toxicity (persistent ataxia and hypoactivity, ptosis, ocular discharge), decreased body weight gain, and histopathological evidence of testicular atrophy. The increased absolute liver weight observed in the 49 mg/m^3 female rats was not considered adverse because no histological alterations were observed at the 49 or 480 mg/m^3 concentrations.

Bättig et al. (1958) exposed 8 male rats (strain not reported) to air concentrations of 1700 ppm of Fleet-X DV 99 solvent for 4 months (8 hours/day, 5 days/week). Other rats (sex, strain,

and number not specified) were exposed to 500 ppm for 70 days (8 hours/day, 5 days/week). As described earlier, Fleet-X DV 99 is a solvent containing 97.5% aromatic hydrocarbons (>50% 1,2,4-trimethylbenzene and >30% 1,3,5-trimethylbenzene) and 2.5% of paraffinic and naphthenic hydrocarbons. The 500- and 1700-ppm concentrations would be approximately 2523 and 8155 mg/m³, respectively, if the aromatic hydrocarbon fraction of the vapors were comprised exclusively of trimethylbenzenes. Within 2 weeks of exposure, 4 of the 8 rats exposed to 1700 ppm died and were replaced, while none of the animals in the 500-ppm group died. Histopathologic examinations, performed only on 1700-ppm animals that died, revealed cloudy swelling and fatty infiltration in the kidneys, peripheral fatty infiltration in the liver, an increase in secondary nodules in the spleen and marked congestion of the pulmonary capillaries with alveolar wall thickening. Alterations in differential white blood cell counts (increase in the percentage of segmented neutrophilic granulocytes and a decrease in the percentage of lymphocytes) were reported at 500 ppm. Increases in drinking water consumption (43-45% higher than in the control group) were observed in the 1700-ppm group. The authors reported that during the exposure period, the 1700-ppm animals were initially “highly excited and aggressive” followed by a period of narcosis and ataxia. Because histopathology was only performed on the animals that died, no histopathology data are available on the 500-ppm rats. Due to the limited scope of the study, a NOAEL or LOAEL cannot be identified. The high concentration of 1700 ppm (8155 mg/m³) is a FEL for mortality.

Bernshtein (1972) exposed rats (number, sex and strain not specified) to 1000 mg/m³ (200 ppm) of a mixture of trimethylbenzenes for 6 months (4 hours/day, 6 days/week). An inhibition of phagocytic activity of the leukocytes was reported. This study was summarized by Sandmeyer (1981) and further experimental details were not provided.

Korsak et al. (1997) examined the effect of acute exposures (6 min) to the trimethylbenzene isomers, 1,2,3-trimethylbenzene (90-95% pure), 1,3,5-trimethylbenzene (99% pure) and 1,2,4-trimethylbenzene (97% pure) on the respiratory rate of Balb/C male mice (8-10/group) at concentrations ranging from 253 to 1591 ppm (1926-9453 mg/m³). The concentration depressing mouse respiratory rate by 50% (RD₅₀) was calculated by least squares regression and the Kruskal-Wallis test was applied for evaluation of protein and enzyme levels in the BAL fluid. All three trimethylbenzene isomers showed irritating effects on the respiratory tract and caused concentration-dependent decreases in respiratory rate. The concentration depressing the respiratory rate in mice to 50% was 519 ppm (2547 mg/m³) for 1,2,4-trimethylbenzene.

The developmental toxicity of 1,2,4-trimethylbenzene was assessed by Saillenfait et al. (2005). Groups of mated (sperm-positive) Sprague-Dawley rats (24/group) were exposed (whole body) to 1,2,4-trimethylbenzene (97% pure) at vapor concentrations of 0, 100, 300, 600 or 900 ppm (0, 491, 1475, 2950 or 4425 mg/m³) for 6 hours/day on gestation days 6 through 20. Maternal food consumption was recorded for the intervals of gestation days 6-13 and 13-21. Maternal body weights were recorded weekly during gestation. At necropsy on gestation day 21, the uterus was weighed and numbers of corpora lutea, implantation sites, resorptions and dead and live fetuses were recorded. Live fetuses were weighed, sexed and examined for external anomalies. Half of the live fetuses from each litter were prepared for visceral examination, the others were subjected to skeletal examination.

All dams survived to scheduled necropsy (Saillenfait et al., 2005). No clinical signs of 1,2,4-trimethylbenzene-induced toxicity were observed. Maternal food consumption was significantly ($p < 0.01$) depressed in the 600- and 900-ppm groups (approximately 12-14% and 15-19%, respectively, relative to controls). The 900-ppm dams exhibited significantly reduced mean body weight gain (22-52% lower than controls) throughout the exposure period. Significantly ($p < 0.01$) reduced body weight gain (30% lower than controls) was observed in the 600-ppm group, but only during the first week of 1,2,4-trimethylbenzene exposure. At necropsy on gestation day 21, mean body weight gain (corrected for gravid uterine weight) was significantly depressed in both 600- and 900-ppm dams (approximately 50% lower than controls). Mean fetal body weight was significantly lower in both 600- and 900-ppm exposure groups (approximately 5 and 11% lower, respectively, than controls). There were no other significant indications of maternal or fetal toxicity. This study identified a NOAEL of 300 ppm (1475 mg/m^3) and a LOAEL of 600 ppm (2950 mg/m^3 , 6 hours/day on gestation days 6 through 20) for maternal and fetal body weight effects. However, the observed fetal toxicity was likely secondary to maternal toxicity because the decreased fetal body weight was noted only at exposure levels resulting in significantly depressed maternal body weight gain.

Other Studies

Limited genotoxicity data suggest that 1,2,4-trimethylbenzene is not mutagenic. 1,2,4-trimethylbenzene produced negative results in the Ames test with *Salmonella typhimurium* strains TA97a, TA98, TA100 and TA102 both in the presence and absence of rat liver S9 metabolic activation (Janik-Spiechowicz et al., 1998). 1,2,4-Trimethylbenzene was not cytogenic in the mouse micronucleus test, but elicited a positive response in sister chromatid exchange (SCE) tests with bone marrow cells of Imp:Balb/c mice treated *in vivo* (Janik-Spiechowicz et al., 1998).

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfDs FOR 1,2,4-TRIMETHYLBENZENE

Limited information is available regarding the oral toxicity of 1,2,4-trimethylbenzene in humans and animals. The nephrotoxicity study by Borriston Laboratories (1984) is too limited in scope to be used to identify a NOAEL or LOAEL for 1,2,4-trimethylbenzene, although the 2000 mg/kg dose (1429 mg/kg-day) is clearly a FEL for increased mortality. The study of Maltoni et al. (1997) is also unsuitable for derivation of an RfD, as only one dose level was employed, decreased survival occurred at this dose level and reporting of the results was inadequate. Thus, the database for 1,2,4-trimethylbenzene is inadequate to derive a provisional RfD.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfCs FOR 1,2,4-TRIMETHYLBENZENE

Several studies have examined the inhalation toxicity of mixtures predominantly containing one or more trimethylbenzene isomers, or pure 1,2,4-trimethylbenzene. Significant

increases in the incidence of CNS toxicity (vertigo, dizziness, headaches) and non-significant increases in the incidences of respiratory effects (bronchitis) and hematological effects (hyperchromic anemia and blood clotting alterations) have been observed in workers exposed to 10-60 ppm (49-295 mg/m³) of a solvent containing >80% trimethylbenzene isomers (Bättig et al., 1958). Many of these effects reported in humans have been observed in experimental animals repeatedly exposed to 1,2,4-trimethylbenzene or other trimethylbenzene isomers or trimethylbenzene mixtures. For example, hematological effects have been reported in experimental animals exposed to a trimethylbenzene mixture (Bernshtein, 1972) or 1,2,4-trimethylbenzene (Korsak et al., 2000). Signs of adverse CNS effects have been observed in animals exposed to mixtures containing 1,2,4-trimethylbenzene (Bättig et al., 1958; IBT, 1981) or 1,2,4-trimethylbenzene alone (Gralewicz and Wiaderna, 2001; Gralewicz et al., 1997a, 1997b; Korsak and Rydzyński, 1996). Results of other animal studies provide evidence of 1,2,4-trimethylbenzene-induced respiratory effects (Korsak et al., 1997, 2000). Other effects observed in animal studies include testicular atrophy in rats exposed to 480 mg/m³ (98 ppm) of a mixture containing 75% 1,2,4-trimethylbenzene for 4 weeks (6 hours/day, 5 days/week) (IBT, 1981) and lung, liver, kidney and spleen effects in rats exposed to 1700 ppm (8155 mg/m³) of a solvent containing >80% trimethylbenzene isomers for 4 months (8 hours/day, 5 days/week) (Bättig et al., 1958).

The lowest estimated level of occupational exposure to the solvent Fleet-X DV 99 (>80% 1,2,4- and 1,3,5-trimethylbenzene) in the study of Bättig et al. (1958) was 10 ppm (49 mg/m³). Assuming that the solvent exclusively contained trimethylbenzene isomers, the 49 mg/m³ exposure concentration can be considered to represent a LOAEL. Although the Bättig et al. (1958) report provides the lowest inhalation LOAEL (49 mg/m³) of any study, it may be an inappropriate study for consideration as the principal study for a number of reasons. Importantly, Bättig et al. (1958) identified spectrophotographically the presence of various aromatic hydrocarbons, to include naphthenic and paraffenic compounds, in addition to 1,2,4-trimethylbenzene in the solvent mixture. While 1,2,4-trimethylbenzene comprised up to 50% of the Fleet-X DV 99 mixture, it is virtually impossible to confidently attribute human toxicities solely to 1,2,4-trimethylbenzene (i.e. the study LOAEL is for the mixture not the individual compound). Additional concerns that warrant exclusion of the Bättig et al. (1958) human study from consideration include inappropriate selection of a human control population [e.g. nutritional status (Vit. C deficient)], and the fact that average Fleet-X DV 99 solvent exposure duration, for the 27 exposed workers examined, was not reported.

An advantage of some of the animal models of 1,2,4-trimethylbenzene inhalation exposure over the Bättig et al. (1958) human study is that controlled atmospheres involved the compound of interest at relatively high purities (e.g. 97% 1,2,4-trimethylbenzene in the Korsak et al., 2000 study). However, available repeated exposure inhalation studies in animals are limited to subchronic exposure duration (4 weeks to 3 months) in which the lowest identified LOAEL for 1,2,4-trimethylbenzene was 491 mg/m³ (Gralewicz and Wiaderna, 2001; Gralewicz et al., 1997a; Korsak and Rydzyński, 1996); furthermore, many of the effects observed in these rodent studies are of unclear toxicological significance and/or have concentration-responses that are difficult to interpret.

Provisional RfCs may be derived based on adverse pulmonary or hematological effects reported in male or female rats, respectively, exposed to 1,2,4-trimethylbenzene (97% pure) for 3 months (Korsak et al., 2000). The selection of the Korsak et al. (2000) study as the basis for deriving RfCs is supported by previous observations in rats (Korsak et al., 1997) and humans (Bättig et al., 1958) exposed to pure 1,2,4-trimethylbenzene or a mixture of trimethylbenzenes, respectively, for ≥ 90 days. Indeed, pulmonary lesions and hematological abnormalities in rats exposed to pure 1,2,4-trimethylbenzene for 3 months (Korsak et al., 2000) are consistent with observations in humans following presumably longer duration exposure to a mixture containing 1,2,4-trimethylbenzene (Bättig et al., 1958).

Subchronic p-RfC

The subchronic p-RfC for 1,2,4-trimethylbenzene is derived from the NOAEL of 25 ppm (123 mg/m^3) identified in the Korsak et al. (2000) rat subchronic inhalation study. Two different toxic effects (pulmonary or hematological) were identified in male or female rats, respectively, in this study at the same LOAEL/NOAEL. As such, two separate subchronic p-RfC derivations are presented below to identify the most sensitive endpoint. Under an assumption of category 3 for decreased clotting time in female Imp:WIST rats, an adjusted experimental NOAEL can be derived using the NOAEL of 123 mg/m^3 and the exposure duration data from Korsak et al. (2000) as follows:

$$\begin{aligned}\text{NOAEL}_{[\text{ADJ}]} (\text{mg/m}^3) &= \text{rat NOAEL} (\text{mg/m}^3) \times 6\text{hr}/24\text{hr} \times 5 \text{ days}/7 \text{ days} \\ &= 123 \text{ mg/m}^3 \times 0.25 \times 0.71 \\ &= 21.8 \text{ mg/m}^3\end{aligned}$$

According to equation (4-48) for extrapulmonary effects [Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994)], a human equivalent concentration ($\text{NOAEL}_{[\text{HEC}]}$) can be calculated as follows:

$$\text{NOAEL}_{[\text{HEC}]} (\text{mg/m}^3) = \text{NOAEL}_{[\text{ADJ}]} (\text{mg/m}^3) \times (\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$$

*blood:gas (b/g) partition coefficients for 1,2,4-trimethylbenzene could not be located, therefore a default value of 1 is used for the term $(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$. The human $\text{NOAEL}_{[\text{HEC}]}$ is equivalent to the duration adjusted rat NOAEL of 21.8 mg/m^3 . A **subchronic p-RfC of $7\text{E}-2 \text{ mg/m}^3$** based on a hematological effect is derived by dividing the $\text{NOAEL}_{[\text{HEC}]}$ of 21.8 mg/m^3 by a composite UF of 300, as follows:

$$\begin{aligned}\text{UF (animal to human)} &= 3 \\ \text{UF (interindividual variability)} &= 10 \\ \text{UF (database deficiencies)} &= 10\end{aligned}$$

$$\begin{aligned}\text{Subchronic p-RfC} &= \text{NOAEL}_{[\text{HEC}]} / \text{UF} \\ &= 21.8 \text{ mg/m}^3 / 300 \\ &= 0.07 \text{ mg/m}^3 \text{ or } 7\text{E}-2 \text{ mg/m}^3\end{aligned}$$

Under an assumption of category 1 for pulmonary toxicity in male rats, the same duration adjusted rat NOAEL of 21.8 mg/m³ is obtained as shown above. Histopathological observations in lung tissue of male Imp:WIST rats exposed to 1,2,4-trimethylbenzene for 3 months indicated inflammatory lesions primarily in the bronchiolar region. Therefore, according to equation 4-22 for Category 1 tracheobronchial effects [Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994)], a NOAEL_[HEC] can be calculated as follows:

$$\begin{aligned}\text{NOAEL}_{[\text{HEC}]} (\text{mg}/\text{m}^3) &= \text{NOAEL}_{[\text{ADJ}]} (\text{mg}/\text{m}^3) \times \text{RGDR}_{\text{TB}}^{\dagger} \\ &= 21.8 \text{ mg}/\text{m}^3 \times 1.6 \\ &= 34.9 \text{ mg}/\text{m}^3\end{aligned}$$

[†] Derivation of the RGDR_{TB} can be found in Appendix 1

A **subchronic p-RfC of 1E-1 mg/m³** based on pulmonary effects is derived by dividing the NOAEL_[HEC] of 34.9 mg/m³ by the same composite UF of 300 identified above:

$$\begin{aligned}\text{Subchronic p-RfC} &= \text{NOAEL}_{[\text{HEC}]} / \text{UF} \\ &= 34.9 \text{ mg}/\text{m}^3 / 300 \\ &= 0.1 \text{ mg}/\text{m}^3 \text{ or } 1\text{E}-1 \text{ mg}/\text{m}^3\end{aligned}$$

The composite UF includes a factor of 3 for extrapolation from animal to human, 10 for interindividual variability, and 10 for database deficiencies. The reduced uncertainty of 3 for animal to human extrapolation is due in part to both the conversion of the rat NOAEL to a human equivalent concentration as well as the consistency of hematological and pulmonary toxicity between rats and humans exposed to 1,2,4-trimethylbenzene. The database deficiencies include lack of developmental toxicity studies in a second species, multigeneration reproductive toxicity studies, and a lack of confidence in the large majority of available animal studies reporting effects of undetermined toxicological significance with concentration-responses that are difficult to interpret. The derivations shown above clearly indicate that decreased clotting time in female rats due to subchronic inhalation exposure to 1,2,4-trimethylbenzene is the more sensitive or health protective endpoint under consideration.

Chronic p-RfC

The **chronic p-RfC of 7E-3 mg/m³** based on decreased clotting time in female rats (Korsak et al., 2000) is derived by dividing the NOAEL_[HEC] of 21.8 mg/m³ by a composite UF of 3000, as follows:

$$\begin{aligned}\text{Chronic p-RfC} &= \text{NOAEL}_{[\text{HEC}]} / \text{UF} \\ &= 21.8 \text{ mg}/\text{m}^3 / 3000 \\ &= 0.007 \text{ or } 7\text{E}-3 \text{ mg}/\text{m}^3\end{aligned}$$

As for the chronic RfC, the composite UF includes a factor of 10 for extrapolation from subchronic to chronic exposure, 3 for extrapolation from animal to human, 10 for interindividual variability, and 10 for database deficiencies.

Confidence in the principal study (Korsak et al., 2000) is low. While it is remarkable that hematological and pulmonary effects are apparently conserved from rats (Korsak et al., 2000) to humans (Bättig et al., 1958), the concentration-response for either compartment in rats (particularly male rats) is difficult to interpret. Specifically, the low inhalation concentration (123 mg/m^3) in female rats from the Korsak et al. (2000) study was clearly a NOAEL for decreased clotting time (hematological compartment); this NOAEL was also identified for pulmonary effects (e.g. proliferation of peribronchial lymphatic tissue, interstitial lymphocytic infiltration of parenchyma, bronchitis and bronchopneumonia) in male rats. Interestingly, female rats seemed slightly more resistant to these pulmonary effects. However, the overall commutative score, following statistical trend analysis, of all pulmonary lesions suggested that the lungs of male and female Imp:WIST rats are significantly affected by inhalation exposure to 1,2,4-trimethylbenzene at the mid dose of 492 mg/m^3 (Korsak et al., 2000). However, paradoxically, in male rats of the high dose group the pulmonary effects decreased compared to animals in the mid dose group. This counter-intuitive concentration-response relationship might suggest a concentration-dependent transition in mode of action for pulmonary toxicity (note the increase in absolute lung weight of male Imp:WIST rats at the mid concentration of 492 mg/m^3 , which is the concentration at which inflammatory foci were identified in lung tissue, and yet in the high concentration group lung weight decreased back to control levels (Korsak et al., 2000); more work would be required to verify.).

According to the derivations provided above it appears that the hematological endpoint (i.e. decreased clotting time in female rats) may be a more appropriate endpoint to consider for inhalation exposure to 1,2,4-trimethylbenzene. Further work in this area is certainly warranted. The human occupational report from Bättig et al. (1958) identified a lower inhalation effect level (e.g. LOAEL = 49 mg/m^3) compared to any of the available animal data. However, the utility of this study in derivation of RfCs is limited by poor reporting of results, undetermined exposure levels, the lack of statistical analysis of results, the lack of information on the exposed and control groups (e.g., age, education level, length of employment), small group sizes and possibly a poorly matched control group (as evidenced by increased incidence of vitamin C deficiency in controls). Also, the controls worked in adjacent rooms and the possibility that they also may have been exposed to trimethylbenzene cannot be excluded. Confidence in the database is low because the database is lacking developmental toxicity data in a second species and reproductive toxicity studies. Reflecting low confidence in the principal study and database, confidence in the provisional subchronic and chronic RfC values is low.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 1,2,4-TRIMETHYLBENZENE

Weight-of-evidence Classification

No information was located regarding the carcinogenicity of 1,2,4-trimethylbenzene or mixtures of trimethylbenzene isomers in humans. The database of information regarding the carcinogenicity of trimethylbenzene in animals is limited to a single carcinogenicity study in which male and female Sprague-Dawley rats (50/sex/group) were administered 1,2,4-trimethylbenzene via oral gavage at doses of 0 or 800 mg/kg for 104 weeks (Maltoni et al.,

1997). Although quantitative survival data were not included in the study report, the authors noted “intermediate” and “slight” reduction in the survival of 1,2,4-trimethylbenzene treated male and female rats, respectively. Under the conditions of the study, oral exposure to 1,2,4-trimethylbenzene did not cause a statistically significant increase in the incidence of animals bearing either malignant tumors or benign and malignant tumors (combined) or in the incidence of neuroesthesioepitheliomas. The study of Maltoni et al. (1997) included a single animal species (rat) and a single 1,2,4-trimethylbenzene dose level (800 mg/kg). Based on limitations in study design and reporting of results and the lack of additional carcinogenicity data in animals, the database of information for 1,2,4-trimethylbenzene is inadequate to establish the potential carcinogenicity of 1,2,4-trimethylbenzene. Limited genotoxicity data demonstrated that 1,2,4-trimethylbenzene was not mutagenic in several strains of *Salmonella typhimurium*, and did not elicit cytogenicity in the mouse micronucleus test, but did elicit a positive response in sister chromatid exchange (SCE) tests with bone marrow cells of Imp:Balb/c mice treated *in vivo* (Janik-Spiechowicz et al., 1998). These data provide inadequate evidence for genotoxic activity.

Collectively, the available carcinogenicity and genotoxicity data do not adequately assess the carcinogenic potential of 1,2,4-trimethylbenzene in humans or animals. Under the current U.S. EPA (2005) cancer guidelines, the human and animal data are inadequate for a determination of the human carcinogenic potential of 1,2,4-trimethylbenzene.

Quantitative Estimates of Carcinogenic Risk

There are no appropriate human or animal data from which to derive an oral slope factor or inhalation unit risk for 1,2,4-trimethylbenzene.

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APPENDIX 1

According to equation 4-22 for Category 1 tracheobronchial effects [Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994)], a $NOAEL_{[HEC]}$ is calculated using the duration adjusted animal NOAEL and a dosimetric adjustment factor (DAF). In this case the DAF is the RGDR for the tracheobronchial region of the lung ($RGDR_{TB}$). The $RGDR_{TB}$ is calculated as follows:

$$RGDR_{TB} = (RGD_{TB})_A / (RGD_{TB})_H = \frac{(V_E/SA_{TB})_A}{(V_E/SA_{TB})_H} \frac{(e^{-SA_{ET}/V_E})_A}{(e^{-SA_{ET}/V_E})_H}$$

where,

Rat

$V_E = 160.07$ ml/min or 0.16 L/min (derived using equation 4-4, default body wt. for Wistar rats, and the default intercept and coefficient values provided in Table 4-6)

$SA_{TB} = 22.5$ cm² (Table 4-4)

$SA_{ET} = 15.0$ cm² (Table 4-4)

Human

$V_E = 13.8$ L/min (default value based on human body weight of 70 kg)

$SA_{TB} = 3,200$ cm² (Table 4-4)

$SA_{ET} = 200.0$ cm² (Table 4-4)

$$= \frac{(0.16 \text{ L/min} / 22.5 \text{ cm}^2)_A}{(13.8 \text{ L/min} / 3,200 \text{ cm}^2)_H} \frac{(e^{-15.0 \text{ cm}^2 / 0.16 \text{ L/min}})_A^*}{(e^{-200.0 \text{ cm}^2 / 13.8 \text{ L/min}})_H}$$

* the exponential portion of the equation is much smaller than 1; thus this half of the equation is negligible.

$$= 0.007 / 0.0043$$

$$RGDR_{TB} = 1.6$$

7-3-2007

Provisional Peer Reviewed Toxicity Values for
2-Chlorophenol
(CASRN 95-57-8)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value

RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2-CHLOROPHENOL (CASRN 95-57-8)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A chronic reference dose (RfD) value of 5E-3 mg/kg-day is available for 2-chlorophenol on IRIS (U.S. EPA, 1988a) and in the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). The HEAST (U.S. EPA, 1997) lists a subchronic RfD for 2-chlorophenol of 5E-2 mg/kg-day. Both RfD values were based on a no-observed-adverse-effect level (NOAEL) of 5 mg/kg-day for reproductive effects in a drinking water study that exposed rats to 2-chlorophenol for 10 weeks prior to mating and during mating, gestation and weaning (Exon and Koller, 1982). Uncertainty factors of 100 and 1000 were used to derive the subchronic and chronic RfDs, respectively. The source documents for the RfD assessments included a Drinking Water Criteria Document (DWCD) (U.S. EPA, 1986a), a Health Effects Assessment (HEA) (U.S. EPA, 1987a), and two Health and Environmental Effects Documents (HEEDs) (U.S. EPA, 1987b, 1990). The Chemical Assessments and Related Activities (CARA) lists (U.S. EPA, 1991, 1994) do not include any additional relevant EPA documents. The Agency for Toxic Substances and Disease Registry (ATSDR, 1999) and the World Health Organization (WHO, 1989) have assessed the health effects of chlorophenols, but did not derive any oral risk assessment values specifically for 2-chlorophenol.

An RfC for 2-chlorophenol is not available on IRIS (U.S. EPA, 1988a) nor in the HEAST (U.S. EPA, 1997). The Agency for Toxic Substances and Disease Registry (ATSDR) and the World Health Organization (WHO) have not derived any inhalation risk assessment values for 2-chlorophenol. Occupational exposure limits for 2-chlorophenol have not been derived by the American Conference for Governmental Industrial Hygienists (ACGIH), the National Institute

for Occupational Safety and Health (NIOSH) or the Occupational Safety and Health Administration (OSHA).

A cancer assessment for 2-chlorophenol is not available on IRIS (U.S. EPA, 1988a). The HEEDs (U.S. EPA, 1987b, 1990) assigned 2-chlorophenol to U.S. EPA (1986b) Cancer Group D (not classifiable as to human carcinogenicity); this classification is also included in the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). The carcinogenicity of 2-chlorophenol has not been assessed by NTP or IARC.

Literature searches were conducted from the 1960's through August, 2006 for studies relevant to the derivation of provisional toxicity values for 2-chlorophenol. Data bases searched included: TOXLINE/TOXCENTER (including BIOSIS, NTIS and Chemical Abstracts subfiles), MEDLINE (including PubMed cancer subset), TSCATS/TSCATS 2, CCRIS, DART/ETIC, GENETOX, HSDB, RTECS, and Current Contents.

REVIEW OF PERTINENT DATA

Human Studies

Relevant information regarding the toxicity of 2-chlorophenol in humans was not located.

Animal Studies

Oral Exposure. In a 14-day study performed in conjunction with EPA, groups of 12 male and 12 adult female CD-1 ICR mice were administered 2-chlorophenol in corn oil by gavage in doses of 0, 35, 69 or 175 mg/kg-day (Borzelleca, 1983; Borzelleca et al., 1985). The highest dose level was approximately 50% of the acute oral LD₅₀ of 347 and 345 mg/kg in male and female CD-1 mice, respectively. Endpoints evaluated during the study included clinical observations, body weight (days 1, 8 and 15), and food and water intake. Endpoints evaluated at the end of the treatment period included hematology [red blood cells (RBC), total and differential white blood cells (WBC), platelets, hematocrit (Hct), hemoglobin (Hgb) and coagulation], serum chemistry [lactate dehydrogenase (LDH), alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), blood urea nitrogen (BUN), bilirubin, protein, glucose, cholesterol, albumin/globulin, phosphorus, potassium, calcium, sodium and chloride], hepatic microsomal activities (cytochrome P₄₅₀, cytochrome b₅, protein, aminopyrine demethylase, aniline hydroxylase, and arylhydrocarbon hydroxylase), immune response and behavioral measurements. The earlier report of the study (Borzelleca, 1983) implies that the immunology endpoints included cell-mediated response (Delayed-type hypersensitivity (DTH) response to sheep RBC, response to concanavalin A), humoral response [splenic Immunoglobulin mu (IgM) antibody forming cells (AFC) to sheep RBC, serum antibody levels to sheep RBC, lymphocyte response to lipopolysaccharide (LPS)], and reticuloendothelial system (RES) function (vascular clearance and uptake of ⁵¹Cr sheep RBC). The Borzelleca (1983) report also implies that the behavioral endpoints included inverted screen test, swimming endurance, locomotor activity, pain sensitivity, olfactory sensitivity, passive avoidance learning, and forepaw grip strength. Other endpoints included sister-chromatid exchange (bone marrow

and/or testes, not otherwise specified), *in vitro* fertilization capability (penetration of ova, fertilization, blastula formation), absolute and relative organ weights, and gross pathology. Histopathological examinations were not performed. The results of this study are qualitatively reported in tabular summaries. Effects included 100% mortality at 175 mg/kg-day, hyperactivity at 35 and 69 mg/kg-day, reduced body weight at 69 mg/kg-day, and reduced brain, liver and spleen weights (effect levels not indicated); additional information on these effects was not reported. No biologically or statistically significant compound-related adverse effects were reported for the other endpoints as indicated by the authors. The 100% mortality in the high-dose animals indicates that 175 mg/kg-day was a FEL for short-term repeated gavage exposures in mice. The authors (Borzelleca et al., 1985) referred to the effects at the lower doses as “slight toxic effects”, but apparently concluded that they were not biologically significant, indicating that 69 mg/kg-day was a NOAEL. Results of acute studies reported by Borzelleca et al. (1985) include an ED₅₀ of 63 mg/kg for reversible motor impairment in mice exposed to a single oral dose of 2-chlorophenol; additional information was not provided.

Gavage studies (10-day and 90-day) of 2-chlorophenol in Sprague-Dawley rats were conducted by the EPA (Daniel et al., 1993). In the 10-day study, groups of 10 male and 10 female 8-week-old Sprague-Dawley rats were administered 2-chlorophenol in corn oil by daily gavage at doses of 0, 13, 64, 129 or 257 mg/kg-day. The highest dose level was approximately 38% of the reported acute LD₅₀ of 670 mg/kg for a rat. Endpoints evaluated during the study included clinical signs (observed for physiological and behavioral responses and mortality), body weight and food and water consumption. Evaluations at the end of the exposure period included hematology [RBC, WBC, Hct, Hgb and mean corpuscular volume (MCV)], serum chemistry (ALP, AST, ALT, LDH, cholesterol, BUN, creatinine, glucose, and calcium), absolute and relative organ weights (brain, liver, spleen, lungs, thymus, kidneys, adrenal glands, heart, and gonads), and gross pathology. Comprehensive histological examinations were performed in the control and high-dose groups; target organs were also histologically evaluated at the lower dose levels. Tissues that were examined included liver, kidneys, urinary bladder, heart, aorta, skin, skeletal muscle, bone, sciatic nerve, spleen, thymus, lymph nodes, respiratory tract (nasal turbinates, trachea, lung with bronchi), gastrointestinal tract (esophagus, stomach, duodenum, jejunum, ileum, cecum, colon, rectum), endocrine system (adrenals, pancreas, pituitary, thyroid/parathyroid), and reproductive system (testes, epididymis, seminal vesicles, prostate, preputial gland, ovaries, uterus, clitoral gland).

There were no treatment-related deaths, significant clinical observations or significant changes in food or water consumption or body weight gain (Daniel et al., 1993). The hematology evaluations found significantly ($p \leq 0.05$) increased RBC count (12% higher than controls) and Hct (28% higher than controls) in the high-dose (257 mg/kg-day) males; these effects were not clearly dose-related and there were no significant changes in hematologic values in females. Serum chemistry changes that were statistically significant included increased glucose levels in females at 129 and 257 mg/kg-day (45 and 42% higher than controls, respectively) and males at 257 mg/kg-day (21% higher than controls); decreased ALP in females at 129 and 257 mg/kg-day (15 and 16% lower than controls); and decreased AST, cholesterol, and LDH in males at 257 mg/kg-day (25, 27 and 55% lower than controls, respectively). Serum LDH values were significantly decreased in females at 64 and 129 mg/kg-day, but not at 257 mg/kg-day. The only serum chemistry changes that appeared to be dose-related were the

increased glucose and decreased ALP in female rats, but the authors reported that these values were within the normal ranges for laboratory rats. Statistically significant organ weight changes consisted of decreases in absolute kidney and heart weights in females at 129 mg/kg-day, but not at other dose levels, and decreases in absolute and relative lung weights in females at all dose levels; quantitative data were not reported. Necropsy findings included enlarged mandibular lymph nodes, reddened lungs and reduced thymus size in all groups of both sexes; these were minimal to mild changes not considered to be treatment-related by the authors. The histological examinations similarly showed lymphoid hyperplasia, mild congestion of the lungs, and mild thymic atrophy in all groups; these effects did not appear to be treatment-related to the authors because they were not significant in severity or incidence (data not reported). Histopathological changes in kidneys, heart, lungs or other tissues were not reported. The lack of any clear treatment-related or biologically significant hematology, clinical chemistry, organ weight or pathological changes indicates that the highest dose level, 257 mg/kg-day, is a NOAEL for 10-day gavage exposure in male and female rats although it is difficult to ascertain the significance of the reported effects due to a lack of data reporting.

In the 90-day study, groups of 10 male and 10 female 8-week-old Sprague-Dawley rats were administered 2-chlorophenol in corn oil by daily gavage at doses of 0, 17, 50, or 150 mg/kg-day (Daniel et al., 1993). Study endpoints were the same as in the 10-day study summarized above; evaluations included clinical signs, body weight, food and water consumption, hematology, serum chemistry (with the addition of triglycerides, total protein, albumin and globulin), organ weights and gross pathology in all groups, and histopathology in the control and high-dose groups. There were no clinical signs of toxicity, unscheduled deaths, or significant changes in food or water consumption or body weight gain. Hematology changes that were statistically significant included increased RBC count in females at 17 and 150 mg/kg-day (3 and 6% higher controls), but not at 50 mg/kg-day; increased Hct in females at 150 mg/kg-day (5% higher than controls); and increased MCV in males at 150 mg/kg-day (3% higher than controls). Serum chemistry changes that were statistically significant included decreased ALP in males at 50 and 150 mg/kg-day (31 and 28% less than controls), decreased AST in males at 50 and 150 mg/kg-day (22 and 19% less than controls), decreased ALT in males at 50 and 150 mg/kg-day (18 and 18% less than controls), and increased glucose at 50 mg/kg-day (16% higher than controls; similar increases occurred at 17 and 150 mg/kg-day but were not statistically significant). Although statistically significant changes were observed for these and several other hematology and clinical chemistry indices, no responses were clearly dose-related, consistent between sexes or, according to the authors, outside normal ranges or biologically significant. There were no clear effects on organ weights; the only statistically significant changes were increased relative liver weight in females at 17 mg/kg-day, increased absolute spleen weight in males at 17 and 50 mg/kg-day, and increased absolute brain weight in males at 50 mg/kg-day; quantitative data were not reported. There were no gross or histopathological changes in either sex. The lack of any clear treatment-related or biologically significant hematology, clinical chemistry or organ weight changes, as well as the lack of any pathological effects, indicates that the highest dose level, 150 mg/kg-day, is a NOAEL for 90-day gavage exposure in rats.

The oral toxicity of 2-chlorophenol was also assessed in 18-day studies with preweanling rats and in 14- and 28-day studies with juvenile rats (Hasegawa et al., 2005). Preweanling Sprague-Dawley SPF rats were administered 2-chlorophenol in olive oil by gavage on postnatal

days (PNDs) 4-21 in dose-finding and main studies. In the 18-day dose-finding study with preweanling rats, groups of 4 males and 4 females were exposed to dose levels of 0, 20, 100 or 500 mg/kg-day. General behavior and body weight were evaluated during the study, and hematology, blood chemistry, gross pathology and organ weights were evaluated on PND 22; histopathology was not assessed. Although not specifically reported, it is assumed that the scope of these evaluations was the same as in the main study with newborn rats summarized below. Effects were limited to 100% mortality by the 9th day of dosing at 500 mg/kg-day; clinical signs were not observed at 20 and 100 mg/kg-day, and no other results were reported. This study identified a FEL of 500 mg/kg-day for lethality in preweanling rats. The next lowest dose level of 100 mg/kg-day is a NOAEL based on the lack of clinical signs and systemic effects, but confidence in this effect level is low due to the small numbers of animals and lack of histological examinations.

In the main 18-day study with preweanling rats, groups of 12 male and 12 female Sprague-Dawley SPF rats were administered 2-chlorophenol in olive oil by gavage in doses of 0, 8, 50 or 300 mg/kg-day on PNDs 4-21 (Hasegawa et al., 2005). Half of the animals were sacrificed on PND 22, and the remaining 6 rats/sex/group were observed without treatment for the following 9 weeks and then sacrificed (on PND 85). Endpoints evaluated during the study included general behavior, body weight and postnatal developmental parameters, including surface righting and visual placing reflex for reflex ontogeny, fur appearance, incisor eruption and eye opening for external development, and preputial separation, vaginal opening and estrous cycle for sexual development. Comprehensive hematology and blood biochemistry evaluations were conducted at the end of the treatment period on PND 22 (6 rats/sex/dose) and end of the observation period on PND 85 (6 rats/sex/dose). Hematology indices included RBC, Hct, Hgb, MCV, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, total and differential WBC, platelet count, and reticulocyte count. Blood biochemistry indices included total protein, albumin, albumin/globulin ratio, glucose, total cholesterol, triglycerides, phospholipid, total bilirubin, BUN, creatinine, AST, ALT, ALP, γ -glutamyl transpeptidase, calcium, inorganic phosphorus, sodium, potassium and chloride. Prothrombin time, activated thromboplastin time, and urine indices (color, pH, occult blood, protein, glucose, ketone bodies, bilirubin, urobilinogen, sediment, volume and osmotic pressure) were evaluated only at the end of the observation period. Organ weights (brain, pituitary, thymus, thyroids, heart, lungs, liver, spleen, kidneys, adrenals, testes, epididymides, ovaries and uterus) and histopathology (organs that were weighed as well as macroscopically abnormal organs) were evaluated on PND 22 (6 rats/sex/dose); it was not indicated if these evaluations were performed on PND 85.

Effects included tremors in 11/12 males and 12/12 females at 300 mg/kg-day; the tremors appeared within 5 minutes of dosing and disappeared within 4 hours in most animals. At 50 mg/kg-day, 1/12 females showed tremors once from 15-30 minutes following dosing on treatment day 9. No tremors were observed in males at 50 mg/kg-day or in either sex at 0 or 8 mg/kg-day. The only other reported effects occurred at 300 mg/kg-day; these consisted of other signs of neurotoxicity (hypoactivity in 2/12 males and 3/12 females and abnormal gait in 1/12 males and 1/12 females), transiently decreased body weight in both sexes (additional information not reported), and histological changes in the kidneys (slight to moderate basophilic renal tubules in 4/6 males and 5/6 females) with increases in relative kidney weight (8% in males and 4% in females). The biological significance of the basophilic renal tubular changes was not discussed.

No results were reported for the 9-week observation period. The 300 mg/kg-day dose is a FEL for preweanling rats based on the occurrence of tremors in 23/24 of the exposed males and females; other signs of neurotoxicity (hypoactivity and abnormal gait) were also observed at this dose level. The next lowest dose of 50 mg/kg-day is a NOAEL because tremors were only observed in 1/12 females once on exposure day 9; the incidence is not statistically different from the control group (0/12) and the occurrence was isolated. Additionally, there were no clinical signs of neurotoxicity in the males exposed to 50 mg/kg-day, or in the 4 males and 4 females exposed to 100 mg/kg-day in the dose-finding study summarized above.

The studies with juvenile rats included a 14-day dose-finding study and a 28-day main study (Hasegawa et al., 2005). In the 14-day dose-finding study, 5-week-old male and female Sprague-Dawley SPF rats were administered 2-chlorophenol in olive oil by gavage in doses of 0, 100, 200 or 500 mg/kg-day; group sizes were 3 per sex at 500 mg/kg-day and were not reported for the other dose levels. General behavior, body weight and food consumption were evaluated during the study, and hematology, blood chemistry, gross pathology and organ weights were evaluated the day after the last treatment; histopathology was not assessed. Although not specifically reported, it is assumed that the scope of these evaluations was the same as in the study with newborn rats summarized above. The only information regarding the results is a statement that no toxic signs were observed, indicating that 500 mg/kg-day is a NOAEL in juvenile rats. Confidence in this effect level is low due to the apparent small numbers of animals and lack of histological examinations.

In the 28-day main study, groups of 12 male and 12 female 5- to 6-week old Sprague-Dawley SPF rats were exposed to 2-chlorophenol in olive oil by gavage in doses of 0, 8, 40, 200 or 1000 mg/kg-day (Hasegawa et al., 2005). It appears that half of the animals were sacrificed following the last treatment and the remaining 6 rats/sex/group were observed without treatment for the following 2 weeks and then sacrificed. Evaluations included general behavior, body weight, food consumption, urinalysis, hematology, blood biochemistry, gross pathology, organ weights and histopathology. Although not specifically reported, it is implied that the scope and schedule of these evaluations are the same as in the 18-day study with preweanling rats summarized above. The only effects in this study were clinical signs of neurotoxicity and histological changes in the liver in most animals only at 1000 mg/kg-day. The clinical signs occurred sporadically in both sexes within 3 hours of dosing and included tremors (4/12 males and 5/12 females), hypoactivity (8/12 males and 5/12 females) and abnormal gait (4/12 males and 7/12 females). The liver effects consisted of slight centrilobular hypertrophy of hepatocytes (6/6 males and 5/6 females); the authors indicated that this suggested a compensatory response for hepatic metabolism. None of the animals showed basophilic renal tubules as observed in the preweanling rats exposed to 300 mg/kg-day on PNDs 4-21 (see above). No results were reported for the 2-week observation period. This study identified a FEL of 1000 mg/kg-day based on the clinical signs of neurotoxicity; the NOAEL is 200 mg/kg-day.

Additional information on effects of repeated oral exposures to 2-chlorophenol is available from a series of reproductive toxicity, immunotoxicity and carcinogenicity studies in Sprague-Dawley rats that were exposed prenatally, postnatally, or both pre- and postnatally to concentrations of 0, 5, 50 or 500 ppm 2-chlorophenol in drinking water (Exon and Koller, 1982, 1983a,b, 1985). Offspring produced in the reproductive study were used in the immunotoxicity

and carcinogenicity studies. In the reproductive study, groups of 12-14 females were exposed to the treated drinking water from 3 weeks of age through breeding (to untreated males) at 90 days of age and subsequently until 3 weeks post-parturition (Exon and Koller, 1982, 1983b, 1985). Table 1 shows the statistically significant reproductive endpoints that were reported by Exon and Koller (1982). The values found in Exon and Koller (1983b, 1985) agree with each other but are slightly different from those found in Exon and Koller (1982), and differ in their statistical evaluation. The reason for the differences is unknown. Maternal and pup weight, percent conception, litter size, and number of stillbirths were evaluated at parturition. Pup survival, body weight and hematology (red and white cell counts, hemoglobin, packed cell volume, and mean corpuscular volume) were evaluated at weaning.

Table 1. Reproductive effects of 2-Chlorophenol in Rats				
Effect	Dose (ppm)			
	0	5	50	500
Litter Size (mean \pm SD)	11.4 \pm 1.2 n=12	11.7 \pm 3.5 n=12	10.1 \pm 2.3 n=12	9.2 \pm 4.3 ^b n=14
Stillborn (incidence)	0/91	2/105	0/91	6/110 ^b
^a Female rats were exposed to 2-chlorophenol in drinking water from 3 weeks of age through mating at 90 days of age and subsequently through pregnancy and lactation.				
^b Significantly different from control group (p \leq 0.05).				
Source: Exon and Koller (1982)				

Statistically significant (p \leq 0.05) changes included 19% reduced mean litter size (live and stillborn pups) at 500 ppm (9.2 \pm 4.3 compared to 11.4 \pm 1.2 in controls) and 5% increased incidence of stillbirths at 500 ppm (6/110 compared to 0/91 in controls) (Exon and Koller, 1982).

Based on the evidence of decreased litter size and an increase in stillbirth incidence, this study identified a NOAEL of 50 ppm and a LOAEL of 500 ppm for reproductive toxicity. The conversion factor for converting the amount of 2-chlorophenol ingested in drinking water (ppm) to a dose (mg/kg-day) was calculated by dividing the reference water consumption of 0.031 L/day for female Sprague Dawley rats in a subchronic study by the corresponding reference body weight in female Sprague Dawley rats (0.031 L/day/0.204 kg = 0.15 L/kg-day) (U.S. EPA, 1988b). Thus, the 5, 50 and 500 ppm doses correspond to estimated drinking water doses of 0.75, 7.5 and 75 mg/kg-day, respectively, and the NOAEL and LOAEL correspond to 7.5 and 75 mg/kg-day, respectively.

In the immunotoxicity studies, offspring from female rats described in the above studies that were exposed to 0, 5, 50 or 500 ppm 2-chlorophenol in drinking water from 3 weeks of age through mating at 90 days until 3 weeks post-parturition were continued on treatment for 10 weeks (Exon and Koller, 1983a) or 15 weeks (Exon and Koller, 1985), at which time immune responses were evaluated. Tests were conducted for humoral immunity (measured as the ratio of serum Immunoglobulin gamma (IgG) antibody levels to bovine serum albumin or keyhole limpet hemocyanin), cell-mediated immunity (measured as delayed-type hypersensitivity response in ears injected with oxazolone), and macrophage function (measured as the ability of peritoneal exudate cells to phagocytize sheep red blood cells *in vitro*) in 4 male and 4 female offspring from each exposure group. Body, liver, spleen, and thymus weights were also evaluated in these

offspring. There were no statistically significant ($p \leq 0.05$) differences between the treated and control groups for any of the immune responses or other end points, indicating that a NOAEL of 500 ppm was identified. Using conversion factors of 0.14 and 0.15 L/kg-day based on subchronic values for water consumption and body weight in male and female Sprague Dawley rats (U.S. EPA, 1988b), respectively, the NOAEL of 500 ppm identified in these studies corresponds to estimated drinking water doses of 70 mg/kg-day in males and 75 mg/kg-day in females.

In the carcinogenicity studies (Exon and Koller, 1983b, 1985), groups of 24-32 male and 24-28 female rats received combined pre- and postnatal exposures to 0, 5, 50 or 500 ppm of 2-chlorophenol in drinking water. Three-week-old females were exposed continuously through mating (90 days of age), pregnancy and lactation, and the offspring received treated drinking water from weaning for 24 months. All rats were observed daily for gross signs of morbidity, and moribund or tumor-bearing rats were sacrificed. Body weight was measured monthly in all rats, and hematology (RBC, WBC, Hct, Hgb and MCV) was evaluated every 2 weeks (Exon and Koller, 1983b) or every 2 months (Exon and Koller, 1985) in 5 males and 5 females per group. Gross and microscopic examinations of major organs and tumor tissues were conducted in all animals. There were no effects on body weight at 15 weeks (Exon and Koller, 1985) or 7 months (Exon and Koller 1983b), the only times for which data were reported. A significant decrease in body weight ($p \leq 0.10$) was observed at 7 months in females at doses of 5 and 500 ppm (7.6 and 5.2% less than controls respectively). Exon and Koller (1985) noted that red blood cell count, packed cell volume and blood hemoglobin concentrations were “generally increased” in both sexes at 500 ppm. These effects were most evident after 14 months of exposure, when the RBC, packed cell volume (PCV) and hemoglobin values were 15, 19 and 16% higher than controls ($p \leq 0.05$), respectively; no other quantitative hematology data were reported. In an earlier report of interim (15-month) findings, however, Exon and Koller (1983b) indicated that 2-chlorophenol did not affect any of the measured hematology parameters. Noncancer histopathologic observations were not reported. Although there were no clear treatment-related or biologically significant body weight or hematology changes, the lack of noncancer histopathology data precludes identification of a NOAEL or LOAEL for chronic toxicity. There were no statistically significant ($p \leq 0.10$) differences between exposed and control groups in tumor incidence, latency or type in either sex. Incidences of total tumors in the 0, 5, 50 and 500 ppm groups were 13, 17, 8 and 18% in males, and 5, 0, 13, and 18% in females, respectively; no other incidence data were reported.

Inhalation Exposure. Relevant information regarding the inhalation toxicity of 2-chlorophenol in animals was not located.

Other Studies

Co-carcinogenicity and Tumor Promotion. In a co-carcinogenicity study (Exon and Koller, 1983b, 1985), groups of 24-32 male and 24-28 female Sprague-Dawley rats were exposed prenatally, postnatally, or both pre- and postnatally to 0, 5, 50 or 500 ppm 2-chlorophenol in drinking water, with prenatal exposure to the known carcinogen ethylnitrosourea (ENU). Comparison groups received prenatal exposure to ENU alone; comparisons were not made to offspring unexposed to ENU or 2-chlorophenol. Rats were exposed to ENU as its precursors,

ethylurea (0.316% in feed) and sodium nitrite (1 ppm in drinking water), on days 14-21 of gestation. Prenatal exposure to 2-chlorophenol involved exposing 3-week-old females through mating (90 days of age) and pregnancy; the dams were not exposed during lactation, and the offspring were observed without treatment from weaning for 24 months. Postnatal exposure to 2-chlorophenol involved exposing offspring from unexposed dams to the treated water from weaning for 24 months. Combined pre- and postnatal exposure to 2-chlorophenol involved exposing 3-week-old females continuously through mating (90 days of age), pregnancy and lactation, and subsequent exposure of the offspring to the treated water from weaning for 24 months. Histological examinations were performed on major organs and grossly observed tumors, but data were only reported for total tumors.

Male offspring of rats treated with ENU and combined pre- and postnatal exposure to 2-chlorophenol, at all treatment levels, had significantly ($p \leq 0.10$) increased incidences of total tumors when compared to the group exposed to ENU alone (Table 2). Significantly higher incidences of total tumors also occurred in male offspring exposed to ENU and 2-chlorophenol given prenatally at 5 and 500 ppm (but not 50 ppm), male offspring exposed to ENU and 2-chlorophenol given postnatally at 5 ppm, and female offspring exposed to ENU and 2-chlorophenol given prenatally or postnatally at 500 ppm. Tumor latency (mean days to tumor) was significantly decreased in rats exposed to ENU with combined pre- and postnatal exposure to 2-chlorophenol at all treatment levels when compared to the group exposed to ENU alone. Although total tumor incidence was increased and time-to-tumor latency was decreased in all groups of male rats with combined pre- and postnatal exposure to 2-chlorophenol compared with those exposed to ENU alone, interpretation of the findings is complicated by a high tumor incidence in the group exposed to ENU alone, lack of a dose-response relationship, and lack of similar effects in females (Table 2). The authors concluded that the results suggest that 2-chlorophenol may act as a co-carcinogen or promoter of carcinogenesis.

Table 2. Tumor Incidence and Latency in Rats Exposed Pre- and Postnatally to 2-Chlorophenol with Prenatal Exposure to ENU (Exon and Koller, 1983b)						
2-Chlorophenol (ppm) (Pre-and Postnatal + ENU)	Total Tumor Incidence (%)			No. Rats/Group		Days to Tumor (mean \pm SE)
	Total	Male	Female	Male	Female	
Unexposed	3	7	0	30	30	422 \pm 40
ENU only	58	54	63	28	24	302 \pm 16
5	85 ^a	92 ^a	79	24	24	245 \pm 14 ^a
50	63	75 ^a	50	24	24	256 \pm 17 ^a
500	68	77 ^a	60	30	30	259 \pm 14 ^a
^a $p \leq 0.10$ compared to ENU positive control group by chi-square test (incidence data) or analysis of variance (least-square means) (latency data).						

The skin tumor-promoting ability of 2-chlorophenol was assessed in 2- to 3-month old female albino Sutter mice (Boutwell and Bosch, 1959). When 25 μ l of a 20% solution of 2-chlorophenol in benzene was applied to shaved back skin twice weekly for 15 weeks following initiation with a single 25 μ l application of 0.3% DMBA (9,10-dimethyl-1,2-benz[a]anthracene) in benzene, 31/35 mice survived compared to 15/20 similarly initiated vehicle control mice. Of the survivors, 61% had skin papillomas compared to 7% in controls, and 10% had skin

carcinomas compared to 0% in controls. When 2-chlorophenol was applied as a 20% solution in dioxane to uninitiated mice twice weekly for 12 weeks, 28/30 mice survived; 46% of the survivors had papillomas and 0% developed carcinomas. A dioxane-treated vehicle control group was not reported.

Genotoxicity. A limited amount of information is available on the genotoxicity of 2-chlorophenol. 2-Chlorophenol did not induce reverse mutations in *Salmonella typhimurium* strains TA98, TA100, TA1535 and TA1537 when tested with or without exogenous metabolic activation (Haworth et al., 1983). 2-Chlorophenol did not induce DNA-repairing genes (*umuDC*) in *S. typhimurium* TA1535/pSK1002 (Ono et al., 1992), or DNA damage in *Escherichia coli* as shown by the induction of prophage lambda (DeMarini et al., 1990), when tested with or without exogenous metabolic activation. Sister-chromatid exchanges were not increased in mice that were exposed to 2-chlorophenol in corn oil by gavage in doses of 35-175 mg/kg-day for 14 days (Borzelleca et al., 1985); bone marrow and testicular cells (specific cell types not indicated) were examined.

DERIVATION OF A PROVISIONAL SUBCHRONIC ORAL RfD FOR 2-CHLOROPHENOL

Subchronic RfD

Information relevant to the derivation of a subchronic oral RfD for 2-chlorophenol is available from one 14-day study in mice (Borzelleca, 1983; Borzelleca et al., 1985) and several studies in rats ranging in exposure duration from 10 days to approximately 16-21 weeks (Daniel et al., 1993; Exon and Koller, 1982, 1983a, 1985; Hasegawa et al., 2005). The preponderance of these studies used gavage exposure and showed frank toxic effects, particularly mortality and clinical signs of neurotoxicity, as summarized in Table 3. The gavage studies identified FELs of 175 mg/kg-day for mortality in mice exposed for 14 days (Borzelleca, 1983; Borzelleca et al., 1985), 300 mg/kg-day for overt neurotoxicity (tremors) and 500 mg/kg-day for mortality in preweanling rats exposed for 18 days on PNDs 4-21 (Hasegawa et al., 2005), and 1000 mg/kg-day for overt neurotoxicity (tremors, hypoactivity and abnormal gait) in rats exposed for 28 days (Hasegawa et al., 2005). Although these are generally well-designed studies with comprehensive evaluations that included clinical signs, body weight, hematology, clinical chemistry, organ weights, histology and, in the study with preweanling rats, postnatal developmental indices, they did not identify more subtle indicators of toxicity and actual data were not supplied in some instances. NOAELs in the gavage studies were 69 mg/kg-day in mice exposed for 14 days (Borzelleca, 1983; Borzelleca et al., 1985), 50 and 100 mg/kg-day in preweanling rats exposed for 18 days on PNDs 4-21 (Hasegawa et al., 2005), 150 mg/kg-day in rats exposed for 90 days (Daniel et al., 1993), and 200 mg/kg-day in rats exposed for 28 days (Hasegawa et al., 2005).

Table 3. Summary of Effect Levels from Oral Toxicity Studies of 2-Chlorophenol

Species	Exposure Duration	NOAEL ^a	LOAEL ^a	FEL ^a	Effects	Reference
mouse	14 days (gavage)	69	ND	175	100% mortality at 175 mg/kg-day. No biologically significant effects at 69 mg/kg-day ^{b,c} .	Borzelleca, 1983; Borzelleca et al., 1985
rat	10 days (gavage)	257	ND	ND	No clear treatment-related or biologically significant effects ^b .	Daniel et al., 1993
rat	90 days (gavage)	150	ND	ND	No clear treatment-related or biologically significant effects ^{b,d} .	Daniel et al., 1993
rat	18 days (PND 4-21) (gavage)	100	ND	500	100% mortality at 500 mg/kg-day. No effects at 100 mg/kg-day but small numbers of rats were tested. Dose-finding study with no histology ^b .	Hasegawa et al., 2005
rat	18 days (PND 4-21) (gavage)	50 ^e	ND	300	Tremors in 23/24 males and females at 300 mg/kg-day. No clear treatment-related effects at 50 mg/kg-day ^{b,d} .	Hasegawa et al., 2005
rat	14 days (gavage)	500	ND	ND	No clinical signs or other effects but small numbers of rats were tested. Dose-finding study with no histology ^b .	Hasegawa et al., 2005
rat	28 days (gavage)	200	ND	1000	Tremors, hypoactivity, abnormal gait and centrilobular hepatocellular hypertrophy at 1000 mg/kg-day. No reported effects at 200 mg/kg-day ^{b,d} .	Hasegawa et al., 2005
rat	16 weeks ^f (drinking water)	7.5	75	ND	Reduced litter size (19%) and increased incidence of stillbirths.	Exon and Koller, 1982, 1985
rat	16-21 weeks ^g (drinking water)	75	ND	ND	No effects on immune responses ^h or body, liver, spleen or thymus weights. Other endpoints not evaluated.	Exon and Koller, 1983a, 1985

ND = not determined

^amg/kg-day^bEndpoints included clinical signs, body weight, hematology, serum chemistry, organ weights and gross pathology.^cEndpoints included immune responses and behavioral tests.^dEndpoints included histopathology.^eThe only reported effect was tremors in 1/12 females that occurred once on treatment day 9.^fFemale rats were exposed from 3 weeks of age through mating to untreated males at 90 days of age and subsequently through pregnancy and lactation.^gOffspring of female rats that were exposed from 3 weeks of age through mating to untreated males at 90 days of age and subsequently through pregnancy and lactation were continued on treatment for 10-15 weeks.^hTests for humoral immunity, cell-mediated immunity and macrophage function were conducted.

Drinking water studies (Exon and Koller, 1982, 1983a and b, 1985) investigated reproductive and immunological toxicity in rats. There were no effects on immune function in rats that were exposed to 75 mg/kg-day via maternal drinking water during gestation and lactation and subsequently by direct consumption for 10-15 weeks (Exon and Koller, 1983a, 1985). Exposure to 75 mg/kg-day in drinking water during pregnancy and lactation significantly ($p \leq 0.05$) affected litter size (19% reduced) and stillbirths (5% increased) in rats (Exon and Koller 1982, 1985); no effects on litter size occurred at 7.5 mg/kg-day. Therefore, reproductive toxicity as evidenced by decreased litter size and an increase incidence in stillbirths was chosen for the development of the subchronic RfD for 2-chlorophenol based on a NOAEL of 7.5 mg/kg-day (Exon and Koller, 1982).

The NOAEL of 7.5 mg/kg-day is divided by a composite uncertainty factor of 1000 to derive a provisional **subchronic RfD of 8E-3 mg/kg-day**, as follows:

$$\begin{aligned} \text{sRfD} &= \text{NOAEL} / \text{UF} \\ &= 7.5 \text{ mg/kg-day} / 1000 \\ &= \mathbf{0.0075 \text{ or } 8\text{E-3 mg/kg-day}} \end{aligned}$$

The composite UF of 1000 includes a factor of 10 for animal-to-human extrapolation, 10 for interindividual variability and 10 for database deficiencies.

The animal-to-human UF of 10 reflects a factor of three ($10^{1/2}$) for pharmacokinetic differences across species and a factor of three ($10^{1/2}$) for pharmacodynamic considerations.

The intraspecies UF of 10 is used to account for variation in sensitivity within human populations because there is limited information on the degree to which humans of varying gender, age, health status or genetic makeup might vary in the disposition of, or response to, the chemical.

An UF for extrapolation from a LOAEL to a NOAEL is not necessary because a NOAEL was chosen for the point of departure for the derivation for the sRfD.

The UF of 10 for database deficiencies is applied due to the lack of comprehensive reproductive and developmental toxicity studies, including a two-generation reproductive toxicity study and a subchronic study in mice (see below).

Confidence in the key study is low because a limited number of reproductive/developmental endpoints (maternal and pup weight, percent conception, litter size and number of stillborn) were evaluated and the adequacy of the reporting is marginal. Confidence in the database is also low. The database includes 18-day, 28-day and 90-day studies in rats that assessed systemic toxicity and postnatal developmental toxicity at doses that include the range of those tested in the key study. Deficiencies in the database include the lack of comprehensive reproductive and developmental toxicity studies (especially important because reproductive effects have been identified as critical for this chemical) and a subchronic toxicity study longer than 14 days in duration in mice, which appeared to be more sensitive than rats to the subchronic effects of the chemical. In addition, a two-generation reproductive toxicity study is not

available. Considering the levels of confidence in the key study and data base and the lack of supporting data for the critical effects, confidence in the provisional RfD is low.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfC VALUES FOR 2-CHLOROPHENOL

No information is available on the subchronic or chronic inhalation toxicity of 2-chlorophenol, precluding derivation of RfC values for this chemical.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2-CHLOROPHENOL

Weight-of-evidence Classification

Information regarding the carcinogenicity of 2-chlorophenol mainly consists of the negative results of a drinking water study in which rats were exposed to 0, 5, 50 or 500 ppm via maternal consumption during pregnancy and lactation and subsequently by direct consumption for 24 months (Exon and Koller 1983b, 1985). There were no significant increases in tumor incidence, latency or type in either sex, but a definitive conclusion regarding carcinogenicity is precluded by the use of marginal numbers of animals for a cancer bioassay (24-32/sex/dose level) and the apparent lack of a MTD, because the only observed effects (body weight and hematology changes) were not clearly treatment-related or biologically significant.

The ability of 2-chlorophenol to act as a promoter or co-carcinogen was investigated in a study with the known carcinogen ENU (Exon and Koller 1983b, 1985). Male rats that were exposed to 0, 5, 50 or 500 ppm of 2-chlorophenol in drinking water via maternal consumption during pregnancy and lactation and subsequently by direct consumption for 24 months, combined with prenatal exposure to ENU, had increased total tumor incidences and decreased time-to-tumor latencies compared to rats exposed to ENU alone. Another study found that dermal application of 2-chlorophenol promoted the formation of DMBA-initiated skin tumors in mice (Boutwell and Bosch, 1959).

2-Chlorophenol has been studied in several short term *in vitro* and *in vivo* animal studies. 2-Chlorophenol did not induce reverse mutations or DNA-repair in *S. typhimurium* (Haworth et al., 1983; Ono et al., 1992), DNA damage in *E. coli* (DeMarini et al., 1990), or sister-chromatid exchanges in orally-exposed mice (Borzelleca et al., 1985).

In accordance with current EPA cancer guidelines (U.S. EPA, 2005), the available data are inadequate for an assessment of human carcinogenic potential.

Quantitative Estimates of Carcinogenic Risk

Derivation of quantitative estimates of cancer risk for 2-chlorophenol is precluded by the lack of data demonstrating carcinogenicity associated with 2-chlorophenol exposure.

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9-20-2007

Provisional Peer Reviewed Toxicity Values for

2-Nitrophenol
(CASRN 88-75-5)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2-NITROPHENOL (CASRN 88-75-5)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Neither a reference dose (RfD), reference concentration (RfC), nor carcinogenicity assessment is available for 2-nitrophenol in the Integrated Risk Information System (IRIS) database (U.S. EPA, 2007), the Health Effects Assessment Summary Table (HEAST) (U.S. EPA, 1997), or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2006). The Chemical Assessments and Related Activities (CARA) database (U.S. EPA, 1991, 1994a) lists a Health Effects Assessment (HEA) (U.S. EPA, 1987) and a Health and Environmental Effects Profile (HEEP) (U.S. EPA, 1985) for Nitrophenols in which limited toxicity data for 2-nitrophenol are available. An Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Nitrophenols (2-Nitrophenol and 4-Nitrophenol) (ATSDR, 1992) also includes only limited toxicity data for 2-nitrophenol. Neither the American Conference of Governmental Industrial Hygienists (ACGIH, 2006), the National Institute of Occupational Safety and Health (NIOSH, 2006) nor the Occupational Safety and Health Administration (OSHA, 2006) has adopted occupational exposure limits for 2-nitrophenol. Health assessments for 2-nitrophenol are not available from CalEPA (2006) or the International Agency for Research on Cancer (IARC, 2006). Pertinent data was found for 2-nitrophenol after examining the Concise International Chemical Assessment Document (CICAD) for mononitrophenols (WHO, 2000). Relevant information for 2-nitrophenol from the National Toxicology Program (NTP, 2006) is limited to genotoxicity assays.

Literature searches covering the time period 1960's to August, 2006 were conducted in PUBMED, TOXLINE, and DART/ETIC to identify information relevant to 2-nitrophenol. TOXCENTER was searched for the time period August, 2001 to August 2006. Databases

searched without date limitations included TSCATS/TSCATS2, CCRIS, GENETOX, HSDB and RTECS. Search of Current Contents encompassed May to August, 2006.

REVIEW OF PERTINENT DATA

Human Studies

No data were located regarding the toxicity or carcinogenicity of 2-nitrophenol in humans following oral or inhalation exposure.

Animal Studies

Oral Exposure. Available repeated-dose oral studies consist of two limited 28-day gavage studies (Andrae et al., 1981; Koerdel et al., 1981; both in German) performed to evaluate OECD guideline 407 and a range-finding developmental toxicity study (IRDC, 1990).

Andrae et al. (1981) administered 2-nitrophenol to groups of Sprague-Dawley rats (10/sex/dose) at gavage doses of 0, 70, 210 or 630 mg/kg-day for 28 days. Because the original German report of this study was not available, information from the CICAD for mononitrophenols (WHO, 2000) was used to summarize the findings. Mid- and high-dose animals exhibited what was described by the WHO (2000) as locomotor inhibition for approximately 2 hours postdosing. Mortality rates were 1/10 in mid-dose males and 4/10 and 6/10 in high-dose males and females, respectively. Gross and histopathological examinations revealed pale liver in 7/20 low-dose rats (not reported by sex), hydropic liver cell swelling in 4/10 and 0/10 high-dose males and females, respectively, and vascular congestion of the liver in all high-dose male and female rats that died prior to terminal sacrifice. Fatty degeneration of the liver was noted in 6/20 control animals, 14/20 low-dose and 13/20 mid-dose rats, but not in high-dose rats. Other treatment-related effects, noted only at the highest dose level, included significantly increased alanine aminotransferase activity in males (data not reported), increased nephrosis in 2 and 5 males and females, respectively, testicular atrophy (1 male) and decreased spermatogenesis (2 males), and follicular atresia (4 females). This report did not contain information on hematological effects. WHO (2000) concluded that a NOAEL could not be determined for this study due to “unclear effects in the liver.”

Koerdel et al. (1981) administered 2-nitrophenol to groups of rats (5/sex/dose) at gavage doses of 0, 22, 67 or 200 mg/kg-day for 28 days. The summary from WHO (2000) was used as the source of study details because the original study was not available. Reported treatment-related effects included decreased food intake in high-dose males and mid- and high-dose females, non-significantly depressed final body weight in all dosed animals, decreased absolute liver and kidney weights in mid-dose groups, increased relative testes weight in low- and mid-dose males (decreased in high-dose males) and increased absolute and relative adrenal weight in all dosed groups. Hematology, clinical chemistry and histopathological examinations gave no indication of treatment-related effects. The study did not show a clear dose-response relationship for any of the endpoints examined.

In a range-finding developmental toxicity study, groups of Charles River COBS CD rats (5 dams/group) were administered 2-nitrophenol (in corn oil) at gavage doses of 0, 50, 125, 250, 500, or 1000 mg/kg-day on days 6-15 of gestation (IRDC, 1990). Body weights were determined during the treatment period and clinical signs were noted. Uterine examinations were performed on gestation day 20. A single high-dose dam died, but cause of death was not determined. Excessive salivation was observed in two high-dose dams. Mean maternal body weight gains in the 0, 50, 125, 250, 500 and 1000 mg/kg-day dose groups were 8, 7, 5, 6, 1 and -8 grams, respectively, for the initial 4 days of treatment (gestation days 6-9) and 52, 56, 54, 55, 45 and 39 grams, respectively, for the entire treatment period (gestation days 6-15). The appearance and behavior of the 50 mg/kg-day group of dams were comparable to the control group. Dose-related increases in the incidence of yellow staining around the nose, mouth and anogenital area were observed at doses ≥ 125 mg/kg-day. Dose-related increases in the incidence of darkly colored urine (probably due to the presence of the test chemical) occurred at doses ≥ 250 mg/kg-day. An increase in the number of early resorptions was observed in the highest dose group (2.3 versus 1.2 in controls), resulting in mean postimplantation loss of 13.8% compared to 8.2% in controls (statistical significance not reported). Among dams surviving until necropsy, no biologically significant treatment-related effects were seen. There were no biologically significant treatment-related effects on mean number of viable fetuses, implantations or *corpora lutea*. No data on hematological parameters were included in this study. This study assessed a limited number of potential adverse endpoints and is therefore of limited usefulness for risk assessment.

Inhalation Exposure. Available information for repeated inhalation exposure is restricted to results of a single 28-day study (Hazleton Laboratories, 1984). Groups of 7-week-old Sprague-Dawley rats (15/sex/group) were exposed to 2-nitrophenol vapors at target concentrations of 0, 5, 30 or 60 mg/m³ for 6 hours/day, 5 days/week for 4 weeks. All rats were subjected to ophthalmoscopic examinations prior to initiation of exposures and immediately preceding terminal sacrifice. Each animal was observed twice daily (pre- and postexposure during the week; morning and afternoon on weekends) for mortality and morbidity. Clinical signs and body weights and weight gains were assessed throughout the study. Following the 11th and 20th exposures, blood was collected by orbital sinus puncture from 10 rats/sex/group and analyzed for methemoglobin concentrations. At termination of the study (day 29), blood was collected via the abdominal aorta from 10 anesthetized rats/sex/group for hematology and serum chemistry. At necropsy, all rats were subjected to comprehensive gross examinations and organ weights were recorded. Comprehensive histopathological examinations were performed on 10 rats/sex in the 0 and 60 mg/m³ exposure groups. Nasal turbinates were examined histopathologically in 10 rats/sex of each exposure group.

Overall mean analytical concentrations deviated from the target concentrations by 0.0, +8.3 and +2.5% for the 5, 30 and 60 mg/m³ exposure groups, respectively (Hazleton Laboratories, 1984). The aerosol content of the exposure chambers was not significantly different from that present in room air. No significant exposure-related ocular lesions were apparent in any of the rats. No animals died during the study. No apparent exposure-related trends in clinical signs were apparent with the exception of yellow stains on the fur of all 2-nitrophenol exposed animals. There were no statistically significant exposure-related effects on mean body weight or weight gain. A statistically significant increase in methemoglobin

levels was noted in male and female rats of the 5 mg/m³ group analyzed on day 15 of the study. However, when animals were analyzed on day 28, the methemoglobin levels were similar to controls. No statistically significant increases were found in the higher dose groups. The change, compared with controls, in methemoglobin levels in treated animals of the low dose groups, while exhibited statistical significance, was not considered biologically significant. Hematology and clinical chemistry findings were unremarkable. Gross pathology revealed no consistent exposure-related trends. Small increases in liver weight, liver/brain weight ratio and spleen/brain weight ratio were seen in the 5 mg/m³ group females, but were not observed in females at higher doses or in any of the treated males. Histopathological examinations revealed squamous metaplasia in epithelium of the nasoturbinates and maxilloturbinates in 1/10, 0/10, 10/10 and 10/10 male rats and 1/10, 1/10, 9/10 and 10/10 female rats of the 0, 5, 30 and 60 mg/m³ exposure groups, respectively. No other apparent exposure-related effects were observed. On the basis of the nasal lesions, this study identified a NOAEL of 5 mg/m³ and a LOAEL of 30 mg/m³ for 2-nitrophenol in rats.

Other Studies

Limited genotoxicity data are available for 2-nitrophenol. The chemical produced negative results in the Ames test with *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 both in the presence and absence of rat liver S9 metabolic activation (Chiu et al., 1978; Dellarco and Prival, 1989; Haworth et al., 1983; Kawai et al., 1987; Koerdel et al., 1981; Massey et al., 1994; Shimizu and Yano, 1986; Suzuki et al., 1983). 2-Nitrophenol did not induce DNA breakage in λ phage DNA (Yamada et al., 1987) or increase reversions from streptomycin dependence to independence in *Escherichia coli* strain Sd-4-73 (Szybalski, 1958). Negative results were reported for mutagenic activity in post-meiotic and meiotic germ cells of male *Drosophila melanogaster* exposed to 2-nitrophenol via feeding (400-500 ppm) or injection (2500 or 5000 ppm) (Foureman et al., 1994).

2-Nitrophenol did not exhibit skin tumor-promoting action in mice receiving dermal applications of a 20% solution twice weekly for 12 weeks (Boutwell and Bosch, 1959).

In rats and mice administered single oral doses of 2-nitrophenol, calculated LD₅₀ values were 2830 and 1300 mg/kg, respectively (Vernot et al., 1977). No information was located regarding the toxicity of 2-nitrophenol following acute inhalation exposure.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC RfD VALUES FOR 2-NITROPHENOL

Oral studies of 2-nitrophenol are limited to two 28-day studies from the German literature available only as brief summaries in WHO (2000) and a range-finding developmental toxicity study. None of these studies appear to have been adequate to derive NOAEL or LOAEL values. The lack of adequate oral data for humans or animals precludes the derivation of a provisional subchronic or chronic RfD for 2-nitrophenol.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfC VALUES FOR 2-NITROPHENOL

Subchronic p-RfC

Results of the only available repeated exposure (28-day) inhalation study of 2-nitrophenol (Hazleton Laboratory, 1984) provide marginally adequate information in rats to derive a provisional subchronic RfC for 2-nitrophenol. This study identified significantly increased incidences of squamous metaplasia of the nasal epithelium in rats as the critical effect following 4 weeks of exposure to 2-nitrophenol vapors for 6 hours/day, 5 days/week. The lowest concentration of 2-nitrophenol associated with squamous metaplasia of the nasal epithelium was 30 mg/m³ in both male and female rats; the associated NOAEL was 5 mg/m³. Because the NOAEL and LOAEL represent essentially 0 and 100% response, respectively, it is not feasible to apply meaningful benchmark dose analysis to the data set. Therefore, the NOAEL of 5 mg/m³ was selected as the point of departure for deriving a subchronic RfC for 2-nitrophenol.

The NOAEL of 5 mg/m³ from intermittent exposure was adjusted to account for a continuous exposure scenario as follows:

$$\begin{aligned}\text{NOAEL}_{[\text{ADJ}]} &= \text{NOAEL} \times 6 \text{ hours}/24 \text{ hours} \times 5 \text{ days}/7 \text{ days} \\ \text{NOAEL}_{[\text{ADJ}]} &= 5 \text{ mg/m}^3 \times 6/24 \times 5/7 = 0.89 \text{ mg/m}^3\end{aligned}$$

According to U.S. EPA (1994b) methodology for respiratory effects of a category 1 gas (a systemic toxicant without significant portal of entry (lung) effects), such as 2-nitrophenol the NOAEL_[HEC] (human equivalent concentration) is calculated by multiplying the NOAEL_[ADJ] for upper respiratory effects by the regional gas dose ratio for extrathoracic effects (RGDR_{ET}). The default RGDR_{ET} is calculated according to the following equation:

$$\text{RGDR}_{\text{ET}} = \frac{\left[\frac{\dot{V}_E}{\text{SA}_{\text{ET}}} \right]_A}{\left[\frac{\dot{V}_E}{\text{SA}_{\text{ET}}} \right]_H} \quad (\text{Equation 4-18; U.S. EPA 1994b})$$

where:

\dot{V}_E = minute volume (cm³/minute)

SA_{ET} = surface area of the extrathoracic region (cm²), and

A, H = subscripts denoting laboratory animal and human, respectively.

Default surface area values for the extrathoracic respiratory region are 15 cm² for the rat and 200 cm² for the human (U.S. EPA (1994b). For the male Sprague-Dawley rat, a reference inhalation rate of 0.27 m³/day (270,000 cm³/day; U.S. EPA, 1988, standard default) produces a minute volume of 187.5 cm³/min (270,000 cm³/day ÷ 1440 min/day). The default minute volume for the human is 13,800 cm³/min (13.8 L/min or 20 m³/day; U.S. EPA, 1994b). Therefore:

$$RGDR_{PU} = \frac{\left[\frac{187.5}{15} \right]_A}{\left[\frac{13,800}{200} \right]_H} = 0.1812$$

The NOAEL_[HEC] is derived as follows:

$$\text{NOAEL}_{[\text{HEC}]} = \text{NOAEL}_{[\text{ADJ}]} \times \text{RGDR}_{\text{ET}} = 0.89 \text{ mg/m}^3 \times 0.1812 = 0.1613 \text{ mg/m}^3$$

The **subchronic p-RfC of 5E-4 mg/m³** based on squamous metaplasia of the nasal epithelium in rats (Hazleton Laboratories, 1984) is derived by dividing the NOAEL_[HEC] of 0.16 mg/m³ by a composite uncertainty factor (UF) of 300, which includes factors of 3 for interspecies extrapolation, 10 for interindividual human variability and 10 for data base deficiencies.

A 3-fold UF is used to account for uncertainty in extrapolating from laboratory animals to humans (i.e., interspecies variability). No information is available regarding the toxicity of 2-nitrophenol in humans. No comparative information is available regarding the toxicokinetics or toxicodynamics of 2-nitrophenol in animals and humans. However, the default dosimetric calculation for deriving an HEC accounts for the uncertainty in the variability in toxicokinetics of humans and rats. A 3-fold UF is applied to account for uncertainty in species differences for toxicodynamics (U.S. EPA, 1994b).

A 10-fold UF is used to account for variation in sensitivity among members of the human population (i.e., interindividual variability). This UF was not reduced due to the lack of human inhalation exposure data.

A 10-fold UF is used to account for uncertainty associated with data base deficiencies. A single 28-day inhalation toxicity study in one animal species (rat) is available (Hazleton Laboratories, 1984). The data base lacks studies of subchronic and chronic toxicity, inhalation neurotoxicity, developmental toxicity and reproductive toxicity (including 2-generation reproductive toxicity). Although the principal study (Hazleton Laboratories, 1984) was only a 28-day study (less than subchronic duration), the minor nature of the effects observed suggests that the 10-fold database UF is adequate to capture the uncertainties associated with use of the less-than-subchronic study in this instance.

Confidence in the principal study (Hazleton Laboratories, 1984) is low-to-medium. The study included comprehensive gross and histopathologic assessments. A major limitation of this study is the less-than-subchronic study duration of 28 days. Confidence in the data base is low because the data base lacks studies of subchronic and chronic toxicity, inhalation neurotoxicity, and developmental and reproductive toxicity (including 2-generation reproductive toxicity). Reflecting low-to-medium confidence in the principal study and low confidence in the data base, confidence in the provisional subchronic RfC is low.

Chronic p-RfC

The lack of adequate subchronic or chronic inhalation data for humans or animals precludes the derivation of a provisional chronic RfC for 2-nitrophenol. Use of the 28-day study (Hazleton Laboratories, 1984) was rejected because of uncertainties in exposure duration and toxicokinetics and dynamics in humans, and a lack of reproduction/developmental studies and which would result in five areas of uncertainties. According to the uncertainty in hematological effects which could become apparent in a chronic study, the database is insufficient to support derivation of chronic p-RfC (U.S. EPA, 1994b).

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2-NITROPHENOL

Weight-of-Evidence Descriptor

No information was located regarding the carcinogenicity of 2-nitrophenol in humans. No lifetime assessments were located regarding the carcinogenicity of inhaled or ingested 2-nitrophenol in animals. 2-Nitrophenol did not exhibit skin tumor-promoting action in mice receiving dermal applications twice weekly for 12 weeks (Boutwell and Bosch, 1959). Available genotoxicity assays of 2-nitrophenol indicate that the chemical is not genotoxic (Chiu et al., 1978; Dellarco and Prival, 1989; Foureman et al., 1994; Haworth et al., 1983; Kawai et al., 1987; Koerdel et al., 1981; Massey et al., 1994; Shimizu and Yano, 1986; Suzuki et al., 1983; Szybalski, 1958; Yamada et al., 1987). In accordance with U.S. EPA (2005) cancer guidelines, there is *inadequate information to assess carcinogenic potential* for 2-nitrophenol, based on the lack of human or animal carcinogenicity data.

Quantitative Estimates of Carcinogenic Risk

There are no human or animal data from which to derive an oral slope factor or inhalation unit risk for 2-nitrophenol.

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Provisional Peer Reviewed Toxicity Values for
Aluminum
(CASRN 7429-90-5)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
i.v.	intravenous
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR ALUMINUM (CASRN 7429-90-5)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

This document has passed the STSC quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

INTRODUCTION

Verified toxicity values for aluminum (Al) and its compounds are unavailable on IRIS or HEAST (U.S. EPA, 2006, 1997), except for a chronic oral RfD of 4E-4 mg/kg-day for aluminum phosphide. However, occupational guidelines and standards have been established for a number of chemical and physical forms of Al, including, from ACGIH, 8-hour TWA-TLVs of 10 mg/m³ for the compound as a metal dust or oxide, 5 mg/m³ as "pyro" powders or welding fumes, and 2 mg/m³ for soluble salts or organic forms of the metal (ACGIH, 1998). From NIOSH, 10-hour TWA-RELs of 10 mg/m³ are specified for "total" Al dust versus 5 mg/m³ for the respirable portion (NIOSH, 1994). NIOSH covers all other forms of the metal by identical values to those specified by ACGIH (ACGIH, 1998). OSHA PELs for Al include an 8-hour TWA value of 15 mg/m³ for "total" metal dust, versus 5 mg/m³ for the respirable portion (NIOSH, 1994). The U.S. EPA's CARA list (U.S. EPA, 1994) cites a HEA for Al (U.S. EPA, 1987), and ATSDR has updated its toxicological profile of the element (ATSDR, 1998).

The U.S. FDA (2000) has specified a maximum aluminum concentration of 25 mcg/L in large-volume parenterals (LVP) used in total parenteral nutrition (TPN). The FDA regulation applies to all LVPs used in TPN, including but not limited to parenteral amino acid solutions, highly concentrated dextrose solutions, parenteral lipid emulsions, sodium chloride and electrolyte solutions, and sterile water for injection.

Research papers pertinent to the potential toxicological and carcinogenic effects of Al were sought through computer searches of the HSDB, RTECS, MEDLINE and TOXLINE (and its subfiles) databases, covering the time period 1995-1999. The literature searches were conducted in June, 1999.

REVIEW OF PERTINENT DATA

The review by Stokinger (1981) gives an account of Al as an all-pervasive component of products that are central to the daily lives of most Americans. For example, the metal is a crucial part of manufactured products for the building, automobile and container industries, while Al as powder or flake is a component in a number of consumer products, such as paints, fireworks, etc. Al complexes and minerals are used in the brewing and paper industries, and as coagulants for water purification. Aluminum oxide finds application in abrasives, as a catalyst or absorbent, and as a component in fillers. Aluminum chloride is included in cosmetic formulations such as deodorants.

Human exposure to Al arises principally from food and water, through its widespread use in food additives, packaging and cooking utensils and Al-containing medications, particularly antacid, buffered aspirin, anti-ulcer and anti-diarrheal formulations (Marquis, 1989; Lione, 1985). Pennington and Schoen (1995) estimated daily Al intakes of 0.1-0.3 mg/kg-day for infants and children 6 months-6 years of age and 0.1-0.18 mg/kg-day for older children and adults, based on the FDA Total Diet Study (1993) and the U.S. Department of Agriculture Nationwide Food Consumption Survey (1987-1988). These data are in broad agreement with those of Wilhelm et al. (1995) who reported the dietary intake of Al in German children (living in the Duisberg area) as ranging from 0.008 to 0.11 mg Al/kg-day. In addition, these values are consistent with a range of 1-20 mg/day (0.014-0.3 mg/kg -day) for normal oral daily Al intake from food and water reported by other investigators (Ganrot, 1986; Iyengar et al., 1987; Wilhelm et al., 1990). However, users of Al-containing medications can ingest much larger amounts of the element, possibly as high as 840-5000 mg/day (12-71 mg/kg-day) from antacids, 126-728 mg/day (1.8-10.4 mg/kg-day) from buffered aspirins and 828 mg/day (11.8 mg/kg-day) from anti-ulcer compounds when taken at recommended dosages (Lione, 1985).

Toxicokinetics of Aluminum

There is a large amount of information available on the absorption, transfer from tissue to tissue and elimination of Al from the body, including data that have been amassed from studies on either human volunteers or laboratory animals. In general, the chemical appears to be poorly absorbed from the gastrointestinal tract, though the portion of the load that is retained will vary depending on the concentration, the chemical species administered, the fasting or fed state of the

host, gastrointestinal pH, animal model, etc. For example, Yokel and McNamara (1988) administered single oral doses of a number of Al compounds (both water soluble and insoluble) to New Zealand white rabbits and obtained absorbed proportions of the load ranging from 0.27% to 27%. Fractional uptake of Al in humans under normal conditions (i.e., with no intake of large quantities of Al from medicine) was estimated to be 0.1-0.3% assuming an intake of 20 mg Al/day (0.3 mg Al/kg-day) and urinary excretion of 20-50 μg Al/day (0.3-0.7 μg Al/kg-day) (Ganrot, 1986). However, little information is available on the actual mechanism by which the element and its compounds are transported across the brush border. (Wilhelm et al., 1990; Lione, 1985).

Although the overall extent of Al absorption is poor following oral exposure, there may be significant intake of the compound by those taking large amounts of Al compounds in patented remedies. As stated, absorption of Al is influenced by gastrointestinal conditions and content because Al can form various complexes with different solubilities and oxidation states depending on pH and interactions with dietary constituents. At low pH (3-5) in aqueous solutions, the soluble (ionic) forms of the Al prevail (Al^{3+}); at high pH (>8), Al in the form of soluble aluminum oxide is present; and at pH 5-8, the element is predominantly in the form of aluminum hydroxide, which is insoluble (van der Voet and de Wolff, 1986; Wilhelm et al., 1990). Ingested constituents that can influence absorption by forming complexes with Al include phosphate, fluoride, calcium, citrate and lactate. For example, Al is used to bind dietary phosphorus and decrease its absorption as a control for hyperphosphatemia, and citrate and lactate are complexing agents that can significantly increase Al absorption (Slanina et al., 1984, 1985, 1986; Partridge et al., 1989; Domingo et al., 1991; Ittel et al., 1991; Lione, 1985; Wilhelm et al., 1990).

A number of recent reports of studies on the gastrointestinal absorption of Al have examined the influence of organic anions such as citrate. In general, the presence of such components appears to enhance the absorption of Al, within narrow limits. For example, Deng et al. (1998) administered a single oral dose of either distilled water, 2 mmoles/L aluminum chloride or 2 mmoles/L aluminum chloride plus 2 mmoles/L sodium citrate to six male Wistar rats/group. Animals were bled at 1, 2 and 4 hours after dosing, then terminated after 6 hours. Inductively coupled plasma (ICP) was used to measure Al concentrations in blood, bone (tibia), kidney, liver and the intestinal wall. Irrespective of treatment, the appearance of Al in the blood of dosed groups peaked after 1 hour, with the concentration of the element at higher levels in those animals receiving citrate in addition to aluminum chloride. In those animals receiving aluminum chloride alone, significant tissue concentrations of the element were restricted to the gastrointestinal wall. Those receiving citrate displayed measurable quantities of the element in several of the other monitored tissues, including bone.

Sutherland and Greger (1998a) used a similar dosing regimen to examine the kinetics of absorption and elimination of Al in male Sprague-Dawley rats that had received a single oral dose of 0, 0.25, 0.5 or 1 mmoles/L/kg body weight aluminum lactate in 1 mL of 16% citrate. Concentrations of Al in serum, liver, kidney or bone (tibia) were measured at various post-dosing time intervals up to 6 hours. Depending on the dose, absorption factors for Al of up to 4.2% of the administered dose were observed, with the greater proportion retained in bone. The authors reported a slower rate of absorption in those animals receiving Al at the higher doses, an

observation potentially indicating reduced gut motility and/or saturation of the transcellular absorption processes at the higher concentrations. Aluminum deposited in kidney and bone appeared to turn-over at a slower rate than in the liver.

The influence of citrate on the gastrointestinal absorption of Al in man was examined directly by Taylor et al. (1998) who administered a drink containing Al and citrate to three volunteers. Aluminum and citrate concentrations were monitored in serial blood and urine samples for up to 24 hours. The kinetics of citrate and Al differed markedly, the former peaking in plasma after 32 minutes, versus 87 minutes for Al. This suggests that Al probably does not cross the gastrointestinal barrier as the citrate. Furthermore, the authors reported that the overall extent of Al absorption had probably not exceeded 1% in their experiment, a finding that contrasts with the higher values reported by Sutherland and Greger (1998a) in Sprague-Dawley rats and by Deng et al. (1998) in Wistar rats.

As discussed in a report by Glynn et al. (1999), gastrointestinal absorption of Al from aqueous media will be almost impossible to predict, because of the likelihood that the element will become absorbed to food particles in the intestinal lumen. Accordingly, depending on the dose, mode of delivery and caloric state of the experimental animal (fed/fasted), significant amounts of aqueous forms of Al will be absorbed only when available binding sites on food have become saturated. This presents an inherently complex overall picture of the element's absorption since, additionally, the normal dietary content of Al will be substantial. Thus, it may be assumed that some sequestered Al will be absorbed along with non-sequestered water soluble forms of the element, while the rest will be retained within the gastrointestinal tract.

Sutherland and Greger (1998b) used their aluminum lactate in 16% citrate dosing regimen to examine the comparative importance of biliary versus urinary excretion of Al. Five to seven male Sprague-Dawley rats/group who had previously received an implanted bile cannula were treated by gavage. Another similarly-treated cohort of five animals/group were housed in metabolic cages immediately after dosing to provide 0- to 3-hour and 3- to 6-hour urine specimens. At termination, all animals were sacrificed and exsanguinated, and tissue, bile and urine samples were measured by graphite furnace atomic absorption spectroscopy. Among the key findings to emerge from this study was the incremental appearance of Al in bile as early as 15 minutes after dosing. However, overall amounts of Al were greater in the 3-hour urine samples than those that had accumulated in bile samples collected within a similar time frame. The fact that control rats excreted 3 times more Al in bile than in urine during the first 3 hours after dosing led the authors to conclude that, at low exposure to Al (in controls receiving Al solely from food), the liver is capable of excreting the element to the bile, a mechanism that becomes saturated as the level of Al administration becomes increased. Thereafter, urinary excretion becomes the primary route of elimination in circumstances of Al overload.

Aluminum can also be absorbed by inhalation as indicated by age-related deposition in the lungs of the general population and exposure-related increased blood and urine concentrations in workers exposed to Al (Bast-Pettersen et al., 1994; Sjogren et al., 1996; Hosovski et al., 1990; Wilhelm et al., 1990; U.S. EPA, 1987). Aluminum occurs primarily in particulate form in the ambient atmosphere and as various dusts and fumes during its production and use. Common forms of inhaled Al include aluminum oxide (alumina; Al_2O_3), pyro powders

(powder and flake Al-treated to reduce surface oxidation), Al welding fume and soluble salts (e.g., aluminum chloride and sulfate) (ACGIH, 1998).

Neurotoxicity as a Primary Toxicological Effect of Aluminum

One of the greatest health concerns regarding Al is its neurological effects. The first evidence for Al-induced neurotoxicity in humans was seen in patients who, as a result of receiving long-term hemodialysis for chronic renal failure, developed a degenerative neurological syndrome (dialysis dementia) characterized by the gradual loss of motor, speech and cognitive functions (Alfrey, 1993). This dementia, attributable to Al in the dialysate, is usually fatal within 6-9 months after the first clinical signs appear. In addition, many patients received high oral doses of Al to act as phosphate binders. Autopsies of these patients revealed increased concentrations of Al in the gray matter and cerebral spinal fluid (CSF) but no evidence of neurofibrillary degeneration (NFD) despite the elevated Al levels. Once the connection between Al and dialysis dementia was established, Al was removed from dialysis fluid and the incidence of dementia rapidly declined, thereby strengthening the argument that Al was a causal agent in dialysis dementia (Ganrot, 1986).

Amyotrophic Lateral Sclerosis (ALS) and Parkinson's Disease (PD) are other neurological diseases which have been associated with Al exposure. ALS is a progressive disease of the Central Nervous System (CNS) that is characterized by an accumulation of neurofibrillary tangles. In Guam, southern West New Guinea and parts of Japan, there is an unusually high prevalence of ALS and PD. This may be related to the natural abundance of Al coupled with the virtual lack of magnesium and calcium in the drinking water supplies and soil of these areas. In a study designed to evaluate effects of high Al and low calcium levels in the diet, much like the conditions associated with Guam and other similar areas, cynomolgus monkeys were placed on a low calcium diet either with or without supplemental Al and manganese (Garruto et al., 1989). Chronic calcium deficiency alone produced neurodegenerative effects, although neurofibrillary changes were most frequently seen in the monkeys on a low calcium diet supplemented with Al and manganese.

Though a cause and effect relationship between Al and three forms of chronic encephalopathy in humans: senile dementia of the Alzheimer type (SDAT, Alzheimer's Disease), endemic Amyotrophic Lateral Sclerosis (ALS) and endemic Parkinsonism-dementia (PD, a mixture of Parkinsonism and senile dementia) has been suggested, there is no firm evidence that it plays a causal role in the development of these diseases (Ganrot, 1986; Lione, 1985). The condition is degenerative and characterized by the progressive loss of speech, motor and cognitive functions, with death typically occurring within 1-6 months. Autopsies of patients revealed increased concentrations of Al in the gray matter and cerebral spinal fluid (CSF), though with no conclusive evidence of NFD or other neuropathological changes despite the elevated Al levels.

The neurotoxicity of Al is well documented in certain animal species. Aluminum induces a spectrum of behavioral abnormalities and brain neurofibrillary degenerative changes in rabbits and cats when injected intracranially or parenterally in high doses, though hamsters and monkeys are less sensitive (Ganrot, 1986; Lione, 1985). Such studies have been designed as models for

the possible neurotoxicological effects of Al in humans. However, it should be noted that the neurofibrillary changes in affected animals differ in morphological detail from those associated with SDAT. As discussed further in the Oral Toxicity section, oral doses of Al can also induce neurobehavioral effects in adult mice and rats and in their developing offspring. In general, such neurotoxic effects of Al appear to be more subtle than those induced through routes of administration that by-pass the gastrointestinal tract, perhaps reflecting the lower doses of Al reaching the brain.

Recent reports of studies on the effects of Al on neurotoxicity in animals have sought to define the biochemical mechanisms that are impaired when Al crosses the blood-brain barrier. However, a unifying concept has yet to emerge, though the passage of the element into various regions of the brain has been clearly demonstrated (Deloncle et al., 1995). Among the many biochemical functions and processes that appear to be perturbed by the presence of Al in the brain are the peroxidation status of biological membranes (Katyal et al., 1997; Deloncle et al., 1999), inhibition of the neuronal glutamate-nitric oxide-cyclic GMP pathway (Cucarella et al., 1998), and the marked reduction of protein- and non-protein-bound thiols and the specific activity of Na^+/K^+ and Mg^{++} ATPases (Katyal et al., 1997). The relative importance of each of these mechanisms and how (or whether) they interact to bring about the observed physiological changes remains unclear.

Other Effects of Aluminum

Osteomalacia was frequently observed among long-term dialysis patients with neurological signs and is commonly attributed to Al overload (Ganrot, 1986; Lione, 1985). This bone condition is characterized by widened osteoid (unmineralized bone matrix) with no fibrosis, reduced mineralization rate, skeletal pain and a strong tendency for fractures, lack of response to vitamin D therapy and increased Al concentration in bone. Effects on bone histology and elevated bone Al levels have also been observed in patients with normal renal function who received total parenteral nutrition with Al-contaminated casein as a protein source, and in parenteral Al loading induced osteomalacia in rats and dogs (Lione, 1985).

There are a number of published reports of studies in which the carcinogenicity of aluminum compounds has been evaluated. These include oral exposure studies in which the compounds were made available to experimental animals in the drinking water or diet (Schroeder and Mitchener, 1975a,b; Oneda et al., 1994), and inhalation epidemiological studies, in which the incidence of tumor formation in persons exposed to aluminum-containing dusts and fumes in an occupational setting was compared to unexposed individuals (Spinelli et al., 1991; Thériault et al., 1984, 1990; Armstrong et al., 1986; Tremblay et al., 1995; Selden et al., 1997; Cullen et al., 1996; Dufresne et al., 1996; Ronneberg and Langmark, 1992). However, it has been generally concluded that the inferential association between exposure to Al and marginally increased incidences of tumors of the bladder and/or lung are confounded because of the co-exposure of subjects in such settings to other harmful and potentially carcinogenic substances, such as polycyclic aromatic hydrocarbons (PAHs and coal tar pitch volatiles (CTPV) (Ronneberg and Langmark, 1992). Therefore, the issue of the potential carcinogenicity of Al compounds remains uncertain.

Human Studies

Oral Exposure

Few reports have been identified that address the toxicological effects of Al in humans exposed orally. Furthermore, in a review, Reiber et al. (1995) pointed to the conflicting findings that have been reported when the incidence of neurological symptoms has been assessed in relation to Al exposure in either cross-sectional, ecological or case-control epidemiological studies. Among the more recent studies that have used this approach, Martyn et al. (1997) discussed the findings of a case-control study involving 441 men in England and Wales who were afflicted with either Alzheimer's disease, brain cancer, dementia or other neurological conditions. Assessing the historical exposure of these subjects failed to establish a link between Al in drinking water at the prevailing concentrations (below 0.2 mg/L) and the incidence of one or more of the conditions under investigation. No data were located regarding the oral carcinogenicity of aluminum compounds in humans.

Inhalation Exposure

Neurobehavioral effects were evaluated in a group of 87 Al foundry workers who were occupationally exposed to 4.6-11.5 mg/m³ Al fumes and dust for a mean of 12.0 years [standard deviation (SD) 4.5 years, shortest exposure 6 years] compared to an unexposed control group (n=60) who were matched for age, job seniority and social status to exposed subjects (Hosovski et al., 1990). It is reported that environmental Al concentrations were measured for each worker separately during the winter and summer, implying that personal sampling may have been used and that the contributing concentrations are time-weighted averages. In certain places, the number of particles ranged as high as 329-1020/cm² air, and dust particle sizes were ≤1, 1-5 and ≤5 microns in 65.6, 26.6 and 7.6% of the samples, respectively. Tests of psychomotor ability (simple and complex reaction time, oculomotor coordination), intellectual ability (Wechsler intelligence, performance intelligence and verbal intelligence quotients and Wechsler subtests on information processing, memory, understanding, calculation, coding, picture completion, picture grouping, object assembling, assembling of cubes and common concepts) and cerebral damage (Bender visual motor test) were conducted. Performance of the exposed workers was found to be significantly (p<0.02) impaired on the complex reaction time, oculomotor coordination, memory, coding, picture completion and object assembling tests. However, the investigators noted that the performance deficits had no clinical manifestations, and that additional studies were probably needed to confirm the possibility of cerebral damage. The study yielded a lowest available non-duration adjusted LOAEL of 4.6 mg Al/m³ for psychomotor and cognitive impairment during repeated 8-hour occupational exposures (Hosovski et al., 1990), that could be corrected for discontinuous exposure (10 m³/20 m³ and 5 days/7 days) to yield a LOAEL_{HEC} of 1.64 mg/m³ Al.

Aluminum oxide powders were administered to Canadian miners (mainly underground gold and uranium miners) in known exposures as a means of prophylaxis against silicosis (Stokinger, 1981; Rifat et al., 1990). Data in which more than 42 million Al treatments (≈150,000 man-years) had been given over a period of 27 years ending in 1971 were reviewed

by Stokinger (1981). The effectiveness of this treatment is uncertain but no lung damage or other ill effects (not specified) were observed. The powders (McIntyre powder) were prepared by grinding Al pellets so that 96% of the particles were $\leq 1.2 \mu\text{m}$ in diameter. During this process most of the particles became oxidized to aluminum oxide; the powder contained 85% aluminum oxide and 15% elemental Al. According to Stokinger (1981), recommended exposure concentrations were 30,000 particles of respirable size per cubic centimeter (ppcc) for 10 minutes/day or 10,000-20,000 ppcc for 20 minutes/day (total treatment days not indicated). Rifat et al. (1990) stated that the recommended exposure was to an Al dust concentration of 20,000-34,000 parts per ml air in the miners' changing rooms before each shift for 10 minutes. Stokinger (1981) reported that the 30,000 ppcc concentration corresponds to $\approx 350 \text{ mg/m}^3$, which is equivalent to an 8-hour average concentration of 2 mg/m^3 . Based on the Stokinger (1981) data and the fact that one unspecified study used levels 30 times higher than advised, the TLV of 10 mg/m^3 is recommended for Al dust (ACGIH, 1998).

The increasing awareness of the potential neurotoxicity of Al has resulted in a number of investigations of the incidence of neurotoxicological symptoms in Al workers. Although treatment with McIntyre powder had not produced apparent adverse effects, a neurobehavioral evaluation of male miners (261 exposed to McIntyre powder, 346 unexposed) who started working between 1940 and 1979 (additional duration data not reported) was performed in 1988-1989 (Rifat et al., 1990). There were no significant differences between exposed and unexposed miners in reported diagnoses of neurological disorder. Results of cognitive testing (Mini-Mental State Examination for general cognitive function, Ravens colored progressive matrices test for reasoning and Symbol Digit Modalities Test for spatial perceptual accuracy and information processing), however, showed that the exposed group had significantly ($p \leq 0.001$) impaired performance on at least one test, and when all test scores were summed. Also, the likelihood of scores in the impaired range increased with duration of exposure.

A neurologic syndrome was described in Al smelting plant potroom workers (White et al., 1992). Twenty-five men were evaluated for suspected work-related neurologic illness based on findings in three patients studied previously. The average duration of employment was 18.7 years (SD, 3.6; range, 12-23 years), 15 of the patients were working at the time of evaluation, and 10 had taken early retirement or medical leave due to workplace-related symptoms (mean length of time since exposure was 1.3 years ranging from 0.2-5 years). Quantitative exposure level data were not reported, but 21 of the workers had been employed in the potroom prior to installation of fume hoods for a mean duration of 5.3 years (range 3-7 years). Symptoms most often reported by the patients were frequent loss of balance (88%), memory loss (84%) and joint pain (84%); other symptoms included dizziness (80%), numbness (80%), parasthesias (72%) and tremor (68%). Neurologic examinations showed mild to moderate signs of lack of coordination (tremor, dyssynergy of upper extremity limb movement or ataxia) in 84% of the patients. Neuropsychologic effects were evaluated in 21 of the patients using the Wechsler Adult Intelligence Scale-Revised (intellectual functioning), Wide Range Achievement Test-Revised (academic functioning), Halstead-Reitan Neuropsychological Test Battery (neuropsychological assessment) and Minnesota Multiphasic Personality Inventory (personality functioning). Memory function was assessed with the Wechsler Memory Scale (14 patients) and Wechsler Memory Scale-Revised (8 patients). The memory function evaluation showed mild to moderate impairment on subtests of immediate recall for verbal or visual information (70-75% of the

tested patients) and delayed verbal or visual recall (50-70%). Other effects included mild or moderate impairment on Halstead-Reitan tests of abstract reasoning and flexible thinking (42% of the tested patients), memory for tactile information (53%) and sustained attention and discrimination of tonal and speech patterns (44 and 64%, respectively). On the Wechsler memory and Halstead-Reitan tests, mild and moderate impairment was defined as scores 1.5-2 and ≥ 2 standard deviations below the mean of the normal population, respectively. Most (89%) of the patients tested with the Minnesota Multiphasic Personality Inventory had abnormally elevated scores (≥ 2 SDs above the population mean) indicative of clinical depression. Significant positive correlations were found between severity of incoordination (signs and symptoms) and degree of exposure (qualitative) before the introduction of the ventilation hoods.

White et al. (1992) noted two other studies that described neurologic problems among Al smelter workers. Thus, an evaluation of 444 electrolysis workers found neuropsychiatric changes in 123 (28%), “neurotic syndromes” in 89 (20%) and “slight pyramidal and cerebellar changes” in 39 (9%) (Langauer-Lewowicka and Braszczyńska, 1983). In the second study, symptoms including mental confusion, concentration and memory problems were described in six potroom workers (Cawthon, 1988).

In another study of Al production workers, neuropsychological effects were assessed in 38 elderly men who had been exposed for at least 10 years exclusively in the potroom (n=14), foundry (n=8) or other manual labor departments of the same plant (n=16, control group) (Bast-Pettersen et al., 1994). The mean ages and employment durations of the groups were in the ranges of 62.5-63.5 and 19.2-19.6 years, respectively. The men were examined soon after or just before retirement in 1991. Limited environmental monitoring data indicates that the degree of Al exposure varied between the subgroups and over the years. Average annual total dust concentrations in the potroom were reduced significantly from 9.5 mg/m³ in 1977 to 3.0 mg/m³ in 1990. Aluminum levels were not specifically reported, but the average Al content in the total potroom dust was approximately 20% by weight; other constituents of the dust included fluoride and coal tar pitch components. Data from an Al uptake/excretion study of workers from the same plant indicated that the level of Al exposure was approximately 8 times higher in the potroom than in the foundry (0.48 and 0.06 mg/m³, respectively) (Drablos et al., 1992). Medical examinations (including lung function, standard laboratory tests and serum and urine Al concentrations) and a neuropsychological test battery were performed. The battery assessed six mental functions (neuropsychiatric symptoms, motoric/sensoric, reaction time, psychomotor speed/efficiency, memory/learning and intelligence) using a questionnaire and 15 different objective tests. Some subtle deficits were found in potroom workers that were not considered to be indicative of a significant neurological syndrome. The findings in potroom workers included a subclinical tremor as indicated by results of a static steadiness test [time scores on one of two test indices were significantly worse in comparison with the control group (84% slower, p=0.03)], and possible tendencies (i.e., test results that were about 1 SD below normal mean values but not statistically significant) for increased risk of impaired visuospatial organization (Block Design subtest of the Wechsler Adult Intelligence Scale) and psychomotor tempo (one Halstead Reitan Trail Making test). Although these findings were not considered to be indicative of a neurologic syndrome, it was suggested that they may be early signs of CNS impairment. Additionally, the finding of a subclinical tremor seems to be consistent with the tremor and other

signs of incoordination observed in 84% of the patients in the White et al. (1992) study summarized above.

Studies of Al welders are consistent with those of Al smelter workers in indicating that occupational exposure to Al can be neurotoxic. CNS function was evaluated in 17 welders who had an average of 15 years (range 5-27 years) experience, with the last 4 years exclusively with Al (Hanninen et al., 1994). Most of the welders had equipment that ventilated the welding masks but the respiratory protection was not always used. The assessment included measurements of serum and urinary Al, neuropsychological tests (simple reaction time, three tests for psychomotor speed, two tests for visual and spatial ability, four memory tests and two verbal ability tests), a symptom questionnaire and neurological interview, quantitative electroencephalography (QEEG) and P-300 event-related auditory-evoked responses. Serum and urine Al levels were 3.5 and 8.5 times higher, respectively, than an unexposed reference population. The welders performed normally on the neuropsychological tests, although correlation analysis of test scores and exposure parameters showed weak negative associations between the four memory tests and urinary Al level and a positive association between the variability (standard deviation) of visual reaction times and serum Al levels. Analysis of the QEEG data showed that serum Al levels were positively correlated with the amount of delta and theta activity in the brain frontal region and negatively correlated with the amount of alpha activity in the frontal region. Results of this study (disturbances of memory and attention, QEEG changes similar to those in patients with Al encephalopathy) were interpreted as consistent with known CNS effects of Al, but insufficient for establishing a definite relationship between Al exposure and effects.

In another study of Al welders, CNS evaluations were performed on 38 men who had at least 5 years exposure (mean 17.1 years) and a control group of 44 railway track welders exposed to metal fumes other than Al (mean 13.8 years) (Sjogren et al., 1996). Limited monitoring data indicated that the median exposure to welding fumes was 10 mg/m^3 and that the Al content was 40% of the total fumes. Symptom questionnaires, psychological tests (simple reaction time, finger tapping speed and endurance, digit span, vocabulary, tracking, symbol digit coding, cylinders, olfactory threshold and Luria-Nebraska motor scale), neurophysiological indices [electroencephalography, P-300 auditory-evoked responses, brain-stem auditory evoked responses and diadochokinesis (ability to perform rapidly alternating movements with one limb)] and blood and urine Al levels were assessed. The blood and urine Al concentrations were approximately 3 and 7 times higher in the Al welders than in the controls, but there were no clear correlations between duration of exposure to Al and concentration of Al in blood or urine. The Al welders reported more acute CNS symptoms (e.g., concentration difficulties) and had decreased motor function in five tests (finger tapping in non-dominant hand, two tasks from the Luria-Nebraska motor scale, pegboard peg movement with dominant hand, amplitude of diadochokinesis in dominant hand) when compared to the control group. Urinary Al concentration was significantly correlated with acute CNS symptoms, but not with any of the performance measures. To further study possible dose-effect relationships of Al exposure, the Al welders were combined with the control group and divided into three exposure categories according to urinary Al levels, using the 50th and 75th percentiles as category dividers. The group with the highest mean urinary Al level had significantly more acute CNS symptoms and significantly reduced performance on one of the motor function tests (a Luria-Nebraska motor

scale task) when compared to the group with the lowest Al level. In an earlier study of 65 welders with ≥ 10 years of exposure to Al fumes, the highest exposure category (based on exposure duration) was 2.8 times more likely than unexposed workers to have three or more neuropsychiatric symptoms (Sjogren et al., 1990).

A body of epidemiological evidence has pointed to an increased incidence of cancers of various kinds in workers employed in the aluminum production industry. However, as discussed in a review by Ronneberg and Langmark (1992), the concern about potential cancer hazards in the aluminum industry has primarily arisen because of exposures to polycyclic aromatic hydrocarbons (PAHs) and coal tar pitch volatiles (CTPVs) rather than to Al *per se*. Thus, while a number of studies have provided inferential data linking occupationally exposed aluminum workers with an increased risk of developing tumors of the bladder or lung (Gibbs, 1985; Thériault et al., 1984, 1990; Armstrong et al., 1986; Spinelli et al., 1991; Pearson et al., 1993; Tremblay et al., 1995), it would be unwise to ascribe any excess tumor formation to the effects of Al in view of the concurrent exposure to well-documented carcinogenic PAHs such as benzo(a)pyrene. The issue is further complicated by the likely exposure of production workers to other substances such as fluorides, sulfur dioxide, aromatic amines and asbestos (Ronneberg and Langmark, 1992; Tremblay et al., 1995; Dufresne et al., 1996), and to the possible effects of cigarette smoking in affected individuals. Consequently, these studies have failed to provide direct evidence for the carcinogenicity of Al fumes and dusts.

Animal Studies

Oral Exposure

Numerous subchronic animal studies were located in the biomedical/toxicological literature but only those that define the threshold region of the oral dose-response relationship are summarized in this paper. A major limitation of many of the studies of Al toxicity is the lack of complete information on total dietary (e.g., food and drinking water) intake of Al and of other elements that are known to effect Al biokinetics and toxicity (e.g., calcium and magnesium). Estimated or reported dosages used in studies in which Al content of the basal diets are not reported must be assumed to underestimate the actual experimental dosages. The magnitude of the underestimate may be considerable. For example, a range of Al contents of 200-1200 mg Al/kg for commercial grain-based diets (Golub et al., 1992b) would provide 30-200 mg Al/kg bw-day in a subchronic or chronic mouse bioassay [based on U.S. EPA (1988) default values for body weight and food intake]. On this basis, studies in which complete dietary Al intakes were not reported or could not be estimated may provide some information about the hazards of oral exposure to Al but are inappropriate for establishing NOAELs or LOAELs for the critical effect of Al. NOAELs and LOAELs from studies that provide estimates of total Al dosages, or otherwise provide information relevant to determining the NOAEL/LOAEL boundary for the critical effect of Al are presented in Table 1 and are summarized below.

Systemic toxicity

Groups of 10 female Sprague-Dawley rats were administered aluminum nitrate nonahydrate in sugar-containing drinking water at doses of 360, 720 and 3600 mg/kg-day (26, 52

and 259 mg Al/kg bw-day, respectively) for 100 days (Domingo et al., 1987). A control group received sugar-containing distilled water only. Sugar had been added to the drinking water of all groups to reduce the taste-aversive effects of Al. The level of Al in the diet was not reported. Animals were housed in metabolic cages to facilitate the collection of fecal and urine samples. Food and water consumption were measured daily, body weights were noted weekly and blood samples were taken at monthly intervals and at termination to monitor clinical chemistry and hematological parameters. At termination, all animals were necropsied, and the weights of major organs (brain, heart, lungs, kidneys, liver and spleen) were monitored. Aluminum concentrations were measured in various tissues, pieces of which were processed for histopathological examination. A significant decrease ($p < 0.05$) in body weight gain was observed in the 259 mg Al/kg-day group, attributed by the authors to decreased food intake. Overall, no consistent variations in hematological (hemoglobin, hematocrit) or clinical chemistry (SGOT, SGPT, alkaline phosphatase, urea, creatinine, total protein, cholesterol, glucose) parameters were observed. No histopathological alterations in the heart, liver, kidney, spleen, brain and cerebellum were observed. Interpretation of these data was complicated by the concurrent exposure of the rats to high doses of nitrate of up to 475 times the RfD for nitrate (1.6 mg nitrate-nitrogen/kg-day) which is based on methemoglobinemia in humans (U.S. EPA, 1999). Therefore, because of nitrate co-exposure, the absence from the study design of a food-restricted control group and uncertainty surrounding the contribution of Al in food, the apparent effect of Al on body weight gain cannot be conclusively attributed to Al alone.

Some recent studies have identified a number of potential toxicological responses in laboratory animals exposed orally to Al compounds in a subchronic or chronic dosing regimen. In most cases, however, only one dose level was employed in the study compared to controls, and since the amount of Al in the diet was not given, the resulting dose level represents an incremental dose of Al compared to that of controls as baseline. However, while these studies may offer inadequate quantitative dosimetric information for NOAEL/LOAEL identification and consequent RfD development, they provide a qualitative indication of a range of potential toxicological responses that might be induced in humans exposed to the element. For example, Garbossa et al. (1998) studied the potential for water-soluble Al to affect the erythropoietic integrity of late erythroid progenitor cells in the bone marrow. Three groups of five male Wistar rats/group were either (1) gavaged with citrate at a dose of 1.0 μmol Al/g-day (27 mg/kg-day), 5 days/week, for 15 weeks, (2) had drinking water containing 100 mmol Al/L made available to them as the citrate for the same length of time or (3) maintained as controls. As calculated by the authors, the dose associated with the applied concentration of Al in drinking water approximated to 14-17 μmol /g-day (420 mg/kg-day). Rats had access to a standard chow diet, though with no indication of the baseline concentration of Al provided therein. At the end of the in-life phase of the study, all rats were sacrificed, and samples of blood were obtained for hematological investigation. Femoral bone marrow cells were flushed with physiological medium, stimulated with recombinant human erythropoietin, then monitored for the comparative incidence of colony-forming units-erythroid (CFU-E). Further tests were carried out to monitor the osmotic fragility and average life-span of erythrocytes from each test group. The animals in the group receiving Al at the higher dose showed decreased hematocrit, hemoglobin concentration, median osmotic fragility and erythrocyte life-span values compared to controls. The content of Al increased in the serum and bone of both exposed groups, the distribution of concentrations in bone correlating inversely with the extent of an animal's CFU-E development.

That Al in drinking water may have the ability to cause histopathological changes and altered hepatic enzyme activities was suggested by Basu et al. (1997) who made available aluminum chloride in drinking water to groups of eight male Sprague-Dawley rats at a dose of 50 mg/kg-day (10.1 mg Al/kg-day) for 40 days. Additionally, other groups of similarly-treated rats received drinking water containing either 0, 50, 100, 200 or 400 ppm (mg/L) added calcium (Ca), as the chloride. The authors reported increased specific activities of acid and alkaline phosphatases in liver 10,000 x g supernatants from Al-receiving animals versus controls, and in alkaline phosphatase activity in equivalent kidney preparations. The presence of Ca in the drinking water appeared to reverse these changes, plus the accompanying histopathological features associated with them.

Konishi et al. (1996) examined the ability of Al and Ca to cause opposite and potentially harmful effects in laboratory animals, in relation to the well-documented association between Al and the onset of osteomalacia. Male STD Wistar rats were divided into four groups (n=4), receiving either (1) a normal diet (Group I), (2) a normal diet supplemented with Al (Group II), (3) a Ca-deficient diet (Group III) or (4) a Ca-deficient diet with supplemental Al (Group IV), for 10 weeks. Blood samples were taken at termination, and then animals were perfused with paraformaldehyde/glutaraldehyde fixative. Levels of Ca, iron (Fe) and Al in serum and bone were measured by atomic absorption spectrophotometry, and sections of the resected right tibia were prepared for histopathological examination after decalcification in 5% formic acid in 10% formalin.

There were statistically-significant changes in body weight gain when those of groups 3 and 4 were compared to animals from groups 1 and 2, the values for the latter groups remaining constant from about 4 weeks of dosing. In discussing their histopathological findings, the authors described no decrease in the thickness of cortical bone in Group II compared to control, while bone specimen from Groups III and IV showed “an increase in osteoid as well as osteoblasts and osteoclasts”, in addition to other disturbances of ossification. Such effects were considered to suggest bone fragility, with changes being more marked in Group IV compared to III. The amount of Al in the tibia of exposed rats was significantly greater in Group II than in Group I, whereas the average levels in Groups III and IV showed a further increase in Al deposition, most notably in group IV. There were also differences among the groups in the concentration of Fe in bone (tibia), and in the concentrations of Al, Ca, Fe and the levels of parathyroid hormone in blood. The authors concluded that Ca deficiency appeared to potentiate the deposition of orally administered Al in bone, and the attendant inhibition of ossification. Iron deposition was also thought to play a role in the osteogenic disturbance, where Ca is deficient.

A histopathological investigation indicated profound changes in the cerebrovascular and neuronal integrity when male Long-Evans rats (n=9) were exposed for 52 weeks to 0.5 ppm aluminum fluoride in drinking water (Varner et al., 1998). This corresponded to an Al dose of 0.019 mg/kg-day, based on a default drinking water consumption of 0.057 L/day, and a default body weight of 0.472 kg for male Long-Evans rats (U.S. EPA, 1988). Dual control groups received either NaF (fluoride controls) or double distilled deionized water. Tissue levels of Al were measured in brain, liver and kidney by the use of a direct current plasma technique.

Animals receiving aluminum fluoride showed poor survival compared to the other groups, with 6/9 having died by week 48. The tissue concentrations of Al were increased in the brain and kidney compared to both the control groups, with Al-fluorescence being used to demonstrate that Al deposition was mostly in the vasculature. Morphological and histopathological changes due to treatment were apparent in the liver, kidney and spleen. Some changes in neuronal integrity were also evident in the hippocampus and neocortex. Other cytological changes in the brain were associated with chromatid clumping, pyknosis and vacuolation.

A report by Somova et al. (1997) describes a study in which 10 male Wistar rats/group received either 0, 5 or 20 mg/kg-day aluminum chloride by gavage in water for 6 months. At termination, all animals were exsanguinated, then subjected to a necropsy in which excised pieces of liver, kidney and cardiac and skeletal muscle were taken for histopathological examination. Pieces of brain were examined by electron as well as light microscopy, and all tissues were monitored for Al concentration by atomic absorption spectrophotometry. As tabulated by the authors, Al in plasma and all of the listed tissues was dose-dependently increased to levels that were statistically significantly greater than controls. However, though described in qualitative terms and illustrated photographically, the Al-induced lesions did not receive a quantitative treatment in the report. Thus, while at least some of the low dose rats displayed NFD (neuro fibrillar degeneration) of the hippocampal region of the brain, insufficient data are provided in the report to apply this observation to the identification of a NOAEL or LOAEL.

Dietary experiments

Six Beagle dogs/sex/group were fed a diet providing either, in males, 0, 118, 317 or 1034 mg/kg-day sodium aluminum phosphate (0, 3.4, 9.0 or 29.4 mg Al/kg-day, respectively) or, in females, 0, 112, 361 or 1087 mg/kg-day sodium aluminum phosphate (0, 3.2, 10.3 or 30.9 mg Al/kg bw-day, respectively), for 6 months (Katz et al., 1984). No information was available on the level of Al in the diet, and no compound-related effects on body weight gain, hematological and clinical chemistry parameters (parameters not specified) or histopathological endpoints (major organs and tissues examined) were observed. A highest NOEL of 30.9 mg Al/kg-day could be tentatively identified in this study, but this would not include the contribution of Al from the basal diet, nor reflect the identification of any toxicological effects, since the NOEL occurred at the upper limit of the dose-response curve.

Neurotoxicity

A number of studies have been reported in which neurotoxicological/neurobehavioral effects have been explicitly evaluated. In others, the effects of Al on neurological developmental have been addressed. For example, Golub et al. (1989) fed diets containing Al as the lactate at 25 (controls), 500 or 1000 mg Al/kg diet (3.3, 65 or 130 mg Al/kg-day) to groups of 15 female Swiss-Webster mice for 6 weeks (Golub et al., 1989). No mice were exposed to lactate alone. While no statistically significant differences in food intake or body weight gain were observed, mice fed the highest Al concentration gained less weight than the controls or low-dose group. As reported by the authors, a significant decrease (20%) in spontaneous motor activity (i.e., total, vertical and horizontal movement) was observed in the 130 mg Al/kg-day group. Activity in the

65 mg Al/kg-day group was not significantly different than the controls. Thus, the highest NOAEL is 65 mg Al/kg-day and the LOAEL is 130 mg Al/kg-day.

Neurobehavioral effects of aluminum lactate were evaluated in groups of 12 female N:NIH Swiss-Webster mice (4.5-5.5 weeks old) that were fed 25 (controls) or 1000 mg Al/g diet for 90 days (Golub et al., 1992a). Based on a food factor of 0.19 kg diet/kg body weight/day calculated using an algorithm relating food consumption to body weight (U.S. EPA, 1988) and reported body weight data (the time-weighted average weight is 25.4 g), the dosage in the treated mice is estimated to be 190 mg Al/kg bw-day. No mice were exposed to lactate alone. A neurobehavioral test battery used by Donald et al. (1989) was administered at the beginning of the experiment (day 0) and after 45 and 90 (± 3) days, with motor activity evaluated at the latter two time points. Aluminum levels were measured in brain, femur and liver at the end of the exposure period.

Body weight was significantly increased in the treated mice but no exposure-related changes in food intake or overt signs of neurotoxicity were observed. Results of the neurobehavioral tests showed significantly decreased hindlimb grip strength at 90 days, decreased air puff startle response at 90 days and decreased auditory startle response at 45 days in the treated mice. Spontaneous motor activity was reduced at 90 days as indicated by decreased total activity counts, horizontal activity counts and percentage of intervals with high activity counts. Aluminum concentrations in the brain and liver were increased approximately 3-fold in the treated mice, but brain and liver lipid peroxidation indices were not altered.

Male Wistar rats (6-8 per group) were exposed continuously for 6 months to food containing 1.52 mg Al/kg (normal diet) or 1000 mg Al/kg as aluminum chloride with citrate (Florence et al., 1994). The average daily Al intake was estimated to be 0.13 or 84 mg Al/kg bw-day, assuming a body weight of 0.305 kg (arithmetic mean of default mature weight of male Wistar rats and the starting weight in this study of 0.11 kg) and a food intake of 0.026 kg food/kg bw-day, calculated using an algorithm relating food intake to body weight (U.S. EPA, 1988). The citrate content of the diet was in a 1:1 stoichiometric proportion to Al, therefore, the estimated daily intake was 598 mg/kg-day. Rats exposed to Al developed histopathological abnormalities in brain tissue, not specific to any brain region, characterized by extensive cytoplasmic vacuolization in astrocytes, swelling of astrocytic processes, particularly of astrocyte end-feet abutting blood vessels. Neurons also exhibited vacuolization and nuclear inclusions. Although no specific behavioral assays were reported, the investigators noted that "no significant behavioral changes were observed". Accordingly, the functional significance of the histopathological lesions is uncertain. The lesions appear to differ from the NFD observed with parenteral Al exposures (Kowall et al., 1989; Wakayama et al., 1993); or from exposures to Al in combination with calcium deprivation (Garruto et al., 1989; Kihira et al., 1995; Mitani, 1992). The LOAEL for histopathological changes in the brain was 84 mg Al/kg-day.

Male Sprague-Dawley rats (40 per group) were exposed in drinking water to 0, 50 or 100 mg Al/kg bw-day as aluminum nitrate with citric acid for 6.5 months beginning at 21 days of age, 8 months of age or 16 months of age (Domingo et al., 1996). The citric acid dosage was 355 or 710 mg/kg-day in the 50 or 100 mg Al/kg bw-day groups, respectively. Controls did not receive citric acid. Dietary Al intake was not reported; the rats were maintained on Panlab rat

chow. Animals from control and exposed groups were subjected to a number of neurobehavioral tests, and at termination, Al levels were measured in various excised regions of the brain. The authors observed the highest Al levels in the olfactory bulb and rhachidical bulb, while the cortex and thalamus were the regions showing the lowest Al content. However, compared to controls, there were no significant effects ($p > 0.05$) of Al (with citric acid) on spontaneous motor activity (open-field) or passive avoidance operant training or performance (grid floor shock, light/dark shuttle box). Thus, the NOAEL was 100 mg Al/kg-day with citric acid; although this does not include the Al contribution from food. This study is listed on Table 1 because the NOAEL, although probably underestimated because of unreported Al intake from food, is still lower than the LOAELs from other studies.

Groups of six male albino rats were administered 0 or 25 mg Al/kg bw-day as aluminum nitrate in normal saline by gavage, 10% ethanol in drinking water, or 25 mg Al/kg bw-day by gavage combined with 10% ethanol in drinking water, 6 days/week for 6 weeks (Flora et al., 1991). The level of Al in the diet was not reported. Urinary Δ -aminolevulinic acid (ALA), blood ALA-dehydratase (ALAD), blood zinc protoporphyrin (ZPP), glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) in serum and liver and brain biogenic amines and their metabolites [dopamine (DA), norepinephrine (NE), 5-hydroxytryptamine (5-HT), homovanillic acid (HVA) and 5-hydroxyindolacetic acid (5-HIAA)] were evaluated at the end of the treatment period. Treatment with Al alone caused significantly increased blood ALAD ($p < 0.01$), decreased liver GPT ($p < 0.05$), decreased brain DA ($p < 0.01$), increased brain NE ($p < 0.05$) and decreased brain 5-HT ($p < 0.05$). Compared to treatment with Al alone, concurrent exposure to ethanol and Al produced significantly decreased ALAD, increased ALA, increased ZPP, increased liver GPT, increased serum GOT and increased brain HVA. Significant changes found only in the combined Al and ethanol group included increased serum GPT, increased brain NE and decreased brain 5-HT. Treatment with ethanol alone only inhibited blood ALAD. The rats were co-exposed to relatively high levels of nitrate [comparable to those in the Domingo et al. (1987) subchronic study], but it seems likely that some of the changes (i.e., effects on brain chemicals) are related to aluminum which is known to be neurotoxic. Because the toxicological significance of the changes is unclear due to lack of evaluation of neurobehavioral performance and other endpoints, there is uncertainty whether the 25 mg Al/kg-day dose is a NOAEL or a LOAEL, an uncertainty compounded by the absence of information about the level of Al in the basal diet.

Reproductive/developmental toxicity

A number of studies have been carried out to examine the effects of Al compounds on developmental toxicity, particularly their effects on postnatal neurobehavioral development. For example, Bernuzzi et al. (1989) exposed groups of 6-12 pregnant Wistar rats to aluminum chloride or aluminum lactate in the diet on gestational days 1 through 21. The rats received nominal daily doses of 0, 100, 300, 400 mg Al/kg as aluminum chloride or 0, 100, 200 or 400 mg Al/kg as aluminum lactate. No rats were exposed to lactate alone, and information regarding level of Al in the basal diet was not reported. On the average, there was a less than 10% decrease in maternal body weight gain and no effect on food or water intake. No significant difference in litter size was observed. However, postnatal mortality increased 55% and 26% in offspring of the rats exposed to 300 or 400 mg Al/kg-day, respectively. The offspring of dams

fed ≥ 300 mg Al/kg-day weighed significantly less than controls on postnatal day 1. Decreased body weight was also observed on postnatal days (PD) 4 and 14 in the offspring of rats fed 400 mg Al/kg-day as aluminum lactate. The following tests were used to assess neuromotor development (maturation): righting reflex, grasping reflex, negative geotaxis, suspension test and locomotor coordination. The tests were performed on PDs 4, 6, 9, 12 and 20, respectively. Impairment of neuromotor development (righting and grasping reflexes) was observed in the pups exposed to ≥ 200 mg Al/kg-day. Impaired grasping reflex was also observed in the 100 mg/kg-day aluminum lactate group. Offspring of rats fed 400 mg/kg-day also exhibited altered performance on the locomotor coordination test.

A follow-up study by the same research group found that ingestion of 400 mg Al/kg bw-day as aluminum lactate had no effect on postnatal mortality, body weight and righting and grasping reflex tests (Muller et al., 1990), although significant differences between control and exposure groups were noted in locomotor coordination and operant conditioning tests. Significant differences between controls and exposed groups in the negative geotaxis test were limited to those pups of dams treated during the second and third weeks of gestation, a finding interpreted by the authors to indicate the possibility of long-term effects on the central nervous system of trans-placenta exposure to Al during a later organogenic phase. According to Muller et al. (1990), the contradictions between this and their earlier study (Bernuzzi et al., 1989) could be related to environmental modifications. In particular, the mothers and pups were much more protected in the Muller et al. (1990) study than in the previous one because they were housed in plastic cages instead of wire mesh cages and received cotton to build nests. Body temperature of the pups, therefore, may have been more adequately maintained in the Muller et al. (1990) study. As discussed in this study, toxicity in pups can be confounded by insufficient body temperature, and delayed pup weight gain could explain the differences in neuromotor performance.

Muller et al. (1990) administered diets supplemented with 0 or 400 mg Al/kg bw-day as aluminum lactate to groups of 6-9 pregnant Wistar rats on days 1-7, 1-14 or 1-21 of gestation. No rats were exposed to lactate alone, and information regarding level of Al in the basal diet was not reported. Neuromotor development was assessed on postnatal days 4, 6, 9, 12 and 20 using tests of righting reflex, grasping reflex, negative geotaxis, suspension and locomotor coordination, respectively. Learning ability was also tested on PD 65 using operant conditioning. No effects on maternal body weight or food intake were observed in dams exposed on gestational days 1-7 or 1-14. In the dams exposed on gestational days (GD) 1-21, a significant decrease in maternal body weight (26 and 35%, respectively) was observed on days 16 and 19 of gestation. Decreased food intake was also observed on day 19 of gestation. No effects on litter size, postnatal mortality or postnatal body weight were observed. Impairment of neuromotor development ($p < 0.05$) was observed in two of the five tests (negative geotaxis and locomotor coordination); no differences between the three treated groups were observed. For the operant conditioning test, there were significant differences ($p < 0.05$) between the treated and control young rats. No differences between the three treated groups were observed. The LOAEL for developmental toxicity is 400 mg Al/kg-day, but this does not include the contribution of Al from the basal diet.

Groups of 10 pregnant Sprague Dawley rats were administered 180, 360 or 720 mg/kg-day aluminum nitrate nonahydrate by gavage (13, 26, 52 mg Al/kg bw-day) on GDs 6-14

(Paternain et al., 1988). A vehicle (water) only control group was used. The level of Al in the diet was not reported. Aluminum exposed dams gained significantly less weight than the controls. No significant effects on the numbers of litters, corpora lutea, total implants, live fetuses, resorptions or runt fetuses were observed. Significant decreases in fetal body weight and tail length were observed at all three Al doses; decreased fetal body length was also observed at the 52 mg Al/kg-day dose level. No dose-related external or visceral malformations were observed in the offspring. However, a significant increase in the incidence of skeletal malformations (delayed ossification, hypoplastic deformed ribs) was observed at all three treatment levels. In addition, the incidence of hematomas was significantly increased at the high dose. Because the rats were co-exposed to relatively high levels of nitrate [comparable to those in the Domingo et al. (1987) subchronic study], the effects of treatment cannot be conclusively attributed to Al alone, in the absence of a nitrate-exposed control group.

By contrast to the striking findings of potentially teratogenic effects of aluminum nitrate in Sprague-Dawley rats, as described above (Paternain et al., 1988), equivalent experiments by Domingo et al. (1989) in Swiss mice did not reveal any reproductive, developmental or teratogenic effects of Al, when administered to dams as the hydroxide. Domingo et al. (1989) administered by gavage 0, 66.5, 133 or 266 mg/kg-day aluminum hydroxide (0, 23.9, 47.8 or 95.5 mg Al/kg bw-day) to groups of 20 pregnant Swiss mice on GD 6-15. The level of Al in the diet was not reported. The dams were killed on GD 18. No compound-related effects were observed on maternal mortality, clinical signs, body weight, food intake or absolute or relative heart, lung, spleen, liver, kidney and brain weights. In addition, no compound-related effects were observed on numbers of implantations, resorptions, live and dead fetuses, sex ratio and the incidences of external malformations, internal soft-tissue defects or skeletal abnormalities. Therefore, this study identifies a NOEL of 95.5 mg Al/kg-day by default for reproductive, developmental and teratogenic toxicity in mice. However, neuromotor development was not assessed and the contribution of Al from the basal diet was not stated in the report.

A number of studies have been designed to evaluate the influence of citrate or lactate on the potential developmental toxicity of Al. For example, Gomez et al. (1991) exposed groups of 15-19 pregnant Sprague-Dawley rats to either distilled water (controls) or 133 mg Al/kg bw-day in the form of either aluminum hydroxide (384 mg/kg-day), aluminum citrate (1064 mg/kg-day) or aluminum hydroxide (384 mg/kg-day) concurrent with citric acid (62 mg/kg-day) by gavage on GD 6-15. The level of Al in the diet was not reported and no rats were exposed to citric acid alone. Terminations were performed on GD 20. Maternal and fetal evaluations showed exposure-related effects only in the group exposed to aluminum hydroxide and citric acid concurrently. Significant changes included reduced maternal body weight gain on GDs 6-20 (but not at sacrifice on day 20), reduced fetal body weight and some skeletal variations (increased delayed occipital and sternbrae ossification and increased absence of xiphoides). No effects were seen on maternal food consumption or clinical signs, maternal absolute or relative liver, kidney or brain weights, gravid uterine weight, corpora lutea/dam, implantations/litter, pre- or postimplantation loss/litter, viable or nonviable implants/litter, fetal sex ratio or fetal malformations (external, visceral or skeletal). This study identified a stand alone minimum LOAEL of 133 mg Al/kg-day for non-neurobehavioral developmental toxicity of aluminum hydroxide and aluminum citrate in rats. Although confidence in this LOAEL is low (because aluminum hydroxide administered concurrently with citric acid induced did developmental

effects and because the dose does not include a contribution of Al from the basal diet) the value is consistent with the developmental NOAEL of 95.5 mg Al/kg-day for aluminum hydroxide in mice (Domingo et al., 1989).

In a similar experimental protocol, groups of 11-13 pregnant female Swiss albino (CD-1) mice were administered 57.5 mg Al/kg bw-day as either aluminum hydroxide (166 mg/kg-day), aluminum lactate (627 mg/kg-day) or aluminum hydroxide (166 mg/kg-day) concurrent with lactic acid (570 mg/kg-day) by gavage on gestation days 6-15 (Colomina et al., 1992). Other groups were treated with lactic acid alone (570 mg/kg-day, equivalent to the amount in 627 mg/kg of aluminum lactate) or distilled water (controls). The level of Al in the diet was not reported. Fetal evaluations were performed on GD 18, including examinations for skeletal and visceral abnormalities in approximately two-thirds and one-third of the pups, respectively. The investigators noted that the dose of Al (57.5 mg/kg-day) is equivalent to ingestion of 3.5 g Al/day by a 60 kg person, which is higher than the usual quantities of Al ingested therapeutically for peptic disorders. Maternal body weight gain was significantly lower than control values in the aluminum lactate-treated mice when evaluated over GDs 6-9 (92%), 6-12 (55.6%) and 0-18 (38.5%) and in the mice treated with combined aluminum hydroxide and lactic acid evaluated over GDs 6-12 (37.8%), 6-15 (42.7%) and 0-18 (15.7%). The decreased maternal weight gain in the aluminum lactate group was accompanied by significantly reduced food consumption during gestation days 6-18. Significant developmental and/or teratological effects in the aluminum lactate group included 16% reduced fetal body weight ($p < 0.01$) and increased incidences of cleft palate (13.2%, $p < 0.05$), dorsal hyperkyphosis (i.e., excessive flexion of spine) (13.5%, $p < 0.05$) and delayed parietal ossification (15.4%, $p < 0.01$). These developmental effects were not observed in any of the control or aluminum hydroxide exposed pups, and the only other significant changes in the other groups were decreased maternal relative liver weight and delayed fetal parietal ossification in the lactic acid only exposure group. Other types of internal or skeletal malformations or variations were not found in any of the fetuses. Additionally, no effects were seen on maternal absolute or relative kidney weight, gravid uterine weight, numbers of implantation sites/litter, live or dead fetuses, resorptions, postimplantation loss/litter, litters with dead fetuses or fetal sex ratio in any of the groups. By analogy to the findings of the Domingo et al. (1989) and Gomez et al. (1991) studies, the lack of developmental effects of aluminum hydroxide at the tested dose could be related to low solubility and absorption.

In a more recent study, pregnant Swiss mice were administered gavage doses of 0 or 104 mg Al/kg bw-day as aluminum hydroxide on days 6-15 of gestation (Colomina et al., 1994). Dietary Al intake was not reported; the mice were maintained on Panlab rodent chow. Compared to controls, there were no effects ($p > 0.05$) of Al on maternal body or organ weight, number of implantations per litter, number of resorptions per litter, number of dead fetuses per litter, percentage of positive post-implantation loss, sex ratio or fetal body weight per litter. Gross external, visceral or skeletal examination of fetuses revealed no abnormalities or developmental variations. Thus, the NOAEL for development effects from this study is 104 mg Al/kg-day, however, this does not include the Al contribution from food. Thus, based on this study and the previous study (Colomina et al., 1992), aluminum lactate appears to be more potent as a developmental toxicant in mice than the less water soluble aluminum hydroxide.

Groups of 16 pregnant Swiss-Webster mice were fed 25 (control group), 500 or 1000 mg Al/kg diet as aluminum lactate throughout gestation and lactation (Donald et al., 1989). The control diet was fed to pups that were selected for post-weaning neurobehavioral assessment. Reported maternal doses were 5, 100 and 200 mg Al/kg bw-day at the beginning of pregnancy and 10.5, 210 and 420 mg Al/kg bw-day near the end of lactation. No mice were exposed to lactate alone. There were no treatment-related changes in maternal survival, body weight (measured on GD 0 and 16 and PDs 0, 5, 10, 15 and 20), food intake, toxic signs or neurobehavior (evaluated after pups were weaned at PD 21 using the same test battery used for the pups and described below), or on litter size or postnatal growth and development in pups as assessed by body weight, toxic signs on PDs 0-55, and by crown-rump length on PDs 0 and 20. Neurobehavioral maturation was tested in two pups per litter on PDs 8-18 with a 12-item test battery (fore- and hindlimb grasp, fore- and hindpaw placement on sticks of 2 widths, vibrissa placing, visual placing, auditory and air puff startle, eye opening and screen grasp, cling and climb). A neurobehavioral test battery was administered to six pups per litter at age 25 days (4 days postweaning) or 39 days (fore- and hindlimb grip strengths, temperature sensitivity of tail, negative geotaxis, startle reflex to air puff and auditory stimuli) or age 21 and 35 days (foot splay). The pre-weaning neurobehavioral testing showed that a significant ($p=0.007$) number of pups in the high dose group had impaired vertical screen climb performance. The postweaning neurobehavioral assessment showed significantly ($p<0.05$) altered performance on several tests. These included decreased forelimb grip strength at age 39 days in the low dose group, increased hindlimb grip strength at age 25 days in both low and high dose groups, increased foot splay distance at age 21 days in both low and high dose groups and at age 35 days in the low dose group, and increased forelimb grip strength at age 25 days and decreased thermal sensitivity at age 25 and 39 days in the high dose group. There were no treatment-related changes in concentrations of Al in pup liver or bone (brain tissue was not analyzed).

In a more recent study of similar design by the same group of investigators, groups of 14 and 9 female Swiss Webster mice (6-8 weeks old) were fed 25 (control) or 1000 mg Al/g diet as aluminum lactate, respectively, during gestation and lactation (Golub et al., 1992b). The 1000 mg/g concentration was selected based on the demonstration of neurobehavioral effects in weanlings at this level (Donald et al., 1989). No mice were exposed to lactate alone. Using food intake and body weight values estimated from reported data, maternal doses are estimated to be approximately 4.3 and 174 mg Al/kg bw-day at the beginning of gestation and 4.8 and 607 at the end of the lactation period. At birth, litters were fostered either within or between groups to provide four groups of offspring that were exposed to excess Al via maternal diet during gestation, lactation, both or neither (i.e., 25 ppm during gestation and lactation, 1000 ppm during gestation and 25 ppm during lactation, 25 ppm during gestation and 1000 ppm during lactation, and 1000 ppm during gestation and lactation). Maternal effects included significantly ($p\leq 0.015$) reduced (10-12%) body weight gain and food intake in the treated group during late pregnancy and lactation, and signs of neurotoxicity (hindlimb splaying and dragging) in one treated dam at postnatal day 21 (weaning); this dam had seizures and died 4 days later. No treatment-related effects on litter size, birth weight, crown-rump length, righting ability at birth, sex ratio or postnatal survival were observed. Both gestation-only and lactation-only exposure caused significantly ($p<0.05$) decreased body weight gain in the treated pups beginning on postnatal day 10; combined gestation and lactation exposure produced the greatest decrease (approximately 24% at weaning). Neurobehavioral testing using the same battery as Donald et al. (1989) was

performed at weaning on the dams and on a total of 12, 16, 12 and 6 pups (1 male and 1 female pup per litter) from the control, gestation-only, lactation-only and combined gestation and lactation groups, respectively. Results of this testing showed effects only in pups, including significantly decreased forelimb grip strength after gestation-only exposure, increased hindlimb grip strength after both gestation and lactation exposure, decreased temperature sensitivity after lactation-only exposure, and longer negative geotaxis latency after lactation-only exposure. In general, the findings of this study are consistent with those of Donald et al. (1989) in showing neurodevelopmental effects at the 1000 mg/kg dietary concentration, although intake dosages are dissimilar at the end of lactation. Using the dosage at the beginning of gestation, this study defines a LOAEL of 174 mg/kg-day for developmental effects.

The Donald et al. (1989) study differs from that of Golub et al. (1992b) in that offspring were not fostered, were tested at a later age (25 vs. 21 days), were allowed 4 days of recovery from the treated diet prior to testing, participated in other behavioral tests currently, and experienced no growth retardation. The effects found only in the cross-fostered groups in the Golub et al. (1992b) study (lower forelimb strength after gestation exposure and altered negative geotaxis latencies after lactation only exposure) were not observed by Donald et al. (1989). Increased footsplay was observed by Donald et al. (1989) but not by Golub et al. (1992b), perhaps due to an opposing effect of smaller pup body size in this study. Neither gestation or lactation exposure affected pup brain or liver Al concentrations, but lactation exposure caused significantly lower manganese and iron concentrations in liver and manganese concentrations in brain.

In a further extension of the two previous studies (Donald et al., 1989; Golub et al., 1992b), pregnant female Swiss-Webster mice were exposed continuously to a semi-purified diet containing 7 (control), 500 or 1000 mg Al/kg from the time of conception, through pregnancy and lactation (Golub et al., 1995). At weaning, pups were exposed to the same Al diet as their mothers (500 or 1000 mg Al/kg) until they were 150-170 days of age or were switched to the control diet (7 mg Al/kg) for the same time period. Based on reported dosages in previous studies by the same investigators, estimated daily dosages for mice exposed to 1000 mg Al/kg diet were as follows: 200 mg/kg bw-day in pregnant mice, 420 mg/kg-day in lactating mice and 130 mg/kg-day in offspring (Golub et al., 1994); doses for the mice exposed to 500 mg Al/kg diet were assumed to be approximately half of that of mice fed 1000 mg Al/kg, or 100 mg/kg-day in pregnant mice, 210 mg/kg-day in lactating mice and 65 mg/kg-day in offspring. Compared to the control diet, the Al diet had no effect on dam weight, gestation length, litter size, pup weight, offspring growth or organ weights. Operant conditioning (nose poke) of offspring for delayed spatial alternation or discrimination reversal tasks was initiated at 50 days of age and continued 5 days/week for a total of 35 sessions. A neurobehavioral test battery was conducted when the offspring were 150-170 days of age (forelimb and hindlimb grip strength, temperature sensitivity, negative geotaxis, air puff and auditory startle response). Maternal and pre-weaning exposure to 500 mg Al/kg significantly affected ($p < 0.05$) operant training in the offspring, but not performance after training in delayed spatial alternation or discrimination reversal tasks (i.e., decreased number of training sessions to achieve the training criteria). This exposure also significantly decreased forelimb and hindlimb grip strength and puff startle response ($p < 0.05$). Pre-weaning and combined pre- and post-weaning exposure to 1000 mg Al/kg significantly increased ($p < 0.05$) incidence of cagemate aggression at the time behavioral

testing. No effects were observed on auditory startle response, temperature sensitivity or negative geotaxis in offspring. Histopathological examination of the brain and spinal cord revealed no treatment-related changes. Thus, the LOAEL for combined maternal and pre-weaning exposure on neurobehavioral effects in mice would approximate to 100 mg Al/kg-day (estimated daily maternal dosage).

Pregnant Charles River CD rats were administered gavage doses of 0, 250, 500 or 1000 mg Al/kg bw-day ("experiment A") or 0, 5, 25, 50, 250 or 500 mg Al/kg bw-day ("experiment B") as aluminum lactate in distilled water on GDs 5-15 (Agarwal et al., 1996). Dietary Al intake was not reported. Offspring were examined for body weight, anogenital distance, oestrus cycle regularity (after puberty), duration of pseudopregnancy induced by mechanical stimulation of the cervix, oocyte production induced by an injection of human chorionic gonadotropin, and male and female gonad weights. Aluminum had no effect on litter size and no consistent effects on birth weight were observed. For example, birth weights were decreased in male offspring from dams that received 250 mg Al/kg-day, but not at higher dosages, and the effect was observed only in experiment A. Female offspring birth weights decreased at certain dosage levels in experiment A and increased at these same dosage levels in experiment B. Similar inconsistencies between experiment A and B were observed for gonadal weights, anogenital distance, time to puberty (vaginal opening), duration of pseudopregnancy or numbers of superovulated oocytes. A significantly increased ($p < 0.05$) number of abnormal oestrus cycle lengths (defined as less than 4 days or greater than 5 days) occurred in offspring from dams that received 250 mg Al/kg-day (in experiment A, the endpoint was not measured in experiment B). However, the effect was most pronounced in the first three oestrus cycles (of five observed) and not detected by the 5th cycle. Thus, the NOAEL for temporary disturbance of the oestrus cycle in offspring of dams administered Al is 250 mg Al/kg-day. NOAELs for all other reproductive endpoints in this study were 1000 mg Al/kg-day. These NOAELs do not include the contribution of Al in food.

In a three-generation study, Ondreicka et al. (1966) exposed initial groups of seven female and three male Dobra Voda mice to either 0 or 19.3 mg Al/kg bw-day as aluminum chloride in drinking water. The diet also contained 160 to 180 ppm Al, giving an estimated intake of 27-31 mg/kg-day based on default values for food consumption and body weight for chronic exposure of mice (U.S. EPA, 1988). Using this estimate, the total Al intakes (drinking water and food) were 27 mg/kg-day (controls) and 46.3 mg/kg-day (exposed group). The P_0 group produced three litters (designated F_{1a} , F_{1b} and F_{1c}) and the F_{1a} group produced two litters (designated F_{2a} and F_{2b}) from which the weanlings were exposed to Al in the drinking water starting at 4 weeks of age. There was no difference in body weight gain among the groups in the P_0 generation, a result that contrasted with the striking decrease in this parameter in the treated F_{1b} , F_{1c} , F_{2a} and F_{2b} groups. Though no effects on erythrocyte count, hemoglobin levels or histopathology of the liver, spleen and kidneys were observed in the P_0 , F_1 or F_2 generations at the end of the study and no significant differences were seen in the number of litters or offspring between the exposed and control groups, the study identified a LOAEL of 46.3 mg Al/kg-day, based on the observed changes in body weight gain.

Other toxicological effects of aluminum

In a study designed to determine the effects of oral Al exposure on susceptibility to bacterial infection, female Swiss-Webster mice (13-14 per group) were exposed to a diet containing 25 (control), 500 or 1000 mg Al/kg as aluminum lactate during pregnancy, through lactation and for 10 days following weaning of the pups (Yoshida et al., 1989). Based on reported dosages in previous studies by the same investigators, estimated daily dosages for mice exposed to 1000 mg Al/kg diet are as follows: 200 mg/kg-day during pregnancy and 420 mg/kg-day during lactation; doses for the mice exposed to 500 mg Al/kg diet are assumed to be approximately half of that of mice fed 1000 mg Al/kg, or 100 mg/kg-day in pregnant mice and 210 mg/kg-day in lactating mice (Golub et al., 1994). At weaning, dams and pups were inoculated with a tail vein injection of *Listeria monocytogenes* and monitored for mortality for 10 days. In a separate experiment, female mice, 6 weeks of age, were exposed to the same dietary Al levels for 6 weeks and then inoculated with *L. monocytogenes*. Estimated Al dosages were 5, 98 or 195 mg Al/kg bw-day for the 25, 500 or 1000 mg Al/kg dietary levels, respectively, based on a default food factor of 0.195 kg diet/kg bw-day assuming a reference "subchronic" food intake and body weight for female B6C3F1 mice over the period from weaning to 90 days (U.S. EPA, 1988). Inoculation resulted in significantly greater ($p < 0.025$) mortality in dams exposed to 500 or 1000 mg Al/kg diet compared to controls. There were no differences in mortality between the groups of inoculated pups or between groups of inoculated adult mice exposed to Al for 6 weeks. The LOAEL for pregnant mice was 100 mg Al/kg bw-day and the NOAEL for adult, non-pregnant mice was 195 mg Al/kg bw-day. Although the exposure duration in this study was only 7 weeks, it is included in Table 1 because it provides the only dose-response data on the effects of Al on resistance to pathogens.

Carcinogenicity studies

Schroeder and Mitchener (1975a) exposed 52 Long-Evans rats/sex/group to 0 or 5 ppm Al as potassium aluminum sulfate in drinking water for life. Based on default values for drinking water consumption and body weight for this strain of rat in a chronic study (U.S. EPA, 1988), these values are equivalent to Al doses of 0.472 and 0.67 mg/kg-day, for males and females, respectively. Study endpoints included body and heart weight; serum glucose, cholesterol and uric acid; and urinary protein, glucose and pH. All animals were necropsied at the time of natural death, and histological examinations were carried out on heart, lung, kidney, liver, spleen and gross tumors, for approximately 50% of the animals in the group. The only remarkable finding was a significant increase ($p < 0.005$) in gross tumor incidence in exposed male rats [13/25 (52%) compared to 4/26 (15%) in controls], although the tumor sites were not reported. Six of the tumors in the exposed males (46% of total) were considered malignant compared to two malignant tumors (50% of total) in the male controls. There were no significant differences in tumor incidences between exposed and control females.

In another study by the same investigators, 54 Swiss mice/sex/group were exposed to drinking water containing 0 or 5 ppm Al as aluminum potassium sulfate for life (Schroeder and Mitchener, 1975b). Based on default values for drinking water consumption and body weight for B6C3F1 mice in a chronic study (U.S. EPA, 1988), these values approximate to Al doses of 1.2 mg/kg-day in both males and females. Study endpoints included body weight, gross pathology,

and some limited histology of the heart, lung, liver, kidney and spleen. The incidences of gross tumors were 15/41 (36.6%) and 11/38 (28.9%) in exposed and control males, respectively, and 19/41 (46.3%) and 14/47 (29.8%) in exposed and control females, respectively, differences that did not achieve statistical significance by Fisher's exact test, although incidences of multiple tumors and lymphoma leukemia were considered by the authors to be significantly increased in females ($p < 0.025$ and $p < 0.05$, respectively). However, a definitive assessment of aluminum carcinogenicity in both this and the rat study (Schroeder and Mitchener, 1975a) is precluded by the limitations of the pathology examinations and reporting.

In a more recent study, the tumorigenic potential of aluminum potassium sulfate was assessed in B6C3F1 mice chronically exposed in the diet (Oneda et al., 1994). Sixty animals/sex/group were fed a diet containing 0, 1.0, 2.5, 5.0 or 10.0% (w/w) for 20 months. These concentrations of aluminum potassium sulfate (as the dodecahydrate) are equivalent to 0, 569, 1422, 2844 and 5687 ppm Al. Using food factors calculated with an algorithm relating food consumption to body weight (U.S. EPA, 1988) and body weight data estimated from growth curves reported by the investigators, the dosages of aluminum are estimated to be 0, 95, 237, 483 or 1024 mg Al/kg-day in males and 0, 97, 242, 512 or 1110 mg Al/kg-day in females. Clinical signs, food consumption, and body weight were evaluated weekly. Hematology, clinical chemistry or urine endpoints were not assessed. Necropsies that included organ weight measurements and comprehensive histological examinations (including brain) were performed on all animals, including those that died during the course of the study. Survival rates were higher than control values in all treated male and female groups, ranging from 86.7-95.0% compared to 73.3% in males and 86.7-91.7% compared to 78.3% in females. No changes in food consumption were observed, but body weight gain was increased in both sexes at 95-97 and 237-242 mg Al/kg-day (weights were 10-23% higher than controls at end of study), was similar to controls in both sexes at 483-512 mg Al/kg-day, and decreased in both sexes at 1024-1110 mg Al/kg-day (11-16% lower than controls at end of study). There were no exposure-related increased incidences of tumors, other proliferative lesions or non-neoplastic lesions. In fact, the incidence of spontaneous hepatocellular carcinomas was significantly decreased in males at 1024 mg Al/kg-day (5.5% compared to 20.5% in controls, $p < 0.01$).

Inhalation Exposure

Groups of 20 weanling Fischer 344 rats/sex and 20 weanling Hartley guinea pigs/sex were exposed to 0, 0.25, 2.5 or 25 mg/m³ aluminum chlorhydrate [$\text{Al}_2(\text{OH})_5\text{Cl} \cdot x(\text{H}_2\text{O})$] for 6 hours/day, 5 days/week for 6 months (Steinhagen et al., 1978). Analysis of the aluminum chlorhydrate by the investigators showed it to contain 24.5% Al, indicating that the animals were exposed to 0, 0.061, 0.61 and 6.1 mg Al/m³. Body weights were measured weekly for the first 8 weeks and biweekly thereafter. At the end of the exposure period, 10 animals (5/sex) of each species were sacrificed for organ weight measurements (heart, lung, liver, kidney, spleen and brain) and histological examination of the lungs, liver and kidney. In addition, comprehensive histological examinations were performed on animals in the control and 6.1 mg AL/m³ groups. The remainder of the animals was used for hematology evaluation (RBC, WBC, hematocrit and hemoglobin) and Al measurements in blood and tissues. Apparent effects of Al included multifocal granulomatous pneumonia in both species at ≥ 0.61 mg Al/m³, significantly increased absolute and relative lung weights in both species, and decreased body weight gain in rats and

minimal lung edema in guinea pigs at 6.1 mg Al/m³. The granulomatous reaction was characterized by foci of giant vacuolated particle-containing macrophages in the lungs and macrophages that did not appear to contain vacuoles or other evidence of phagocytized material in the peribronchial lymph nodes. There was a significant dose-related accumulation of Al in the lungs of both species at ≥ 0.061 mg Al/m³. However, a NOAEL of 0.061 mg/m³ could be identified for the onset of compound-induced histopathological effects.

In other studies, groups of 14-30 guinea pigs, rats and hamsters were exposed to fine metallic Al powders (pyro, atomized and flaked) at concentrations of 15, 30, 50 or 100 mg powder/m³ air for 6 hours/day, 5 days/week for 6 months (Gross et al., 1973). Alveolar proteinosis occurred in exposed animals of all three species after 2 months of exposure, but fibrosis or other pulmonary changes did not develop. Similarly, groups of 23 or 46 rats and 48 hamsters were exposed to undetermined concentrations of Al fumes or Al powder (20% Al, 80% Al(OH)₃) for morning hours only or morning and afternoon for up to 20 months (Christie et al., 1963). Effects were similar for both forms of Al in both species, including initial increased alveolar macrophage proliferation followed by nodular hyalinized areas, with development of pneumonia but no fibrosis.

Exposure to 2.18 mg Al fibers/m³ for 6 hours/day, 5 days/week for up to 86 weeks produced slightly increased alveolar macrophages and some irritation of the nasal passages in a group of 50 Alderly Park rats (Pigott et al., 1981). Finally, a study by Drew et al. (1974) observed the development of granulomatous nodules also developed in male hamsters that were exposed to 8 mg Al/m³ of *Alchlor* (a propylene glycol complex of aluminum-chloride-hydroxide) for 6 hours/day, 5 days/week for 20 or 30 exposures. The alterations persisted at the longest post treatment observation (6 weeks) and consistently developed at the bifurcation of the bronchioloalveolar ducts, which is a likely site of particulate deposition.

DERIVATION OF A PROVISIONAL CHRONIC RfD FOR ALUMINUM

This survey of the toxicological effects of Al in rodents suggests that neurotoxicological and developmental (including neurodevelopmental) endpoints are among the most sensitive indicators of Al toxicity. However, as vehicles for the development of toxicity values such as a provisional chronic RfD, the latter group of studies are considered to be more appropriate, since the level of exposure to Al appears to be better characterized. In fact, neurobehavioral deficits have been observed in mice and rats exposed during various stages of development and in subchronic studies (Bernuzzi et al., 1989; Donald et al., 1989; Golub et al., 1989, 1992a, b, 1995; Muller et al., 1990), as described above. These deficits include impaired operant learning, changes in grip strength, altered startle response and impaired motor coordination. In addition, several studies have shown that oral Al can produce histopathological changes in the CNS, although the histopathological lesions have yet to be causally related to the neurobehavioral deficits. Thus, Florence et al. (1994) reported histopathological changes in the brain of rats exposed to dietary Al for 6 months, the changes including the appearance of vacuolation of the cell body and cell processes of astrocytes in the brain and swelling of astrocytic processes. In addition, more localized vacuolization of neurons in the brain also was observed. These changes

were observed in rats exposed to elevated Al in the diet and are distinct from the NFD that has been observed in rats, rabbits and monkeys maintained on elevated dietary Al in combination with reduced dietary calcium (Garruto et al., 1989; Kihira et al., 1994; Mitani, 1992; Yano et al., 1989; Yoshida et al., 1990) or in rabbits administered intracisternal or intraventricular injections of Al (Kowall et al., 1989; Wakayama et al., 1993). Interpretation of the low-calcium studies is complicated by the observation that NFD was observed in animals maintained on low-calcium diets without excess Al and was enhanced by the addition of excess Al to these diets (Garruto et al., 1989; Kihira et al., 1994). Furthermore, Al has been shown to inhibit the gastrointestinal absorption of calcium (Orihuela et al., 1996), an effect that may exacerbate the calcium deprivation induced by low calcium diets. Thus, it is not clear whether calcium deprivation enhances the neurotoxicity of Al or Al exacerbates the adverse effects of calcium deprivation.

Donald et al. (1989) and Golub et al. (1995) are co-principal studies that identify a LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice exposed to dietary aluminum lactate (soluble aluminum) during gestation and lactation. The neurotoxicity associated with this LOAEL is consistent with LOAELs from other developmental and subchronic neurobehavioral studies in mice and rats which used higher dietary dosages of aluminum lactate or aluminum chloride (Golub et al., 1989, 1992a,b; Bernuzzi et al., 1989; Muller et al., 1990). Of the above, Golub et al., (1995) is the only study in which a histopathological examination of the brain and spinal cord was conducted and no abnormalities were reported. The Florence et al. (1994) study indicates that histopathological abnormalities of the CNS can occur in rats exposed subchronically to 84 mg/kg-day; although this is lower than the LOAEL for neurobehavioral effects, it was not chosen as the principal study because the functional significance of the histopathological lesions are uncertain.

A number of studies were identified that, at face value, appeared to indicate LOAELs at lower doses than the 100 mg Al/kg-day value selected herein, for example, Paternain et al. (1988) and Colomina et al. (1992). However, in these as in many of the studies under consideration, insufficient information on dietary Al (Al content and/or feed type) was reported to permit a reliable estimation of the overall dose level to which the animals were subjected.

Other developmental studies with aluminum hydroxide and/or citrate in mice and rats identified a NOAEL which are equivalent (95.5 mg Al/kg-day), or a minimum LOAEL that was greater (133 mg Al/kg-day) than the 100 mg Al/kg-day critical LOAEL (Domingo et al., 1989; Gomez et al., 1991), an overlap potentially related to differences in effective doses due to variations in unreported Al dietary content and factors affecting absorption such as chemical form (e.g., the use of less absorbable aluminum hydroxide). In addition, the LOAEL of 43.3 mg Al/kg-day for decreased body weight gain in mice exposed to aluminum chloride for 180-390 days (Ondreicka et al., 1966) was thought be inappropriate for risk assessment due to the small sample size and to the poor reporting of study details. Aluminum nitrate caused alterations in levels of brain biogenic amines and hepatic and hematological indices in rats exposed to 21.4 mg Al/kg-day for 6 weeks (Flora et al., 1991). This dose is not a LOAEL because insufficient information is available to determine if the effects are adverse.

Therefore, the LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice (Donald et al., 1989, Golub et al., 1995) is selected as the basis for the provisional chronic

RfD. The LOAEL is considered minimal because the results of the postweaning neurobehavioral test battery indicate that performance deficits may be marginal. In particular, of the three observed effects (decreased forelimb and increased hindlimb grip strengths, increased hindlimb foot splay distance), one effect (increased grip strength) has unclear toxicological significance and two effects (increased grip strength and foot splay distance) did not persist after 2 weeks of no further exposure.

Application of an uncertainty factor (UF) of 100 (3 for use of a minimal LOAEL, 10 for interspecies extrapolation and 3 for intrahuman variability where the critical effects have been observed in a sensitive sub-group) results in a provisional RfD of

$$\text{p-RfD} = 1\text{E-0 mg Al/kg-day.}$$

The provisional RfD of **1E-0 mg Al/kg-day** is approximately 3-fold higher than estimated normal daily Al intake of approximately 0.2-0.3 mg/kg-day (Iyengar et al., 1987; Ganrot, 1986; Wilhelm et al., 1990). Chronic users of medications such as antacids, buffered aspirins and antiulceratives would be expected to ingest much larger amounts of Al, possibly as high as 10-70 mg/kg-day. However, these subjects would not represent the most sensitive population (developing infants), as indicated by the animal data.

Low confidence is placed in the co-critical studies, because they only identify a LOAEL for a sensitive effect and evaluated comparatively small numbers of animals. Confidence in the data base is low because the most reliable supporting data for neurotoxicity of Al in humans are of limited general relevance (e.g., dialysis encephalopathy is manifested in patients with impaired renal function and excessive Al uptake from intravenous exposure). In fact, neurotoxicity remains to be assessed in animals chronically exposed to Al, and developmental morphology has not been adequately investigated in two animal species. These limitations in the Al data base do not increase uncertainty in the RfD; therefore, a data base uncertainty factor was not used. However, reflecting the low confidence in the co-critical studies, there is low overall confidence in the RfD.

DERIVATION OF A PROVISIONAL CHRONIC RfC FOR ALUMINUM

Al seems to be the most likely cause for the generally and consistently reported psychomotor and cognitive effects (particularly signs of impaired coordination) in Al production workers and welders (Bast-Pettersen et al., 1994; Rifat et al., 1990; Hosovski et al., 1990; White et al., 1992; Hanninen et al., 1994; Sjogren et al., 1990, 1996). In addition, there is strong evidence that Al is neurotoxic by other routes of exposure. Thus, a degenerative neurological syndrome (dialysis dementia) has been documented in humans with chronic renal failure, apparently due to an increased exposure to Al from dialysis treatment and/or ingestion of phosphate binding agents which contain Al (Alfrey, 1993). This syndrome is characterized by gradual loss of motor, speech and cognitive functions. Neurotoxicity, particularly neuromuscular effects such as decreased motor activity, startle responsiveness and grip strength, has also been observed in mice following subchronic oral exposure and in the offspring of mice and rats exposed orally during gestation and/or lactation. Based on this information, as well as evidence

that Al is absorbed by Al production workers and welders, the hypothesis that the occupational studies are indicative of a neurotoxic effect of Al appears to be justified. However, the only occupational study that has yielded suitable monitoring data is that of Hosovski et al. (1990), in which workers were exposed to presumed time-weighted average (TWA) concentrations of 4.6-11.5 mg Al/m³ magnitude for an average of 12 years. Using 4.6 mg Al/m³ as the LOAEL for psychomotor and cognitive impairment for an 8-hour occupational exposure (Hosovski et al., 1990) and corrections for discontinuous exposure (10 m³/20 m³ and 5 days/7 days), the LOAEL_{HEC} is 1.64 mg/m³. Applying an uncertainty factor of 300 for intrahuman variability (10), use of a LOAEL (10) and an incomplete database (3) yields a provisional RfC of

$$\text{p-RfC} = 1.64 \text{ mg/m}^3 / 300 = 5\text{E-}3 \text{ mg/m}^3.$$

The lack of inhalation developmental studies may increase uncertainty in the database because oral data in animals indicate that neurotoxic and morphological developmental effects may occur at lower doses than neurotoxicity in adults. Additionally, there is uncertainty related to the lack of corroborating data on air concentrations associated with neurotoxicity. Confidence in the critical study is low to medium because only a LOAEL was identified. Confidence in the database is medium because (1) there are no corroborating data on effect levels (NOAELs and additional LOAELs), (2) no data are available for developmental neurotoxicity by the inhalation route and (3) a well-designed two-generation reproduction study is lacking. Reflecting the low to medium confidence in the critical study and database, there is low to medium confidence in the provisional RfC.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR ALUMINUM

Weight-Of-Evidence Classification

A considerable number of epidemiological studies have examined the incidence of excess tumor formation in persons occupationally exposed to Al in the form of dusts or fumes. In general, a body of inferential evidence exists for an increase in cancer of the bladder and lung through such occupational exposure to Al, although conclusions linking these responses to the effects of Al are confounded by attendant co-exposure to other harmful emissions such as PAHs and by cigarette smoking. A 20-month exposure of B6C3F1 mice to Al potassium sulfate dodecahydrate in the diet at concentrations up to 10% w/w displayed no indication of compound-related carcinogenicity and, in general, no indication of adverse toxicological effects of any kind (Oneda et al., 1994). Similarly, the life-time exposure of Swiss mice and Long-Evans rats to 5 ppm Al as aluminum potassium sulfate in drinking water provided no convincing evidence for the carcinogenicity of Al compounds (Schroeder and Mitchener, 1975a,b). Gene reversion experiments on Al compounds resulted in negative results in *S. typhimurium* (Ahn and Jeffrey, 1994). Taking all of the evidence of Al carcinogenicity together, and in accordance with the U.S. EPA (2005) cancer guidelines, aluminum is classified as *inadequate information to assess carcinogenic potential*. The basis for this classification is insufficient evidence in epidemiological/occupational studies, lack of demonstrated carcinogenicity or mutagenicity in

available animal studies, lack of positive evidence of non-carcinogenicity and lack of mode of action data for aluminum.

Quantitative Estimates of Carcinogenic Risk

Due to insufficient data, a provisional oral slope factor and inhalation unit risk could not be developed.

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Table 1. Summary of oral toxicity data for aluminum^a

Study	Type	Species	Al	Exposure Concentration (ppm)	Exposure Dosage (mg Al/kg-day)	Exposure Frequency and Duration	Critical Effect	NOAEL (mg Al/kg-day)	LOAEL (mg Al/kg-day)	FEL (mg Al/kg-day)
Ondreicka et al., 1966	Subchronic 3-gen dietary	Dobra Voda mice	chloride	--	27 (control), 46	Continuous, 180-390 days	Decreased body weight gain in F1 and F2.	--	46	--
Golub et al., 1989	Subchronic dietary	S-W mice	lactate	25 (control), 500,1000	3.3 (control), 65,130	Continuous, 6 weeks	Decreased spontaneous motor activity; decreased weight gain.	65	130	--
Golub et al., 1992a	Subchronic dietary	S-W mice	lactate	25 (control), 1000	190	Continuous, 90 days	Decreased hindlimb grip, decreased spontaneous motor activity, decreased startle response.	--	190	--
Florence et al., 1994	Subchronic dietary	Wistar rat	chloride (with citric acid)	1.52 (control), 1000	0.13 (control), 84	Continuous, 6 months	Histopathological changes in brain astrocytes and neurons.	--	84	--
Domingo et al., 1996	Subchronic drinking water	Sprague Dawley rats	nitrate (with citric acid)	--	0, 50, 100 (plus unreported dietary Al)	Continuous, 6.5 months	Operant conditioning and performance	100	--	--
Yoshida et al., 1989	Subchronic dietary	S-W mice	lactate	25 (control), 500, 1000	5 (control), 98, 195	Continuous, 7 weeks	Increased mortality from <i>L. monocytogenes</i> inoculation	195	--	--
Donald et al., 1989	Developmental dietary	S-W mice	lactate	25 (control), 500, 1000	5 (control), 100, 200	Continuous, gestation and lactation	Neurobehavioral effects.	--	100	--
Golub et al., 1992b	Developmental dietary	S-W mice	lactate	25 (control), 1000	4 (control), 174	Continuous, gestation and lactation	Neurobehavioral effects.	--	174	--
Golub et al., 1995	Developmental dietary	S-W mice	lactate	7, 500, 1000	1 (control), 100, 200	Continuous, gestation, lactation to maturity	Neurobehavioral effects.	--	100	--

Table 1. Summary of oral toxicity data for aluminum^a

Study	Type	Species	Al	Exposure Concentration (ppm)	Exposure Dosage (mg Al/kg-day)	Exposure Frequency and Duration	Critical Effect	NOAEL (mg Al/kg-day)	LOAEL (mg Al/kg-day)	FEL (mg Al/kg-day)
Yoshida et al., 1989	Developmental dietary	S-W mice	lactate	25 (control), 500, 1000	4 (control), 100, 200	Continuous, gestation and lactation	Increased mortality of dams from <i>L. monocytogenes</i> inoculation	--	100	--

^aStudies for which total dosages were reported or could be estimated (unless otherwise noted).



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March 24, 2008

Chloe Metz, Region 2

Onondaga Lake

ASSISTANCE REQUESTED: Toxicity values for approximately 50 chemicals

ENCLOSED INFORMATION: Attachment 1: PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR DIBENZOFURAN (CASRN 132-64-9)

Attachment 2: PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR ENDOSULFAN SULFATE (CASRN 1031-07-8)
Derivation of a Carcinogenicity Assessment

Attachment 3: PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR ENDOSULFAN SULFATE (CASRN 1031-07-8)
Derivation of a Chronic Oral RfD

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (3)

cc: STSC files

DRAFT - Do not cite or quote. For internal use only.

6-11-2007

Provisional Peer Reviewed Toxicity Values for

Dibenzofuran
(CASRN 132-64-9)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor

p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR DIBENZOFURAN (CASRN 132-64-9)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

This document has passed the STSC quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

INTRODUCTION

RfD and RfC values for dibenzofuran (DBF) were not available on IRIS (U.S. EPA, 2007) or in the HEAST (U.S. EPA, 1997). There is a Class D cancer assessment on IRIS (U.S. EPA, 2007). Dibenzofuran was included in a Drinking Water Toxicity Profile from 1992 (U.S. EPA, 1992), although no oral toxicity value was listed. The Office of Water did not include dibenzofuran on the latest Drinking Water Regulations (U.S. EPA, 2006a) or the Drinking Water Contaminant Candidate List (U.S. EPA, 2006b). The CARA list (U.S. EPA, 1991, 1994)

included a Health Effects Assessment (HEA) (U.S. EPA, 1987) and a Reportable Quantity Document (U.S. EPA, 1989) for Dibenzofuran. The HEA concluded that additional toxicity testing was necessary and did not derive a toxicity value due to the lack of data (U.S. EPA, 1987). The 1987 HEA for Dibenzofuran neither identified nor included discussion of Thomas et al. (1940), the primary source of data used in this PPRTV document. By contrast, the 1989 Reportable Quantity Document for Dibenzofuran (U.S. EPA, 1989) used Thomas et al. (1940) as the basis for derivation of composite scores and the corresponding reportable quantities for dibenzofuran.

ATSDR had not published a Toxicological Profile for dibenzofuran (ATSDR, 2006). NTP did not study the toxicity of dibenzofuran (NTP, 2006). WHO (2006) provided no relevant information. Available data on carcinogenicity, mutagenicity, metabolism, and other biological effects were summarized for dibenzofuran by the National Cancer Institute (NCI, 2000). Data on the adverse health effects of various halogenated dibenzofurans were available; however, the biological activity varies greatly among these congeners. U.S. EPA (1986a) did not recommend risk assessment by analogy to any of these more widely studied chemicals. NCI (2000) reported that the most structurally related chemical was dibenzo-p-dioxin. NCI (1979) reported that no excess tumors were induced in rats or mice fed dibenzo-p-dioxin up to 10,000 ppm in the diet.

Updated literature searches for noncancer and cancer data were conducted for data available through April 2006. The databases searched included: TOXLINE, MEDLINE, CANCERLIT, CCRIS, TSCATS, HSDB, RTECS, GENETOX, DART/ETICBACK, and EMIC/EMICBACK. Inhalation RfC values were not derived for dibenzofuran, because no human or animal inhalation data were found and the marginal ingestion data seemed inadequate to consider for inter-route extrapolation. However, a subchronic oral p-RfD value was derived, based on a LOAEL point of departure (POD) in Thomas et al. (1940). Chronic toxicity of dibenzofuran is discussed in the appendix. No data were identified from which to derive cancer risk values.

REVIEW OF PERTINENT DATA

Human Studies

Two cross-sectional studies of exposed workers were identified in the OPPT TSCATS database (Koppers 1980a,b). However, these studies reported exposures to dibenzofuran only in complex mixtures of coal tar products. Neither report noted adverse health effects that could be attributed to dibenzofuran exposure. Existing review documents and a detailed literature search identified no other data regarding the toxicity of dibenzofuran in humans.

Animal Studies

The only long-term toxicity data available for dibenzofuran were from a 200-day rat feeding study reported by Thomas et al. (1940). However, this document also will address the NCI (1979) data for dibenzo-p-dioxin, which NCI (2000) considered to be the chemical most structurally related to dibenzofuran.

NCI (1979) reported that unsubstituted dibenzo-p-dioxin, a structural analog of dibenzofuran, exhibited very low toxicity and no evidence of carcinogenicity in Osborne-Mendel rats and B6C3F1 mice, even when the maximum tolerated dose was approached (10,000 ppm in diet). Groups of 35 rats of each gender ingested dibenzo-p-dioxin at 5000 or 10,000 ppm in diet for 110 weeks. Groups of 50 mice of each gender ingested the same doses for 87 or 90 weeks. Controls consisted of groups of 35 untreated rats of each gender and 50 untreated mice of each gender. Mean body weights of the dosed male and female rats and mice were lower than those of the corresponding controls; the depression in the amount of weight gained in the dosed male mice was, however, relatively slight. Except for the male rats, survival at the end of the bioassay was lower in the dosed groups of both rats and mice than in the corresponding control groups. At week 90, at least 57% of the rats and 54% of the mice were still alive. In some male and female rats there was a dose-related increase in the incidence of hepatotoxic alterations characterized by fatty metamorphosis or necrosis. Also in mice, toxic hepatic lesions including liver degeneration, necrosis, fibrosis and/or cirrhosis were observed in slightly increased numbers in the dosed mice — particularly in the high-dose females. No tumors were induced in rats or mice of either gender at incidences that were significantly higher in the dosed groups than in the corresponding control groups. The authors concluded that unsubstituted dibenzo-p-dioxin exhibited very low toxicity and was noncarcinogenic in Osborne-Mendel rats and B6C3F1 mice, even when the maximum tolerated dose was approached (10,000 ppm in diet).

The Thomas et al. (1940) report consisted of two studies, a primary 200-day dibenzofuran feeding study and a follow-up 78-day study. In the primary study, groups of five female albino rats (strain not specified), approximately 30 days old, consumed 0, 250, 500, 1000, 2000, or 4000 ppm of dibenzofuran in their food for 200 days. In addition, two female rats consumed 8000 ppm of dibenzofuran in their diet for a shorter period (approximately 100 days). According to the authors, none of the animals exhibited any abnormal activity or behavior, nor was food intake appreciably altered by dibenzofuran administration, although it was noted that the rats receiving dibenzofuran tended to consume more water than controls. The authors also reported no effect on body weight gain at any dose during the exposure period; however, decreases in body length and absolute organ weights were observed in all dibenzofuran-exposed groups at necropsy. The authors also reported that the treated animals had unusually large amounts of abdominal fat, which they interpreted as accounting for the lack of effect on body weight gain. Quantitative data were not provided to support the assertions of no appreciable

changes in food intake or body weight gain, decreases in organ weight and overall length, and excess abdominal fat. In addition, the authors did not report whether a dose-response effect was observed for changes in body length or organ weight, or for excess abdominal fat.

Histological examination of the liver, kidney, spleen, heart, and adrenals was performed in rats exposed to dibenzofuran at 500 ppm and higher, and in the control animals (Thomas et al., 1940). The low dose group (250 ppm) apparently was not examined for histopathology. In the kidney, histological examination of rats exposed to concentrations of 500 ppm and higher revealed fine, brown-pigmented granules in the epithelial cells of proximal convoluted tubules in the deeper parts of the renal cortex. This effect was noted among all rats receiving dibenzofuran, and both the amount of pigmented material within cells and the frequency of occurrence among cells increased with dose of dibenzofuran. In addition, the two rats fed diet containing 8000 ppm dibenzofuran exhibited prominent, irregular dilatation of the collecting tubules with coagulated material resembling protein; other tubules in these two rats were slightly dilated and contained more granular and amorphous material than controls. These effects were reported as occurring without cellular degeneration or glomerular abnormalities. Some (frequency not specified) of the kidneys from rats receiving 4000 ppm showed similar, but less severe, changes. These lesions were not reported among rats fed the lower doses of dibenzofuran. However, quantitative data were not reported. In the spleen, slight hyperplasia of the Malpighian bodies was reported among several rats (frequency not given) in the 4000 and 8000 ppm groups. No alterations, other than reduced organ weight, were noted in the liver, heart, or adrenals of the treated rats.

In the follow-up study to determine whether dietary dibenzofuran affected water balance, an effect noted qualitatively (increased water consumption) in female rats receiving dibenzofuran in their food, Thomas et al. (1940) exposed groups of five male rats (average initial body weight 255 grams) to 0 or 5000 ppm of dibenzofuran in the diet for 78 days. Treated rats exhibited greater water consumption and urine output than controls, suggesting that dibenzofuran altered water balance. The excess in urine output was greater than the excess in water consumption in the treated group, suggesting a slight dehydration of tissues. The authors reported that no alterations in hematological parameters were observed (hemoglobin and erythrocyte, leukocyte, and reticulocyte counts). Tables 1 and 2 have summarized the hematological data reported in the 78-day study.

TABLE 1. Blood cell types in rats exposed to DBF in normal diet for 78 days					
Dose	Rats "N"	Hemoglobin	Erythrocytes	Reticulocytes	White cells
0	10	16.3%	8.12×10^6	3.0%	1.44×10^4
5000 ppm	5	16.6%	9.07×10^6	2.35%	1.65×10^4

TABLE 2. Average differential white blood cell counts in 78-day exposed rats vs. "normal rat blood"

Dose	Rats "N"	Lymphocytes	Polymorphonuclear neutrophils	Monocytes	Basophiles	Eosinophils
"Normal"	---	67.9%	27%	5.3%	0.77%	2.1%
5000 ppm	5	63.8%	33.5%	1.18%	0.64%	0.94%

In contrast to qualitative observations reported among the female rats exposed to similar concentrations in the 200-day primary study, the male rats treated for 78 days tended to consume less food than the controls and had a slightly lower rate of body weight gain than the control group. These data and water consumption data are summarized in Table 3. The authors noted that the odor and taste of dibenzofuran at 5000 ppm in the food was distinctly noticeable and may have contributed to this effect. Histological examination was not performed on tissues from these rats.

TABLE 3. Weight gain in male albino rats fed DBF for 78 days vs. controls

Dose	Rats "N"	Weight gain	Food ingestion	Water ingestion
0	5	321 g	6108 g	9652 cc
5000 ppm	5	243 g	5482 g	10,316 cc
Difference	-----	78 g (24%)	626 g (10%)	664 cc (6.9%)

The literature search revealed additional, peripheral data for dibenzofuran, including those for soil nitrification organisms (Sverdrup et al., 2002), drought resistance of certain insects (Sjursen et al., 2001), plant seedling growth (Sverdrup et al., 2003), fungi-specific enzyme systems (Kurihara et al., 2002), and a study of human intellectual effects of exposure (Schantz, 2001) that mistakenly referred to unhalogenated dibenzofuran. Abstracts for these studies reported the following conclusions.

- 75 mg DBF/kg (soil) NOEL for soil nitrification and no effects on soil bacterial diversity (Sverdrup et al., 2002)

- No dose-related decrease in drought tolerance in adult soil-dwelling insects, *Folsomia fimetaria* (Sjursen et al., 2001)
- 20% reduction in plant seedling weight when exposed to 43-93 mg DBF /kg soil (Sverdrup et al., 2003)
- No change in expression of NADH-ubiquinone oxidoreductase (NUO) among DBF-exposed fungus, *Phanerochaete chrysosporium* (Kurihara et al., 2002)

DERIVATION OF A PROVISIONAL SUBCHRONIC ORAL RfD VALUE FOR DIBENZOFURAN

The only subchronic or chronic toxicity data available for dibenzofuran were from the 200-day and 78-day feeding studies described by Thomas et al. (1940). These studies, though of apparently high quality for their era, had a number of major short comings, including the following:

- only qualitative data were reported for most endpoints
- only five organs were examined in the pathology
- the lowest dose group was not subjected to pathology examinations

No pertinent developmental or reproductive data were found for dibenzofuran. The LOAEL data from the Thomas et al. (1940) 200-day feeding study provided the POD for this derivation, because no NOAEL was reported. Data from the 78-day study were used to confirm food ingestion rates estimated using default rates in U.S. EPA, 1986b. Benchmark dose modeling was considered infeasible because adverse effects and the dose-response nature of the response were reported only qualitatively.

The lowest dose tested in the 200-day Thomas et al., 1940 study, 250 ppm in diet, was selected as the LOAEL POD for the aggregate critical effects of reduced length and organ weight, and excess abdominal fat. Ingestion data from the 78-day study was used to estimate the actual doses to the animals treated at the LOAEL, as follows. The 78-day feeding study was conducted under the same conditions as the 200-day primary study. This estimation made the following assumptions.

- Data from the 78-day study (Thomas et al., 1940) were more likely to represent actual food intakes than the default reference food factor from U.S. EPA, 1986b
- Rats in the 200-day study (Thomas et al., 1940) eating a diet treated with 250 ppm dibenzofuran consumed quantities of food closer to the control amounts (6108 g/diet/5 rats) than to the quantities of food treated with 5000 ppm dibenzofuran (5482 g/5 rats) in the 78-day study

- Growth of rats eating the 250 ppm diet in the 200-day study (Thomas et al., 1940) more closely approximated controls than those eating 5000 ppm, and that the 78-day weight provided a reasonable average weight for the 200 day study period.

In the 78-day study, Thomas et al. (1940) reported that a group of 5 control rats ingested a total of 6108 grams of food over the 78 days and grew from 1.273 kg to 1.594 kg/group, while experimental rats ingested 5482 g of food treated with 5000 ppm dibenzofuran and grew from 1.274 kg to 1.517 kg/group of 5 treated rats. The following calculations used food consumption data from the 78-day study to estimate dibenzofuran consumption in the 200-day study at the POD (250 ppm) for the critical effect of reduced length and organ weight, and excess abdominal fat among the exposed rats.

$$(6108 \text{ g diet}/5 \text{ rats}) / 78 \text{ days} = 78.3 \text{ g/diet}/5 \text{ rats/day}$$

$$(78.3 \text{ g}/5 \text{ rats/day}) \times (250/10^6) = 0.0196 \text{ g DBF}/5 \text{ rats/day} = 19.6 \text{ mg}/5 \text{ rats/day}$$

$$19.6 \text{ mg DBF}/5 \text{ rats/day} / (1.594 \text{ kg}/5 \text{ rats}) = 12.3 \text{ mg DBF}/\text{kg/day}$$

The estimated dibenzofuran dose of 12.3 g/kg/day was essentially the same as the dose of 12.5 g/kg/day calculated using the EPA default reference food factor (U.S. EPA, 1986b).

Based on the data available, the following uncertainty factors were applied to derive a subchronic oral p-RfD.

- 10 for variability in human susceptibility
- 10 for the uncertainty in animal-to-human extrapolation
- 1 for using data from a 200-day study (in rats) to derive a subchronic p-RfD
- 3 ($10^{0.5}$) for using a minimal LOAEL instead of a NOAEL
- 10 for deficiencies in the database, including the lack of reproductive and developmental data, and the minimal data details reported in the key study

The uncertainty factors noted above provide a composite UF of 3000 ($10^{3.5}$).

In the absence of a NOAEL, a LOAEL could be several orders of magnitude above the actual no adverse effect dose, since it merely represents the lowest dose tested. Nevertheless, the uncertainty factor for using a minimal LOAEL instead of a NOAEL was reduced from 10 to 3 ($10^{0.5}$) because the following findings suggested that the smaller uncertainty factor would be more appropriate in this case. While many of the dose levels tested and the organism effects considered in the following reports would be difficult to relate to humans, together they seem to

emphasize the relatively low toxicity and mild effects of dibenzofuran across a variety of species.

- The Thomas et. al (1940) study noted relatively minor effects in rats, even at very high doses, up to thirty times the LOAEL dose selected as the POD
- Peripheral data in other species indicated very minor effects or no effects among organisms exposed to dibenzofuran
 - 75 mg DBF/kg (soil) NOEL for soil nitrification and for soil bacterial diversity (Sverdrup et al., 2002)
 - No dose-related decrease in drought tolerance in the adult soil-dwelling insects, *Folsomia fimetaria* (Sjursen et al., 2001)
 - 20% reduction in plant seedling weight when exposed to 43-93 mg DBF /kg soil (Sverdrup et al., 2003)
 - No change in expression of NADH-ubiquinone oxidoreductase (NUO) among DBF-exposed *Phanerochaete chrysosporium* fungi (Kurihara et al., 2002)
- NCI (1979) reported no tumors and relatively low toxicity among rats and mice fed diets containing 5000 ppm and 10,000 ppm dibenzo-p-dioxin, a structural analog to dibenzofuran. Effects reported were hepatic lesions, slight reductions in weight gain and nephropathy (in male rats)

Applying the composite UF of $10^{3.5}$ (~3000) to the dietary LOAEL POD of 12.3 mg DBF/kg-day for the combined critical effects of reduced length and organ weight and excess abdominal fat observed in female albino rats allowed the following calculation of the subchronic p-RfD.

$$\begin{aligned}
 \text{Subchronic oral p-RfD} &= \text{LOAEL} / (\text{UF} \times \text{MF}) \\
 &= (12.3 \text{ mg/kg/day}) / (10^{3.5} \times 1) \\
 &= 4 \times 10^{-3} \text{ mg/kg-day} \\
 &= 4 \text{ } \mu\text{g dibenzofuran/kg-day}
 \end{aligned}$$

The data were insufficient to derive a chronic oral p-RfD value using an acceptable composite uncertainty. However, the Appendix of this document contains a Screening Value that may be useful in certain instances. Please see the attached Appendix for details.

DERIVATION OF PROVISIONAL INHALATION RfC VALUES FOR DIBENZOFURAN

Provisional inhalation RfC values were not derived for dibenzofuran because no useful inhalation exposure data were identified and data were insufficient to attempt inter-route extrapolation from the marginal ingestion data.

STATEMENT OF CONFIDENCE

Confidence in the principal study is low. Thomas et al. (1940) examined a number of endpoints, including histological examination of several major organs. The study had an adequate number of dose groups, but was limited by inclusion of only five rats in each group. Although only female rats were used for the 200-day portion of the study, male rats were used for the shorter water balance study (78 days). Thomas et al. (1940) did not report whether the critical effect selected displayed a dose-response relationship. However, the reductions in growth and organ weights, and the increase in abdominal fat were supported by histological changes noted in the kidney and impairment of water balance at higher doses. Because the critical effects were observed among rats receiving the lowest dose tested, one cannot be certain that the effects noted at 250 ppm (12.5 mg/kg-day), would not have been present at lower doses. Thus, it is uncertain whether 250 ppm is a true LOAEL. Confidence in the database and the resulting RfDs is low because of the limited toxicity data base for dibenzofuran, including lack of human studies and chronic, developmental, or reproductive oral animal studies. However, some confidence is gained from the relatively low toxicity and lack of tumors among rats and mice fed high doses of dibenzo-p-dioxin (NCI, 1979), the chemical identified by NCI (2000) as most structurally related to dibenzofuran. Nevertheless, risk managers are advised to consider any other available data before applying this p-RfD.

Suppliers and users of dibenzofuran should be encouraged to conduct toxicology studies, such as that initiated by EPA in 1978 (NCI, 2000) but then terminated because of lack of funding. The absence of inhalation, toxicokinetic, and metabolic data would justify especially encouraging studies to seek such information.

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APPENDIX

DERIVATION OF A SCREENING VALUE FOR DIBENZOFURAN

For reasons noted in the main PPRTV document, it is inappropriate to derive provisional toxicity values for Dibenzofuran, chronic RfD. However, information is available for this chemical which, although insufficient to support derivation of a provisional toxicity value, under current guidelines, may be of limited use to risk assessors. In such cases, the Superfund Health Risk Technical Support Center summarizes available information in an Appendix and develops a "Screening Value." Appendices receive the same level of internal and external scientific peer review as the PPRTV documents to ensure their appropriateness within the limitations detailed in the document. In the OSRTI hierarchy, Screening Values are considered to be below Tier 3, "Other (Peer-Reviewed) Toxicity Values."

Screening Values are intended for use in limited circumstances when no Tier 1, 2, or 3 values are available. Screening Values may be used, for example, to rank relative risks of individual chemicals present at a site to determine if the risk developed from the associated exposure at the specific site is likely to be a significant concern in the overall cleanup decision. Screening Values are not defensible as the primary drivers in making cleanup decisions because they are based on limited information. Questions or concerns about the appropriate use of Screening Values should be directed to the Superfund Health Risk Technical Support Center.

The Thomas et al. (1940) study provided insufficient data to derive a chronic oral p-RfD value with uncertainty in an acceptable range. The 200-day rat minimal LOAEL POD of 12.3 mg/kg-day was considered to derive a **screening chronic oral reference dose** by applying a composite uncertainty factor of 10,000 (10^4), including 10 for variability in human susceptibility, 10 for animal-to-human extrapolation, 3 ($10^{0.5}$) for extrapolating from 200-day rat data to a chronic screening value, 3 ($10^{0.5}$) for using a minimal LOAEL instead of a NOAEL, and 10 for deficiencies in the database, including the lack of developmental data and the minimal data details reported in the key study.

Applying the minimal LOAEL dietary POD of 12.3 mg DBF/kg-day and the composite uncertainty factor of 10,000 (10^4) allowed the following calculation:

$$\begin{aligned}
 \text{Screening chronic oral p-RfD} &= \text{LOAEL/UF} \\
 &= (12.3 \text{ mg/kg-day})/10^4 \\
 &= \underline{\underline{1 \times 10^{-3} \text{ mg/kg-day}}} \\
 &= 1 \text{ } \mu\text{g dibenzofuran/kg-day}
 \end{aligned}$$

Confidence in the key study was low, because of the lack of detail on the critical effects and other deficiencies noted in this document. Given the lack of additional studies, confidence in the database also was low, leading to low overall confidence in the screening toxicity value. Users are advised to consider any other available data and to consult with the STSC before using this screening p-RfD.

Provisional Peer Reviewed Toxicity Values for

Endosulfan sulfate

(CASRN 1031-07-8)

Derivation of a Carcinogenicity Assessment

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit

NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
ENDOSULFAN SULFATE (CASRN 1031-07-8)
Derivation of a Carcinogenicity Assessment**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

An assessment of the carcinogenicity of endosulfan sulfate is not available on IRIS (U.S. EPA, 2002), or in the HEAST (U.S. EPA, 1997) or Drinking Water Standards and Health Advisories list (U.S. EPA, 2000). The CARA list (U.S. EPA, 1991a, 1994) includes a Health Effects Assessment for α - and β -endosulfan (U.S. EPA, 1987) that included no cancer data for endosulfan sulfate. Subsequent U.S. EPA documents on endosulfan have also reported no cancer data for endosulfan sulfate (U.S. EPA, 1991b, 1999). IARC (2002) has not evaluated endosulfan sulfate for carcinogenicity. Review endosulfan documents by ATSDR (2000), which included endosulfan sulfate, and WHO (1984), as well as the NTP (2002) status reports, were also consulted for relevant information. Literature searches were conducted from 1998 to December 2001 for studies relevant to the derivation of an oral slope factor for endosulfan sulfate. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, RTECS, GENETOX, HSDB, CCRIS, TSCATS, EMIC/EMICBACK and DART/ETICBACK.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No studies were located regarding the carcinogenic effects of endosulfan sulfate in humans following exposure by any route.

Animal Studies

No studies were located regarding the carcinogenic effects of endosulfan sulfate in animals following exposure by any route.

Other Studies

Dorough et al. (1978) observed no evidence of mutagenicity with or without S-9 liver homogenate in *Salmonella typhimurium* strains TA1535 or TA1978 (up to 100 µg/plate), or TA98 or TA100 (up to 1000 µg/plate).

FEASIBILITY OF DERIVING A PROVISIONAL ORAL SLOPE FACTOR FOR ENDOSULFAN SULFATE

Derivation of a provisional oral slope factor for endosulfan sulfate is precluded by the lack of human or animal cancer data.

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Provisional Peer Reviewed Toxicity Values for

Endosulfan sulfate
(CASRN 1031-07-8)

Derivation of a Chronic Oral RfD

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
ENDOSULFAN SULFATE (CASRN 1031-07-8)
Derivation of a Chronic Oral RfD**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

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 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

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Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

An RfD for endosulfan sulfate is not available on IRIS (U.S. EPA, 2002), the Drinking Water Standards and Health Advisories list (U.S. EPA, 2000a), or the HEAST (U.S. EPA, 1997). Existing regulations for endosulfan sulfate have been based on data for endosulfan. Endosulfan is composed of two isomers: α -endosulfan (70%) and β -endosulfan (30%). Technical endosulfan is at least 94% endosulfan isomers, but also contains a small amount of endosulfan sulfate, which occurs as a result of oxidation, biotransformation, or photolysis of endosulfan isomers (ATSDR, 2000). For endosulfan sulfate, the national recommended water quality criteria human health values for consumption of "water + organism" and "organism only" are 110 and 240 $\mu\text{g/L}$, respectively (U.S. EPA, 1999a), derived from the RfD for endosulfan (U.S. EPA, 2002). WHO (1982) derived a temporary acceptable daily intake for man of 0 - 0.008 mg/kg body weight for α -endosulfan, β -endosulfan and endosulfan sulfate combined, based on unpublished endosulfan

feeding studies in dogs and rats; NOAEL values were 0.75 and 1.5 mg/kg, respectively (Hazleton Laboratories, 1959a,b). The CARA list (U.S. EPA, 1991a, 1994) includes an Ambient Water Quality Criteria Document (U.S. EPA, 1980) and a Health Effects Assessment for α - and β -endosulfan (U.S. EPA, 1987). A Health and Environmental Effects Document for Endosulfan was also identified (U.S. EPA, 1991b). Other EPA documents (1999b, 2000b, 2001a,b,c), a Toxicological Profile for endosulfan (ATSDR, 2000), the NTP (2002) status report, and IARC (1998) and WHO documents (1982, 1984, 1988, 1989) were consulted for relevant information. Literature searches were conducted from 1998 to December 2001 for studies relevant to the derivation of an RfD for endosulfan sulfate. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, RTECS, GENETOX, HSDB, CCRIS, TSCATS, EMIC/EMICBACK and DART/ETICBACK.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No studies were identified that investigated the effects of repeated-dose oral exposure of humans to endosulfan sulfate.

Animal Studies

No animals studies were identified that investigated the effects of repeated-dose oral exposure to endosulfan sulfate.

Other Studies

Endosulfan sulfate is the primary metabolite of both α - and β -endosulfan, and has been detected in serum and tissue samples of orally-exposed animals (Braun and Lobb, 1976; Cole and Casida, 1986; Das and Garg, 1981; Deema et al., 1966; Gorbach et al., 1968; Schuphan et al., 1968) and people following acute ingestion of endosulfan (Boereboom et al., 1998; Coutselinis et al., 1978; Demeter et al., 1977; Demeter and Heyndrickx, 1978). Risk assessments and reviews for endosulfan (U.S. EPA, 2001a,b,c; ATSDR, 2000; IARC, 1998; WHO, 1984, 1988) have concluded that α -endosulfan, β -endosulfan, and endosulfan sulfate are approximately equal in toxicity; however, limited toxicity data support this conclusion.

Dorough et al. (1978) reported the results of acute lethality testing in female albino rats (27-30 g) using the method of Deichmann and LeBlanc (1943) for α -endosulfan, β -endosulfan, endosulfan sulfate and four other endosulfan metabolites. Compounds were administered orally in a 1:1 mixture of water:Tween 80. The LD₅₀ approximation for endosulfan sulfate was 8

mg/kg. Lethal doses of this compound caused convulsions and death within 1 hour of treatment, but survivors showed “almost no symptoms of poisoning.”

In vitro assays showed that endosulfan sulfate and endosulfan affected similar endpoints, and that the sulfate was less toxic. Endosulfan sulfate did not induce mutations in *Salmonella typhimurium* (Dorough et al., 1978). Dubey et al. (1984) observed that the toxicity of endosulfan sulfate was approximately half the toxicity of endosulfan in measurements of rat liver mitochondrial respiration and enzyme activities. Ruch et al. (1990) also observed that endosulfan sulfate was approximately half as effective as endosulfan in the inhibition of gap junctional intercellular communication in primary cultured F344 rat and B6C3F1 mouse hepatocytes. Flodstrom et al. (1988) reported that very similar toxicities were observed for endosulfan sulfate, α -endosulfan, β -endosulfan, technical endosulfan and analytical endosulfan in cytotoxicity assays and intercellular communication assays in WB rat liver cells and wild type and mutant Chinese hamster V79 lung fibroblast cells. Endosulfan sulfate and α -endosulfan exhibited similar activities in a cell free assay using extracts from adult rat prostate tissue (Brieske et al., 1997); both slightly reduced binding of the androgen [^3H]-methyltrienolone to androgen receptor protein.

FEASIBILITY OF DERIVING A PROVISIONAL RfD FOR ENDOSULFAN SULFATE

Derivation of a provisional reference dose for endosulfan sulfate is precluded by the lack of adequate human or animal studies.

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From: <Nunes.Robert@epamail.epa.gov>
To: <conklit@obg.com>, <sinhap@obg.com>
CC: <sledward@gw.dec.state.ny.us>, <txsmith@gw.dec.state.ny.us>, <sivak.mich...
Date: 3/27/2008 1:21 PM
Subject: Fw: Toxicity Request for Onondaga Lake (Wastebeds 1-8) NY
Attachments: pic27876.jpg; Onondaga Lake - Metz 1.pdf; Onondaga Lake - Metz 2.pdf; Was
tebeds 1-8 toxicity request.pdf

Tom, Ricky - Forwarded here are NCEA recommended toxicity values for the
WBs 1-8 HHRA. Since some of these chemicals are also being evaluated in
the WB B/HB HHRA, OBG should utilize them for that site as well.
Similarly, NCEA recommendations which are being utilized for chemicals
evaluated for WB B/HB should be used in the WBs 1-8 HHRA if the
chemicals are also being evaluated there.

If you have any questions, please let me know.

Thank you.

Bob Nunes
New York Remediation Branch
Emergency and Remedial Response Division
US EPA Region II
290 Broadway, 20th Floor
New York, NY 10007-1866
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----- Forwarded by Robert Nunes/R2/USEPA/US on 03/27/2008 11:30 AM -----

Chloe
Metz/R2/USEPA/US

To
03/25/2008 06:04 PM Robert Nunes/R2/USEPA/US@EPA
cc

Michael Sivak/R2/USEPA/US@EPA

Subject
Fw: Re: Toxicity Request for
Onondaga Lake (Wastebeds 1-8) NY

Bob, attached are the NCEA recommended toxicity values and supporting

PPRTV documentation for Wastebeds 1-8. OBG should use the values in the attached spreadsheet to evaluate chemicals not in IRIS. Some of the chemicals may overlap with the request that Michael had for Harbor Brook/Wastebed B, in which case the recommendations provided here can be used for that site as well.

Michael, Marian looked into the 1,4-dichlorobenzene schedule and it looks like May 2008 is more than optimistic, so we shouldn't expect a new IRIS value any time real soon.

Let me know if you have any questions.

Thanks,
Chloe

Chloe Metz
Superfund Program
US EPA, Region 2
290 Broadway, 18th Floor
New York, NY 10007

212.637.4449 (voice)
212.637.3083 (fax)

-----Forwarded by Chloe Metz/R2/USEPA/US on 03/25/2008 05:54PM -----

To: Chloe Metz/R2/USEPA/US@EPA
From: SUPERFUND STSC
Sent by: Stacey Lewis/CI/USEPA/US
Date: 03/24/2008 12:50PM
cc: SUPERFUND STSC@EPA
Subject: Re: Toxicity Request for Onondaga Lake (Wastebeds 1-8) NY

(Embedded image moved to file: pic27876.jpg)

Hello Chloe,

We are waiting on a decision regarding the use of the sRfC presented in 2-Nitrophenol's PPRTV as an RfC, but the three files attached address the remaining portions of your request. The first file, "Chloe Metz - Onondaga Tox request.pdf", is a table outlining where toxicity information can be found if available. The remaining two files are the PPRTVs that are available for the requested chemicals. We will notify you as soon as a decision is made concerning 2-Nitrophenol's sRfC and RfC.

Please feel free to contact the Center if you have any questions or concerns regarding this response.

Respectfully

Stacey Lewis
STSC

(See attached file: Onondaga Lake - Metz 1.pdf)(See attached file:
Onondaga Lake - Metz 2.pdf)(See attached file: Wastebeds 1-8 toxicity
request.pdf)



Superfund Technical Support Center

National Center for Environmental Assessment

U.S. Environmental Protection Agency

26 West Martin Luther King Drive, MS-AG41

Cincinnati, Ohio 45268

Jon Reid/Director, Pat Daunt/Administrator

Hotline 513-569-7300, FAX 513-569-7159, E-Mail: STSC.Superfund@epa.gov

March 28, 2008

Michael Sivak
U.S. EPA, Region 2

ASSISTANCE REQUESTED: PPRTVs for 2,4-Dimethylphenol, 2,4-Dinitrophenol, 2,6-Dinitrotoluene, 2-Chlorophenol, 2-Methylnaphthalene and 2-Nitrophenol (*Onondaga Lake*)

ENCLOSED INFORMATION:

- Attachment 1: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2,4-DIMETHYLPHENOL (CASRN 105-67-9)**
- Attachment 2: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2,4-DINITROPHENOL (CASRN 51-28-5)**
- Attachment 3: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2,6 -DINITROTOLUENE (CASRN 606-20-2)**
- Attachment 4: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2-CHLOROPHENOL (CASRN 95-57-8)**

Attachment 5: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR 2-METHYLNAPHTHALENE (CASRN
91-57-6)**

Attachment 6: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR 2-NITROPHENOL (CASRN 88-75-5)**

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (6)

cc: STSC Files

6-27-2007

Provisional Peer Reviewed Toxicity Values for
2,4-Dimethylphenol
(CASRN 105-67-9)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2,4-DIMETHYLPHENOL (CASRN 105-67-9)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

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INTRODUCTION

2,4-Dimethylphenol (2,4-DMP), also known as m-xylenol, 2,4-xylenol or m-4-xylenol, is a naturally occurring, substituted phenol derived from the cresol fraction of petroleum or coal tars. 2,4-DMP has the empirical formula $C_8H_{10}O$ (Figure 1). It is used in the manufacture of a wide range of commercial products for industry and agriculture. There are six isomeric forms of dimethylphenol that exist (2,3-, 2,4-, 2,5-, 2,6-, 3,4- and 3,5-dimethylphenol).

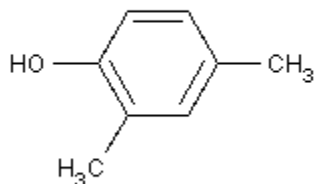


Figure 1. 2,4-Dimethylphenol Structure

The EPA's Integrated Risk Information System (IRIS) (U.S. EPA, 2007) lists a chronic oral reference dose (RfD) of $2E-2$ mg/kg-day for 2,4-dimethylphenol based upon data in an unpublished 90-day gavage study in mice (U.S. EPA, 1989). The chronic RfD was derived from

the NOAEL of 50 mg/kg-day for clinical signs of toxicity (lethargy, prostration and ataxia) and a composite uncertainty factor of 3000 (10 for interspecies variability, 10 for intraspecies variability and 30 for lack of chronic toxicity data, data in a second species and reproductive/developmental studies). IRIS does not list a chronic inhalation reference concentration (RfC) or derive a cancer oral slope factor or inhalation unit risk for 2,4-DMP (U.S. EPA, 2007). The HEAST (U.S. EPA, 1997) lists a subchronic RfD of 0.2 mg/kg-day based on the NOAEL from the same principal study identified by IRIS and an uncertainty factor of 300. The Drinking Water Standards and Health Advisories list (U.S. EPA, 2006) does not include an RfD or carcinogenicity assessment for 2,4-dimethylphenol. An Ambient Water Quality Criteria Document for 2,4-DMP does not include an RfD or carcinogenicity assessment, but does lists a criterion level of 400 µg/L based upon undesirable organoleptic qualities, which is more a function of aesthetic property of water than a health effect (U.S. EPA, 1980). The Chemical Assessments and Related Activities (CARA) list (U.S. EPA, 1991, 1994) includes both a Health and Environmental Effects Profile (HEEP) (U.S. EPA, 1986) and a Health Effects Assessment (HEA) (U.S. EPA, 1985) for dimethylphenols. Neither the HEEP (U.S. EPA, 1986) nor the HEA (U.S. EPA, 1985) derived toxicity values for 2,4-DMP, citing insufficient data. Neither the ATSDR (2006), National Toxicology Program (NTP) (2006), International Agency for Research on Cancer (IARC) (2006) nor the World Health Organization (WHO) (2006) has produced documents regarding 2,4-DMP. No occupational exposure limits have been derived by the Occupational Safety and Health Administration (OSHA), the National Institute of Occupational Safety and Health (NIOSH), or the American Conference of Governmental Industrial Hygienists (ACGIH).

Literature searches for studies relevant to the derivation of provisional toxicity values for 2,4,-DMP (CASRN 105-67-9) were conducted from 1965 to August 2006 in TOXLINE (supplemented with BIOSIS and NTIS updates), MEDLINE, TSCATS, RTECS, CCRIS, DART, EMIC/EMICBACK, HSDB, GENETOX, CANCERLIT and Current Contents.

REVIEW OF PERTINENT LITERATURE

Human Studies

No studies investigating the effects of subchronic or chronic oral or inhalation exposure to 2,4-DMP in humans were identified.

Animal Studies

Oral Exposure

Chronic Studies – No studies investigating the effects of chronic oral exposure to 2,4-DMP in animals were identified.

Subchronic Studies – Studies on the subchronic toxicity of oral exposure to 2,4-dimethylphenol have been conducted in rats exposed for 10 and 90 days (Daniel et al., 1993) and

mice exposed for 14 (U.S. EPA, 1987) and 90 days (U.S. EPA, 1989). Additional studies evaluating the effects of subchronic exposure of animals to oral 2,4-DMP were not identified.

Groups of 10 male and 10 female Sprague-Dawley rats (80 days old) were administered 0 (vehicle control), 60, 120, 600 or 1200 mg/kg body weight of 2,4,-DMP in corn oil by gavage once daily for 10 consecutive days (Daniel et al., 1993). Rats were observed daily for mortality and physiological and behavioral signs of toxicity. Body weights were recorded on days 0, 4 and 6 of treatment and at the end of the study. Blood samples taken at the end of the treatment period were analyzed for the following: white blood cell (WBC) count, red blood cell counts (RBC), hemoglobin (Hgb), hematocrit (Hct), mean corpuscular volume (MCV), glucose, blood urea nitrogen (BUN), creatinine, alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), cholesterol, lactate dehydrogenase (LDH) and calcium (Ca^{++}). Organ weights were recorded and gross examination of comprehensive tissues was performed at the end of the treatment period. All tissues from the control and 600 mg/kg groups were examined microscopically. As target organs for 2,4,-DMP were identified, target tissues from the 60 and 120 mg/kg groups were examined microscopically.

All male and female rats treated with 1200 mg/kg body weight 2,4-DMP died prior to completion of the 10-day treatment period (time of death not reported) (Daniel et al., 1993). The study authors attributed the cause of death to 2,4-DMP-induced severe stomach lesions. Mortalities in other groups were as follows: 1 male in the control group, 1 female in the 120 mg/kg group, and 2 females and 1 male in the 600 mg/kg group. The cause of death or relationship to treatment was not reported. No mortalities occurred in the 60 mg/kg group. Clinical and behavioral signs of toxicity were not reported for any dose group. In surviving animals, food and water intake, final body weight and body weight gain were not significantly different from controls in any 2,4-DMP group. Relative liver weight was significantly increased in females, but not males, in the 600 mg/kg group compared to control (Table 1). No increase in relative liver weight was observed in males or females in other 2,4-DMP groups. Relative weights of other organs were not affected by treatment. Absolute organ weights were not reported.

Effects on hematological and clinical chemistry parameters were observed only in the high-dose group, except for decreased AST in females in the 60 mg/kg-day group and decreased Ca^{++} in males in the 120 mg/kg-day group, as shown in Table 2 (Daniel et al., 1993). In general, relative to control, the observed effects were minimal. Significant increases in WBC and Hgb values were observed in females, but not males, in the 600 mg/kg-day treatment group. No other effects on hematological parameters were observed for any treatment group for females or males. In females, mean serum glucose and cholesterol levels were significantly increased in the 600 mg/kg group and AST levels were significantly decreased in the 60 and 600 mg/kg dose groups (Table 2); however, AST levels were not significantly different from control in the 120 mg/kg group. In male rats, serum Ca^{++} was decreased in the 120 and 600 mg/kg groups and AST was significantly decreased in the 600 mg/kg group (Table 2). Serum cholesterol was increased in both males and females treated with 600 mg/kg-day (Table 2).

Table 1. Effect of Oral Treatment of Rats with 2,4-DMP (10 Day Exposure) on Final Body Weight and Relative Liver Weight (Daniel et al., 1993)				
Parameter	Treatment Group (mg/kg-day)			
	0	60	120	600
Females				
Number of animals	10	10	9	8
Final body weight (g)	234.2±10.1 ^a	237.8±13.0 (101.5)	233.8±11.2 (99.8)	224.9±12.9 (98.0)
Relative Liver weight (%)	2.97±0.24	3.13±0.23 (105.0)	3.19±0.16 (107.0)	3.50±0.32 (117.4) ^b
Males				
Number of animals	9	10	10	9
Final body weight (g)	354.0±19.8	347.9±18.1 (98.3)	358.1±30.2 (101.2)	335.2±21.2 (94.7)
Relative Liver weight (%)	3.05±0.21	3.09±0.22 (101.3)	3.14±0.21 (103.0)	3.29±0.49 (107.9)
^a Values are means ± Standard Deviation (SD); () = percent of control				
^b Significantly different from control (p≤0.05), Analysis of Variance (ANOVA)				

Table 2. Effect of Oral Treatment of Rats with 2,4-DMP (10 Day Exposure) on Hematology and Serum Chemistry Values (Daniel et al., 1993)				
Parameter	Treatment Group (mg/kg-day)			
	0	60	120	600
Females				
WCB (x10 ³)	7.0±1.8 ^a	8.6±1.7 (122.8)	7.3±1.8 (104.3)	9.5±2.3 (135.7) ^b
Hgb (g/dL)	14.9±0.9	15.1±0.5 (101.3)	15.3±0.7 (102.7)	16.2±1.2 (108.7) ^b
Glucose (mg/dL)	95.5±15.4	117.4±14.1 (122.9)	107.8±22.1 (112.9)	138.0±24.2 (144.5) ^b
Cholesterol (mg/dL)	71.6±12.1	78.6±11.6 (109.8)	86.5±9.7 (120.8)	110.9±38.0 (154.9) ^b
AST (IU ^c /L)	111.3±18.7	89.6±13.1 (80.5) ^b	95.3±20.5 (86.5)	84.4±13.4 (75.8) ^b
Ca ⁺⁺ (mg/dL)	10.1±0.5	10.2±0.6 (101.0)	10.5±0.5 (104.0)	10.7±1.2 (103.0)
Males				
WCB (x10 ³)	8.6±1.3	10.5±4.0 (122.1)	9.5±1.6 (110.5)	11.5±0.3 (133.7)
Hgb (g/dL)	15.6±0.5	15.8±0.6 (101.3)	16.0±0.7 (102.6)	16.1±0.4 (103.2)
Glucose (mg/dL)	109.2±12.3	120.4±25.9 (110.3)	124.3±27.9 (113.8)	135.9±27.6 (124.5)
Cholesterol (mg/dL)	62.1±10.6	64.2±8.8 (103.4)	60.3±9.8 (97.1)	65.0±17.0 (104.7) ^b
AST (IU/L)	102.5±18.7	101.1±38.2 (98.6)	99.8±15.3 (97.4)	80.4±13.1 (78.4) ^b
Ca ⁺⁺ (mg/dL)	10.6±0.3	10.1±0.3 (95.2)	9.7±0.6 (91.5) ^b	9.5±1.0 (89.6) ^b
^a Values are means ± SD; () = percent of control				
^b Significantly different from control (p≤0.05), ANOVA				
^c International Units (IU)				

Based on the cause of death (severe stomach lesions) for all rats in the 1200 mg/kg group, the stomach was identified as the primary target organ for exposure to 2,4-DMP by gavage (Daniel et al., 1993). In the 600 mg/kg group, lesions of the forestomach (including epithelial hypertrophy, hyperkeratosis and mucosal vacuolar degeneration) were observed in male and female rats. Although the authors state that the incidence and severity of forestomach lesions increased with dose, specific dose-response data were not presented and no information on stomach lesions in the 60 and 120 mg/kg group was reported. Therefore, it is unclear from this report if forestomach lesions were observed in all 2,4-DMP dose groups. Thus, due to inadequacy of reporting, NOAEL and LOAEL values cannot be determined for this 10-day study.

Groups of 10 male and 10 female Sprague-Dawley rats (80 days old) were administered 0 (vehicle control), 60, 180 or 540 mg/kg body weight of 2,4-DMP in corn oil once daily for 90 consecutive days by gavage (Daniel et al., 1993). Rats were observed daily for mortality and clinical and behavioral signs of toxicity. Body weights and food and water consumption were recorded weekly. Blood samples taken at the end of the treatment period were analyzed for the following: WBC count, red blood cell counts (RBC), platelet count, hemoglobin (Hgb), Hct, MCV, mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), glucose, BUN, creatinine, ALP, AST, ALT, cholesterol, triglycerides, LDH, gamma glutamyl transpeptidase (GGT), total bilirubin, direct bilirubin, total protein, albumin (A), globulin (G), Ca^{++} , sodium (Na^+), potassium (K^+), chloride (Cl^-), phosphate (PO_4) and magnesium (Mg^{++}). At the end of the treatment period, gross pathological examination was conducted on rats from all treatment groups. The stomach of all surviving animals was examined microscopically and histopathological examination was performed on all tissues from the control and high-dose group (180 mg/kg).

All females and 6 of 10 males treated in the 540 mg/kg group died by the fifth day of treatment (Daniel et al., 1993). Subsequently, 6 females and 6 males were added to the 540 mg/kg group, with a total of 3/16 females and 7/16 males surviving for the 90-day treatment period. The cause of death for all animals in the 540 mg/kg group was reported as corrosive effects of 2,4-DMP on the esophagus and stomach, based on findings of the gross pathological examination. No mortalities or clinical signs of toxicity were observed in female or male rats in control or other 2,4-DMP treatment groups. Final body weight was decreased approximately 10% in females in the 180 and 540 mg/kg groups (statistically significant only in the 180 mg/kg-day group) and in males in the 540 mg/kg group. Changes in relative organ weights generally appeared to be secondary to changes in body weight, with small increases in relative brain, liver, kidney, and (in males) testes weights at doses that also produced decreases in body weight (Table 3). Relative thymus weight was significantly decreased in males at 60 mg/kg-day, but not in higher dose males or females.

Hematological analysis revealed a 2.4% increase ($p \leq 0.05$) in MCV in females treated with 540 mg/kg body weight 2,4-DMP compared to control; however, the authors state that the magnitude of change was not considered biologically significant (Daniel et al., 1993). No other

Table 3. Effect of Oral Treatment of Rats with 2,4-DMP (90 Day Exposure) on Final Body Weight and Relative Organ Weights (Daniel et al., 1993)				
Parameter	Treatment Group (mg/kg-day)			
	0	60	180	540
Females				
Number of animals	10	10	10	3
Final body weight (g)	269.0±26.2 ^a	263.1±21.5 (97.8)	240.3±24.4 (89.3) ^b	244.6±21.0 (90.9)
Relative brain weight (%)	0.81±0.06	0.81±0.08 (100)	0.88±0.07 (108.6) ^b	0.93±0.13 (114.8)
Relative kidney weight (%)	0.74±0.04	0.70±0.04 (94.6) ^b	0.80±0.08 (108.1) ^b	0.82±0.04 (110.8)
Relative liver weight (%)	2.90±0.16	2.84±0.31 (97.9)	3.13±0.32 (107.9)	3.21±0.13 (110.7) ^b
Relative thymus weight (%)	0.12±0.03	0.12±0.02 (100)	0.10±0.02 (83.3)	0.11±0.03 (91.7)
Males				
Number of animals	10	10	10	7
Final body weight (g)	492.8±40.2	516.6±56.4 (104.8)	507.3±21.2 (102.9)	442.0±41.0 (89.7) ^b
Relative brain weight (%)	0.48±0.04	0.46±0.05 (95.8)	0.46±0.03 (95.8)	0.52±0.03 (108.3) ^b
Relative kidney weight (%)	0.70±0.04	0.67±0.04 (95.7)	0.69±0.04 (98.6)	0.77±0.06 (110.0) ^b
Relative liver weight (%)	3.17±0.39	2.97±0.32 (93.7)	3.13±0.18 (98.7)	3.30±0.35 (104.1)
Relative thymus weight (%)	0.08±0.01	0.06±0.01 (75.0) ^b	0.07±0.01 (87.5)	0.07±0.02 (87.5)
Relative testes weight (%)	0.72±0.12	0.66±0.09 (91.7)	0.68±0.07 (94.4)	0.83±0.08 (115.3) ^b
^a Values are means ± SD; () = percent of control				
^b Significantly different from control (p≤0.05), ANOVA				

hematology parameters were affected by 2,4-DMP treatment. Effects on clinical chemistry parameters were minor (Table 4). In females, mean phosphate levels in the low dose group and AST levels in the middle-dose group were significantly decreased. In high-dose females, the BUN/creatinine ratio and cholesterol levels were increased and creatinine and chloride levels were decreased. In high-dose males, serum creatinine and AST were significantly decreased, whereas cholesterol, triglycerides and Mg⁺⁺ were significantly increased.

Gross pathological examination at the end of the treatment period revealed a small, red thymus in a small percentage (percentage not reported) of males in the 60 and 540 mg/kg groups, but not in the 180 mg/kg group. Incidence data for gross thymus lesions were not reported and histopathological examination of the thymus was not performed. At the end of the 90-day treatment period, histopathological examination of the forestomach showed hyperkeratosis and epithelial hyperplasia in all males in the 180 and 540 mg/kg groups, 60% of females in the 180 mg/kg group and all females in the 540 mg/kg group. Severity of lesions increased with dose (data on severity not reported). Although histopathological assessment of the stomach was performed in the low-dose group, no data or information were presented; thus, it is unclear if lesions were present in rats treated with 60 mg/kg-day 2,4-DMP. No other treatment-related histopathological changes were observed in male or female rats. The study authors identified 60 mg/kg body weight as the NOAEL since “no biologically significant changes in frequency or severity of adverse effects” relative to control were observed; however, the “biologically

Table 4. Effect of Oral Treatment of Rats with 2,4-DMP (90 Day Exposure) on Serum Chemistry Values (Daniel et al., 1993)

Parameter	Treatment Group (mg/kg-day)			
	0	60	180	540
Females				
Creatinine (mg/dL)	0.71±2.8 ^a	0.68±0.11 (95.8)	0.70±0.13 (98.6)	0.57±0.06 (80.3) ^b
BUN/Creatinine ratio	26±5	26±4 (100)	31±7 (119.2)	41±5 (157.7) ^b
AST (IU/L)	149±29	137±69 (91.9)	121±15 (81.2) ^b	169±11 (113.4)
Cholesterol (mg/dL)	35±7	42±7 (120)	43±9 (122.9)	73±14 (208.6) ^b
Triglycerides (mg/dL)	64±17	65±20 (101.6)	56±27 (87.5)	63±5 (98.4)
Cl ⁻ (mEq/L)	98±4	100±2 (102.0)	99±3 (101.0)	95±2 (96.9) ^b
Mg ⁺⁺ (mEq/L)	2.1±0.2	2.0±0.1 (95.2)	2.1±0.1 (100)	2.2±0.2 (104.8)
PO ₄ (mEq/L)	5.3±0.5	4.5±0.9 (84.9) ^b	5.4±1.0 (101.9)	6.6±0.8 (124.5)
Males				
Creatinine (mg/dL)	0.61±0.06	0.61±0.11 (100)	0.59±0.03 (96.7)	0.54±0.05 (88.5) ^b
BUN/Creatinine ratio	30±6	32±7 (103.2)	30±4 (96.8)	34±5 (109.7)
AST (IU/L)	115±15	165±93 (143.5)	113±26 (98.3)	99±6 (86.1) ^b
Cholesterol (mg/dL)	38±9	40±13 (105.3)	43±12 (113.2)	48±5 (126.3) ^b
Triglycerides (mg/dL) ^c	38±9	40±13 (105.3)	43±12 (113.2)	48±5 (126.3) ^b
Cl ⁻ (mEq/L)	100±1	101±2 (101)	100±1 (100)	100±2 (100)
Mg ⁺⁺ (mEq/L)	1.8±0.2	1.9±0.1 (105.6)	1.9±0.2 (105.6)	2.0±0.1 (111.1) ^b
^a Values are means ± SD; () = percent of control ^b Significantly different from control (p≤0.05), ANOVA ^c For male rats, values for triglycerides as reported by study authors, were identical to those for cholesterol. Comparison of triglyceride and cholesterol concentrations for males and females indicated that triglyceride concentrations for males were incorrectly reported by Daniel et al. (1993).				

significant” effects serving as the basis for the LOAEL of 180 mg/kg body weight-day were not specifically identified. Based on results of this study, the stomach appears to be a target organ for orally administered 2,4,-DMP. Due to ambiguous reporting, it is unclear if 2,4-DMP induced stomach lesions in the 60 mg/kg-day group, introducing significant uncertainty to the NOAEL and LOAEL values reported by the study authors.

The oral toxicity of 2,4-DMP was evaluated in a 90-day study in albino mice (U.S. EPA, 1989). Data from this unpublished study serve as the basis for the chronic RfD for 2,4-DMP listed by IRIS (U.S. EPA, 2007). Groups of 30 male and 30 female albino mice [strain Crl:CD-1(ICR)BR-VAF+] were administered 5, 50 or 250 mg/kg body weight 2,4-DMP in corn oil by gavage for 90 days. Untreated control and vehicle control, consisting of 30 male and 30 female mice per group, were included. Mice were observed twice daily throughout the treatment period for mortality, morbidity and signs of toxicity. A 30-day interim sacrifice was performed on eight

males and nine females from each group. Body weights and food consumption were recorded weekly. Blood was analyzed for hematological (Hgb, Hct, RBC, total and differential leukocyte count, platelet count, reticulocyte count, MCV, MCH and MCHC) and clinical chemistry parameters (Ca^{++} , Cl^- , PO_4 , K^+ , Na^+ , glucose, creatinine, BUN, ALT, AST, LDH, ALP, albumin, globulin, total protein, total bilirubin and cholesterol) at the interim sacrifice (for mice sacrificed at 30 days) and at the end of the 90-day exposure period. Ophthalmologic examinations were conducted prior to study initiation and in all surviving mice at study termination. Necropsy was performed on all animals found dead during the study and in all surviving animals at the end of the 90-day exposure period. Histopathological examination of comprehensive tissues was performed at the end of the treatment period and in all animals dying prior to study completion.

A total of 15 animals (0 in untreated control, 3 in vehicle control, 4 in 5 mg/kg, 3 in 50 mg/kg and 5 in 250 mg/kg groups) died during the treatment period; deaths were attributed to technical errors (ruptured esophagus) and not considered as treatment-related by study authors (U.S. EPA, 1989). Body weight and food consumption were similar to controls for all 2,4-DMP groups. Clinical signs of toxicity were not observed during the first 6 weeks of treatment. From week 7 through the end of the treatment period, squinting, lethargy, prostration and ataxia were observed in high-dose males and females following daily dosing. No treatment-related ophthalmologic findings were observed in any 2,4-DMP group.

At the interim sacrifice, small decreases in MCV (4.3% decrease, $p \leq 0.05$) and MCH (3.7% decrease, $p \leq 0.05$) were observed for females in the high-dose group compared to vehicle control, while larger decreases were observed in BUN levels for females in the mid- (32.5% decrease, $p \leq 0.05$) and high-dose (21.7% decrease, $p \leq 0.05$) groups (U.S. EPA, 1989). A significant increase in cholesterol levels (79% increase, $p \leq 0.05$) was observed for males in the low-dose group. No effects on other hematological or clinical chemistry parameters were observed at the interim sacrifice. At the end of the 90-day treatment period, all hematology parameters in 2,4-DMP treated mice were similar to control. Changes in clinical chemistry and organ weights observed after 90 days of treatment were sporadic, with no dose-related patterns of change. The organ weight data are shown in Table 5. No treatment-related gross pathological or histological findings, including lesions of the stomach, were observed. Based on clinical signs of toxicity in the high-dose 2,4-DMP group, NOAEL and LOAEL values were identified as 50 and 250 mg/kg-day.

According to IRIS (U.S. EPA, 2007), an unpublished 14-day gavage study with 2,4-DMP (U.S. EPA, 1987) was conducted by the same laboratory as the 90-day gavage study in mice (U.S. EPA, 1989). Results of the 14-day study revealed signs of toxicity (lethargy, prostration and ataxia) in males and females exposed to 250 mg/kg-day, the same dose at which signs of toxicity effects were observed in the 90-day study. No additional information pertaining to this study was provided by IRIS (U.S. EPA, 2007). This study was not available for review.

Inhalation Exposure

No studies investigating the effects of subchronic or chronic inhalation exposure to 2,4-DMP in animals were identified.

Table 5. Effect of Oral Treatment of Mice with 2,4-DMP (90-Day Exposure) on Final Body Weight and Relative Organ Weights (U.S. EPA, 1989)					
Parameter	Treatment Group (mg/kg-day)				
	Control	Vehicle Control	5	50	250
Females					
Body weight (g)	27.2±3.9 ^a	26.6±2.4	26.4±2.8	26.6±3.1	26.1±1.6
Liver weight (g)	1.1967	1.1001	1.1136	1.1285	1.1425
Relative liver weight(g/100 g)	4.4289	4.1072	4.1953	4.2614	4.3733
Spleen weight (g)	0.0959	0.0846	0.0854	0.0796	0.0898
Relative spleen weight (g/kg)	0.3500	0.1607	0.1639	0.1518	0.1703
Adrenal weight (g)	0.0124	0.0108	0.0142 ^b	0.0115	0.0110
Relative adrenal weight (g/100 g)	0.0467	0.0403	0.0548 ^b	0.0437	0.0424
Males					
Body weight (g)	33.9±3.3	32.5±2.9	33.3±2.7	32.5±3.3	31.6±3.5
Liver weight (g)	1.4430	1.2658	1.3412	1.2985	1.3166 ^b
Relative liver weight(g/100 g)	4.2744	3.9064	4.0421	3.9955	4.1695 ^b
Spleen weight (g)	0.1081	0.0758	0.0907 ^b	0.0742	0.0747
Relative spleen weight (g/kg)	0.3272	0.2337	0.2740	0.2282	0.2368
Adrenal weight (g)	0.0093	0.0099	0.0108	0.0083	0.0082
Relative adrenal weight (g/100 g)	0.0275	0.0303	0.0324	0.0256	0.0263
^a Values are means ± SD, or means only					
^b Significantly different from vehicle control (p≤0.05)					

Other Studies

Dermal- The immunomodulatory effects of 2,4-DMP were examined in six- to eight-week old male BALB/cA mice following short-term (3-day) dermal exposure (Yamano et al., 2007). Groups of mice (n = 3/group) were exposed to 25 µL of 1M 2,4-DMP (equivalent to 100 mg/kg) or vehicle (acetone/olive oil, 4:1) through application to the dorsum of both ears for 3 consecutive days. Three or five days after the last exposure to 2,4-DMP, auricular lymph nodes (LN) were excised from each mouse and prepared for evaluation using the murine local lymph node assay (LLNA), or were processed for primary cell culture and subsequent cytokine profiling, respectively. The LLNA allows for determination of whether a chemical acts as an immediate or delayed type immunogen which is related to the relative proportions of or balance between type-1 and type-2 T helper (Th-1 and Th-2, respectively) cells. Th-1 and Th-2 cells are differentiated by the types of cytokines produced in response to an immunogen. Th-1 cells secrete pro-inflammatory cytokines such as interferon-γ (IFN-γ), whereas Th-2 cells secrete anti-inflammatory cytokines such as interleukin-4 (IL-4). Thus, these two subsets of T helper cells are in essence functional antagonists of one another. In addition to the LLNA, primary splenocyte cultures from immunologically naïve mice were used for *in vitro* analysis of cell viability and cytokine profiling following 48 hr. of 2,4-DMP exposure. LLNA data suggested

that 2,4-DMP caused lymph node proliferation by acting as an immunogen via the dermal route of exposure. However, 2,4-DMP failed to stimulate lymphocyte secretion of the pro-inflammatory cytokine IFN- γ , or inhibit the anti-inflammatory cytokine IL-4 compared to control. Thus, it appears that 2,4-DMP is a weak inducer of a type-1 reaction in T helper cells (i.e. Th-1) following dermal absorption. Specifically, the results suggest that while dermal 2,4-DMP exposure induces an apparent increase in lymphocyte population of auricular nodes, the immunomodulatory effect (i.e. the ability to tip the balance between a Th-1 or Th-2 type immune response) was not significantly different from vehicle treated controls.

Toxicokinetic – Little information is available regarding the toxicokinetics of 2,4-DMP. In general, dimethylphenol isomers undergo extensive absorption from the gastrointestinal tract (Miyamoto et al., 1969). Results of a kinetic study in male Sprague-Dawley rats indicate that intravenously administered 2,4-DMP undergoes rapid distribution, with accumulation in the brain, liver and fat (Kaka et al., 1982). Metabolism to glucuronide and sulfate conjugates was rapid and nearly complete within 30 minutes of administration (Kaka et al., 1982).

Genotoxicity – All available evidence indicates that 2,4-DMP, like the other dimethylphenol isomers, is not genotoxic. All dimethylphenol isomers tested negative in reverse mutation assays with *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 with and without activation (Pool and Lin, 1982; Florin et al., 1980; Mortelmans et al., 1986). In a reverse mutation assay with *Escherichia coli* strain Sd 4-72, 2,4-DMP tested negative (Szybalski et al., 1958). 2,4-DMP also tested negative in a sister-chromatid exchange assay in isolated human lymphocytes (Jansson et al., 1986).

Tumor Promoting Activity – Although no subchronic or chronic oral or inhalation carcinogenicity studies have been performed on dimethylphenol isomers, data are available to suggest that the 2,4-, 2,5-, 3,4- and 3,5-DMP isomers exhibit tumor promoting activity on mouse skin (Boutwell and Bosch, 1959). All isomers except 2,6-DMP produced a small increase in carcinoma incidence when applied to skin without an initiation. However, the available data are not sufficient to assess the carcinogenicity of 2,4-DMP or other dimethylphenol isomers.

DERIVATION OF A PROVISIONAL SUBCHRONIC ORAL RfD FOR 2,4-DIMETHYLPHENOL

No studies on the effects of oral exposure to 2,4-DMP in humans are available. Subchronic toxicity studies in rats and mice identify the stomach and thymus as possible target organs for oral 2,4-DMP. The 10-day oral toxicity study in rats showed dose-related irritant and corrosive effects of the esophagus and forestomach following administration of 2,4-DMP by gavage (Daniel et al., 1993). Although the study report clearly indicates that histopathological changes to the forestomach were observed in rats treated with 600 and 1200 mg/kg-day, due to inadequate reporting, it is unclear if effects on the forestomach were present at lower doses (60 and 120 mg/kg-day). Irritant and corrosive effects of the esophagus and forestomach were observed in rats exposed to 180 and 540 mg/kg-day for 90 days; however, results of histopathological examination of the forestomach in the 60 mg/kg-day were not reported. Although 2,4-DMP clearly produces adverse effects to the esophagus and forestomach of rats

following administration by gavage, the available dose-response information is not adequate for the basis of the subchronic p-RfD.

Decrease in relative thymus weight was observed in male, but not female, rats treated with 60 mg/kg-day for 90 days, although the magnitude of change was small (Daniel et al., 1993). Gross pathological examination revealed a small, red thymus in a “small percentage” of surviving males in the 60 and 540 mg/kg groups, but not in the 180 mg/kg group. Thus, a clear dose-response relationship was not observed. No information was reported on histopathological examination of the thymus. Since thymus and immune system function of rats was not assessed, the biological significance of decreased thymus weight and small, red thymus is uncertain. Therefore, the effect of 2,4-DMP on thymus weight was not selected as the basis for the subchronic p-RfD.

The 90-day gavage study in mice reported general signs of toxicity, including squinting, lethargy, prostration and ataxia in males and females following daily dosing with 250 mg/kg-day, establishing a NOAEL of 50 mg/kg-day (U.S. EPA, 1989). Although signs of clinical toxicity are not very sensitive endpoints, comprehensive toxicological endpoints were examined, including histopathology, and the study was well-reported. Thus, the NOAEL of 50 mg/kg-day for signs of toxicity was selected as the basis of the subchronic p-RfD. As indicated in the Introduction section of this document, this is the same study and critical effect used to derive the chronic RfD for 2,4-DMP listed by IRIS (U.S. EPA, 2007).

The **subchronic p-RfD of 5E-2 mg/kg-day** was derived from the NOAEL of 50 mg/kg-day for signs of clinical toxicity as follows:

$$\begin{aligned}
 \text{p-RfD} &= \text{NOAEL} \div \text{UF} \\
 &= 50 \text{ mg/kg-day} \div 1000 \\
 &= 0.05 \text{ mg/kg-day or } 50 \text{ } \mu\text{g/kg-day} \\
 &= \mathbf{5E-2 \text{ mg/kg-day}}
 \end{aligned}$$

The uncertainty factor (UF) of 1000 was composed of the following:

- An UF of 10 was applied for interspecies extrapolation to account for potential pharmacodynamic and pharmacokinetic differences between mice and humans.
- A default 10-fold UF for intraspecies differences was used to account for potentially susceptible individuals in the absence of quantitative information or information on the variability of response in humans.
- An UF of 10 was included for database insufficiencies due to the lack of oral developmental studies and a multi-generation reproduction study.

Confidence in the principle study is medium, since it examined appropriate and comprehensive endpoints and identified both LOAEL and NOAEL values. The database for oral exposure to 2,4-DMP includes only two subchronic gavage studies conducted in rats and mice, with different effects in each species. Furthermore, the database provides no information on developmental and reproductive studies. Low confidence in the database and the oral subchronic p-RfD results.

FEASIBILITY FOR DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION p-RfC VALUES FOR 2,4-DIMETHYLPHENOL

No studies investigating the effects of subchronic or chronic inhalation exposure to 2,4-DMP in humans or animals were identified. The lack of subchronic and chronic inhalation data precludes derivation of subchronic and chronic p-RfCs for 2,4-DMP.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2,4-DIMETHYLPHENOL

No studies evaluating the carcinogenic potential of oral or inhalation exposure to 2,4-DMP in humans were identified in the available literature. Cancer bioassays for 2,4-DMP have not been conducted in animals for either oral or inhalation exposure. Under the 2005 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), inadequate information is available to assess the carcinogenic potential of 2,4-DMP.

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9-25-2007

Provisional Peer Reviewed Toxicity Values for
2,4-Dinitrophenol
(CASRN 51-28-5)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose

PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2,4-DINITROPHENOL (CASRN 51-28-5)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

The HEAST (U.S. EPA, 1997) listed a chronic RfD of 2E-3 mg/kg-day for 2,4-dinitrophenol (DNP) based on the IRIS database. The assessments were based on a LOAEL of 2 mg/kg-day for cataract in humans treated with 2,4-DNP as a weight reducing agent (Horner, 1942). The derivation for the chronic RfD included an uncertainty factor (UF) of 1000 (10 for extrapolation from subchronic exposure to chronic exposure, 10 for protecting sensitive individuals, and 10 for the use of a LOAEL). The HEAST also lists a subchronic RfD by adopting the chronic oral RfD from IRIS. The chronic RfD for 2,4-DNP was developed by U.S. EPA in 1991, and is listed on the IRIS database. However, it was not listed on the Drinking Water Standards and Health Advisories list (U.S. EPA, 2006). The toxicity of 2,4-DNP has been summarized in the Ambient Water Quality Criteria for Nitrophenols by U.S. EPA (1980) and the Toxicological Profile for Dinitrophenol by ATSDR (1995). ATSDR proposed an acute-duration Minimal Risk Level (MRL) of 0.01 mg/kg-day, but not for intermediate- or chronic-duration oral exposure to 2,4-DNP.

An RfC for 2,4-DNP was not listed on the HEAST (U.S. EPA, 1997). The health effect data for 2,4-DNP were reviewed by the U.S. EPA RfD/RfC Work Group in 1991 and were determined to be inadequate for the derivation of an inhalation RfC. The American Conference of Governmental Industrial Hygienists has not established a Threshold Limit Value (TLV)-Time-Weighted Average (TWA) and the National Institute for Occupational Safety and Health (NIOSH, 2005) has not established a REL-TWA for this chemical. The Occupational Safety and Health Administration (OSHA) has not developed a Permissible Exposure Limit (PEL)-TWA for 2,4-DNP either.

The 1997 HEAST did not include a cancer assessment for 2,4-DNP. No cancer assessment for this chemical has been developed by IRIS or IARC.

The WHO (2007) has not reviewed the toxicology of 2,4-DNP. Literature searches were conducted for the period from 1980 to August 2007 to identify data relevant for the derivation of provisional RfD, RfC, and cancer assessments for 2,4-DNP. The following databases were searched: TOXLINE, MEDLINE, CANCERLIT, TOXLIT/BIOSIS, Registry of Toxic Effects of Chemical Substances (RTECS), HSDB, GENETOX, CCRIS, TSCATS, EMIC/EMICBACK, and DART/ETICBACK.

This document has passed the Superfund Health Risk Technical Support Center (STSC) quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

REVIEW OF PERTINENT LITERATURE

Human Studies

Numerous occasions of human poisoning by 2,4-DNP have been reported in the literature. The earliest cases of fatal 2,4-DNP intoxication related to its usage as a component of explosives during World War I. Thirty-six cases of fatal occupational dinitrophenol poisoning occurred among employees of the munitions industry in France between 1916 and 1918 (Perkins, 1919). A literature review by von Oettingen (1949) revealed 27 reported cases of fatal occupational dinitrophenol poisoning in the United States for the years 1914 to 1916. In addition, Gisclard and Woodward (1946) reported two fatal cases of dinitrophenol poisoning during manufacture of picric acid where 2,4-DNP was produced as an intermediate. Swamy (1953) also described a case of suicidal poisoning by 2,4-DNP.

Early in the 1930s, 2,4-DNP was widely recommended as a treatment for obesity, and it resulted in both toxic side effects and fatalities. Horner (1942) reported a total of nine deaths resulting from the use of dinitrophenol as a slimming agent. The toxic manifestations of dinitrophenol exposure as reviewed by Horner (1942) included subacute symptoms such as gastrointestinal disturbances (nausea, vomiting, colic, diarrhea, anorexia), profuse sweating, weakness, dizziness, headache, and loss of weight. Acute poisoning has resulted in the sudden onset of pallor, burning thirst, agitation, dyspnea, profuse sweating, and hyperpyrexia. Intense and rapid onset of rigor mortis after death has also been described.

Perkins (1919) reported that postmortem examination of dinitrophenol victims demonstrated no characteristic lesions. Acute edema of the lungs was mentioned but was believed to be secondary to the toxic effects on the vasomotor system. Microscopic lesions of the liver and kidney cells were inconstant and typical changes were lacking elsewhere.

The widespread use of 2,4-DNP as a weight reducing agent in humans during the 1930s also provided some information regarding the chronic effects of this compound in humans. Recommended therapeutic doses of 2,4-DNP for weight control on humans ranged from 2 to 5 mg/kg-day (Dunlop, 1934; Horner, 1942; Tainter et al., 1933). Tainter et al. (1933) administered 2,4-DNP (average daily dose of 0.3 g) to 113 obese patients for as long as four months without demonstrating evidence of cumulative or toxic effects. Based on an assumption of body weight

of 70 kg, the corresponding average daily dose was 4.3 mg/kg-day. The most important side effect noted by the investigators was a skin rash observed in about 7% of the patients treated. The rash was manifested usually after a one-day period of mild itching and consisted of a maculopapular or urticarial type of rash. The itching was intense and in some cases there was considerable swelling. Symptoms subsided in 2 to 5 days following withdrawal from the drug. The next most important side effect noted by the authors was a loss of taste for salt and sweets observed in 5.3% of the patients. This effect also subsided following withdrawal from 2,4-DNP. The investigators failed to detect any effect of 2,4-DNP on liver or kidney function, pulse, blood pressure, or oxygen capacity of the blood. No cases of anemia, agranulocytosis, or malignant neutropenia appeared. Three cases of mild gastrointestinal upset were reported, however.

In a later publication, Horner (1942) reviewed the acute and chronic toxicity of use of 2,4-DNP (including cataract formation) resulting from therapeutic use of the compound. Gastrointestinal symptoms consisting of nausea, vomiting, and loss of appetite were common as a result of 2,4-DNP administration. Cutaneous lesions were the most frequent side effect with an incidence of 8 to 23%. Although the majority of lesions were mild, others were severe. Bone marrow effects of dinitrophenol have also been reported. Eight cases of agranulocytosis were reported, with three fatalities. Thirty cases of neuritis including aberrations of taste and multiple regional involvement, particularly affecting the feet and legs, were recorded. Symptoms appeared after an average of ten weeks, followed ordinary therapeutic doses and persisted for weeks or months. Electrocardiographic evidence of functional heart damage was offered by several investigators and fragmentation of the heart muscle was reported at autopsy in one fatal case. It was generally agreed that 2,4-DNP was rarely injurious to the liver and kidneys when administered in therapeutic doses.

The development of cataracts following dinitrophenol therapy was first described by Horner et al. (1936). Later, over 100 cases of cataract formation following dinitrophenol therapy were reviewed by Horner (1942). Horner described the following characteristic features of 2,4-DNP induced cataracts: (1) they occurred in young women who were physically normal except suffering varying degrees of obesity and were in an age group in which senile cataracts do not occur; (2) they were bilateral and appeared either during or after period of dinitrophenol treatment; (3) an interval of months or years might elapse between the time the last dose was taken and the onset of blurred vision; (4) lenticular changes were strikingly similar and could be demonstrated with the biomicroscope at a time when vision for distance and reading was still normal; (5) after gradual onset, the lenticular changes progressed with startling rapidity until the vision was obscured; (6) treatment was without effect in staying their progress; and (7) surgical removal of the lens was uniformly successful in restoring vision. Cataract formation appears to be the primary reason 2,4-DNP was withdrawn from medical use.

The length of time that 2,4-DNP was taken and the amount of the drug consumed varied widely among cataract victims. In 29 cases, the duration of treatment varied from 3 months to 24 months with an average of 11 months. Neither the length of treatment nor the total dose seemed to have any bearing on the occurrence of cataracts. Individual susceptibility appeared to be a more important factor. Horner (1942) estimated that the incidence of cataracts in patients who had taken dinitrophenol exceed one percent.

The available data do not allow the calculation of a minimum effect level for 2,4-DNP-induced cataract formation in humans. Cataractogenic activity in humans has been observed in a small proportion of patients receiving as little as 2 mg/kg-day. An assessment of the no-effect-level for cataract formation awaits further investigation. Such an assessment is further complicated by the fact that cataract formation in humans, following DNP administration, differs significantly from the situation seen in experimental animal studies.

The existing review documents (U.S. EPA, 1980, 1984; ATSDR, 1995) and an updated literature search did not identify relevant studies regarding the carcinogenicity of 2,4-dinitrophenol in humans following oral or inhalation exposure.

Animal Studies

Short Term Animal Studies

Attempts to find a suitable animal to study cataract development in humans exposed to 2,4-DNP have generally been unsuccessful. Normal mammalian animals have not developed cataracts after oral exposure to 2,4-DNP, although cataracts could be induced in a special strain of mouse (yellow adipose), in vitamin C-deficient guinea pigs, in ducks, and in chickens (ATSDR 1995). Formation of cataracts by acute exposure to DNP was first demonstrated in animals almost 10 years after the problem was known to exist in humans (Gehring and Buerge, 1969a; Ogino and Yasukura, 1957; Feldman et al., 1959, 1960; Bettman, 1946). Experimental cataracts, first produced in ducks and chickens, differ from DNP-induced human cataracts in that they can be formed in acute exposures and may appear in less than one hour. Furthermore, these lesions will disappear spontaneously in animals within 25 hours (Howard et al., 1976). Hence, the usefulness of data on the formation of cataracts in experimental animals following DNP administration in assessing human hazard to dinitrophenol is questionable.

Langerspectz and Tarkkonen (1961) failed to detect histological changes in the adrenals or the liver during 30 day treatment of Swiss albino male mice with twice daily doses of 10 mg of 2,4-DNP/kg (20 mg/kg-day) via the subcutaneous injection.

Subchronic Animal Studies

Tainter and Cutting (1933) administered 2,4-DNP to dogs at intervals of three or more days over a period of 2 to 3 months. Abnormal liver and kidney pathology were not detected but an effect on spleen tissue was noted. Over large areas of the material containing “numerous large faintly staining cells with vesicular polyhedral nuclei.” This study is limited due to the lack of dose information in the summary document (U.S. EPA, 1980).

Groups of three male dogs received daily oral dose of 0, 5 or 10 mg/kg 2,4-DNP in capsules for 6 day/week for 27 weeks (Tainter et al., 1934). There were no important changes in body weight as a result of the continuous administration of 2,4-DNP. Estimations at intervals of three weeks of the amount of sugar, and albumin in the urine and of the hemoglobin and red, white and differential blood cell counts, urea content, icteric index and oxygen capacity of the

blood and fragility of the red cells showed no significant or consistent deviations from the normal or control values. At the end of treatment, the dogs were killed for complete necropsy and histological study of the tissues. No significant pathologic changes were noticed grossly or microscopically. Thus, the highest dose of 10 mg/kg-day (equivalent to continuous dose of 8.6 mg/kg-day) is considered a free-standing NOAEL.

Spencer et al. (1948) studied the subchronic toxicity of 2,4-DNP in rats. Male rats (10-20/dose) were fed diets containing 0, 0.01, 0.02, 0.05, 0.1, or 0.2 g of 2,4-DNP per 100g of food. Rats were maintained on diets containing 2,4-DNP for six months and both hematological and pathological investigations on surviving animals were performed. Based on rat food intake in a subchronic study (U.S. EPA, 1988), the average daily food intake factor for male rat of unknown species is assumed to be 0.091 kg food/kg body weight/day. Thus, the estimated doses were 0, 9.1, 18, 46, 91, and 182 mg/kg-day, respectively. Hematological examination included erythrocyte counts, hemoglobin concentrations, leukocyte counts, differential counts, and bone marrow counts at autopsy. Both gross and microscopic examination of liver, kidney, spleen, lung, heart, adrenal, pancreas, and stomach tissues were also performed. Rats maintained on diets containing 0.02% 2,4-DNP (18 mg/kg-day) grew at a normal rate and the investigators failed to detect discernible ill effects of pathological changes at autopsy. Similarly, pathological changes were not found upon microscopic examination of tissues from rats receiving diets containing 0.05% 2,4-DNP (46 mg/kg-day) although growth of these rats fell 5 to 10% below that of the controls throughout the six-month experimental period. At autopsy the only changes observed in these animals were a very slight depletion of body fat and a very slight increase in the average weight of the kidneys. More reduced growth was also seen in the rats treated with 0.1% of 2,4-DNP (91 mg/kg-day). At the highest dose of 2,4-DNP in their diets (182 mg/kg-day) rats occasionally died and survivors lost weight rapidly. Examination of surviving animals revealed marked emaciation, an empty gastrointestinal tract, a slightly enlarged and dark spleen, and undersized testes. Microscopic examination showed slight congestion and cloudy swelling of the liver, very slight parenchymatous degeneration of the epithelium of the renal tubules, slight congestion and hemosiderosis of the spleen and testicular atrophy. No significant pathological changes were observed in the lung, heart, adrenals, pancreas, or stomach of these animals. Based on these observations, a NOAEL for 2,4-DNP in rats was 18 mg/kg-day.

Chronic Animal Studies

Groups of 5-6 white rats (sex unknown) received 2,4-DNP in the food beginning shortly after weaning when they weighed about 30 g, and continuing until death (Tainter, 1938). The treatment doses included 0, 0.001, 0.005, 0.01, 0.02, 0.04, 0.06, 0.08, 0.12 and 0.24% of 2,4-DNP in the diet. Based on rat food intake in a chronic study (U.S. EPA, 1988), the average daily food intake factor for rat of unknown sex and species is assumed to be 0.078 kg food/kg body weight. Thus, the estimated doses were 0, 0.78, 3.9, 7.8, 16, 31, 47, 62, 94 and 187 mg/kg-day, respectively. The food intakes, growth curves, final weights, and life spans were compared with those of untreated controls. At the time of death, necropsies and histological studies of the tissues were made. The rats were observed closely throughout the entire duration of the experiment. The food intakes were similar for all the groups. Doses of 2,4-DNP in the diet ranging from 0.78 to 31 mg/kg-day did not appreciably modify the growth curves or final weights. Doses of 47 to 94 mg/kg-day decreased the rate of growth, and diminished the final

average weight about 75 g. The average duration of life of about two years was not decreased by doses of 2,4-DNP up to 62 mg/kg-day; a dose of 94 mg/kg-day decreased it by about one-half and 187 mg/kg-day killed the rats in about one month. There was no evidence of any toxic effects of 2,4-DNP on the eyes of these rats, as indicated by direct observations, and ophthalmoscopic study or slit lamp microscopy. At necropsy, and histologically, the tissues of the treated rats were indistinguishable from those of the untreated controls, there being no lesions which could be ascribed to the action of the 2,4-DNP. The NOAEL in rats was identified to be 31 mg/kg-day based on significant decreases in body weight.

Reproductive and Developmental Animal Studies

Based on the available data it appears unlikely that the 2,4-DNP pose a teratogenic hazard to humans. Gibson (1973) examined developmental toxicity of 2,4-DNP in mice. Groups of pregnant mice (7-8 animals/dose) received intraperitoneal (7.7 or 13.6 mg/kg) or oral (25.5 or 38.3 mg/kg) administration of 2,4-DNP during early organogenesis (gestation day 10-12). Nine pregnant females received water served as control. Caesarean section was performed on day 19 of gestation, and the number and position of live, dead and resorbed fetuses was examined. Individual fetuses were weighted, and examined for external anomalies. Fetal crown-rump distance was measured for each fetus. Each litter was divided into two sub-groups for further examination for soft-tissue or skeletal anomalies. Very limited results were provided in the original report. Among the all the endpoints (including number of implantations, resorptions, fetal body weight and fetal crown-rump length) presented in a summary table (Table 8 in the original paper), increased resorptions (mean response/litter), decreased fetal body weight, and fetal crown-rump length occurred in almost all the treated groups (including both i.p. and oral treatments); however, only the decreases in the fetal body weight and crown-rump length in the high i.p. dose group (13.6mg/kg-day) was statistically significant. No other details were provided in the original report. Gibson (1973) ambiguously concluded that dinitrophenol does not produce morphological defects in the offspring, but embryo toxicity occurs at the higher dose levels. The higher doses also produced overt toxic signs (hyperexcitability and hyperthermia) in the dams, but were not lethal. However, it is not clear whether the author referred the higher doses to i.p. high dose of 13.6 mg/kg or to doses ≥ 13.6 mg/kg-day including i.p. high dose and two oral doses (25.5 and 38.3 mg/kg-day). Based on limited information available from the original report, the low i.p. dose of 7.7 mg/kg-day was considered NOAEL.

The toxicity of 2,4-DNP was examined in newborn rats by Koizumi et al. (2001). Groups of Sprague-Dawley rats (6/sex/dose) were administered 2,4-DNP at 0, 3, 10 or 20 mg/kg-day by gastric intubation daily from days 4 to 21 after birth, and killed after overnight starvation following the last treatment. Recovery-maintenance groups at the same dosages were maintained for 9 weeks without chemical treatment and fully examined at 12 weeks old. General behavior was observed daily, and body weight and food consumption were measured more than once a week. At treatment day 17 or 18, papillary reflex, corneal reflex, surface righting, mid-air righting and auricular reflexes were examined as parameters of reflex ontogeny. Furthermore, fur appearance, incisor eruption and eye opening were noted in the lactating period as evidence of physical development, and testes descent and vaginal opening during the early recovery-maintenance period for assessment of sexual maturation. Color, pH, occult blood, protein, glucose, ketone bodies, bilirubin, urobilinogen, urine sediment and volume of the urine were

examined only at the end of the recovery-maintenance period. The blood samples were analyzed for complete hematological parameters as well as blood biochemistry. All the organs were weighted and examined for gross and histopathological changes at the end of treatment. No clinical signs or deaths were encountered. The body weights at 20 mg/kg-day were significantly below control values from dosing day 7 in the males and dosing day 10 in females in the scheduled-sacrifice group. There was also statistically significant lowering of body weight in the 20 mg/kg-day males for the first quarter of the recovery-maintenance period, but not in females. No definitive changes in abdominal fur appearance, incisor eruption, eye opening and testis descent or vaginal opening as well as reflex ontogeny parameters were detected in any dose groups. There were significant changes in absolute weights of testes at ≥ 10 mg/kg-day, and changes in absolute and relative organ weights in several other organs at 20 mg/kg-day. No chemical-related histopathological changes were noted in either scheduled-sacrifice or recovery-maintenance groups. Significant increase in RBC was observed in females receiving 20 mg/kg-day after the treatment but not after the recovery-maintenance period. Although increases in serum glutamate oxaloacetate transaminase (GOT) in males and total bilirubin in females were detected at 10 and 20 mg/kg-day after treatment, those were not considered to be chemical-induced because they were very slight and there was no dose-relationship. The authors considered the dose of 10 mg/kg-day as the NOAEL at which only the lowering of absolute testis weight was observed.

The toxicity of 2,4-DNP was also examined in young rats by Koizumi et al. (2001). Groups of 5 to 6-week old Sprague-Dawley rats (6/sex/dose) were administered 2,4-DNP at 0, 3, 10, 30 or 80 mg/kg-day by gastric intubation daily for 28 days, and killed after overnight starvation following the last treatment. Recovery-maintenance groups at the 0, 30 or 80 mg/kg-day were maintained for 2 weeks without chemical treatment and fully examined at 11-12 weeks of age. Rats were examined for general behavior, body weight, food consumption, urinalysis, hematology and blood biochemistry, necropsy finding, organ weights and histopathological finding. Clear toxic signs, such as decrease in locomotor activity, prone position, ptosis, panting, crawling position and salivation, were observed repeatedly during the dosing period at 80 mg/kg-day in both sexes, and two males and six females died in the same dose group. However, decrease in locomotor activity and salivation in the 30 mg/kg-day group were mostly observed only after the first dosing. The relative liver weights were increased in both sexes of the 80 mg/kg-day scheduled-sacrifice group, and this persisted through the recovery period. Relative organ weights for brain, kidneys and testes were increased only in 80 mg/kg-day males. On histopathological examination, mineralization of the corticomedullary junction in kidneys was observed in both sexes at 80 mg/kg-day in the scheduled-sacrifice and recovery groups, but the change was only statistically significant in males of the scheduled-sacrifice group. On hematological examination, increase in hemoglobin and hematocrit in the recovery period were observed, limited to 80 mg/kg-day males. Although blood chlorine levels were slightly decreased in 30 and 80 mg/kg-day males and total bilirubin was slightly increased in females receiving 10 mg/kg and more, no changes in histopathology or organ weights were observed at 30 mg/kg-day or lower. Prior to this experiment, a dose-finding study in the same age group (4/sex/group) at doses of 0, 0.6, 2, 6, 20 or 60 mg/kg-day had been conducted. The study results from these animals after treatment for 14 days were consistent with the main study. Thus, the authors considered 20 mg/kg-day from the dose-finding study as the NOAEL based on decrease in locomotor activity and salivation at 30 mg/kg-day.

Wulff et al. (1935) examined the effects of 2,4-DNP on the fertility, gestation, and fetal life of rats in an one-generation study. A group of 20 female rats (unknown strain) received 20 mg/kg 2,4-DNP 8 days prior the introduction of males. Nine females received no treatment and five females received 1% sodium bicarbonate solvent served as control. Dinitrophenol was administered intragastrically twice daily throughout cohabitation, and gestation until the respective litters were weaned. The daily average dose was estimated to be 40 mg/kg-day. The average number born in each litter was not affected by dinitrophenol treatment, and the treatment did not appreciably affect the body weight gains of mothers during pregnancy. Neonatal malformations were not detected. Among 2,4-DNP treated rats, however, 25% of the total number of pups were stillborn while only 6.8% of the pups were stillborn in the control group (two groups combined). In addition, the mortality during the nursing period of viable pups born to mothers administering 2,4-DNP was 30.9% as compared with 13.4% for young of control mothers. Two possible explanations for this latter phenomenon were offered by the authors: treated mothers neglected their pups while in a febrile state, and only the more vigorous of the offspring manage to reach the mother for nursing; or, a toxic agent was passed to the young through the milk. Data to distinguish between the two possibilities are not available. Based on developmental toxicity (stillbirth and mortality during lactation), this study provided a free standing LOAEL of 40 mg/kg-day.

Other Studies

Bowman (1967) has studied the effect of 2,4-DNP on the developing chick embryo *in vitro*. At 2,4-DNP concentrations of 18 mg/L or 370 mg/L a syndrome of abnormalities resulted consisting of degeneration and sometimes complete absence of neural tissue accompanied by a reduction in the number of somites. The 2,4-DNP concentrations used in this study are extremely high and the relevance of the experimental findings to the *in vivo* situation in mammals is not clear.

Genotoxicity data for 2,4-DNP were reviewed by ATSDR (1995). Test results were negative for 2,4-DNP in multiple assays for reverse mutation in *Salmonella typhimurium*, with or without metabolic activation. However, the two major metabolites of 2,4-DNP are mutagenic in *S. typhimurium* (and other systems), suggesting that the negative results for 2,4-DNP may indicate failure of the S9 activating system used in these assays to metabolize this chemical. Mixed results were reported for 2,4-DNP in studies of reverse mutation in *Escherichia coli*. There is little evidence that 2,4-DNP produces DNA damage. Assays for phase induction in *E. coli*, SOS response in *S. typhimurium*, unscheduled DNA synthesis in rat hepatocytes, and DNA damage (alkali elution) in Chinese hamster V79 cells were negative. DNA damage (alkali elution) was reported in mouse leukemia L1210 cells and human HeLa cells, but was associated with depletion of ATP. Depletion of ATP was also observed in studies showing decreases in DNA synthesis and mitotic index after exposure to 2,4-DNP. Therefore, positive findings in these studies probably reflects cytotoxicity (decreased cellular metabolic rate), rather than genotoxicity. *In vivo*, 2,4-DNP produced chromosomal aberrations in bone marrow cells of mice treated by intraperitoneal injection.

In a study designed to measure tumor promoting activity, Boutwell and Bosch (1959) examined the ability of 2,4-DNP to promote tumor formation following a single initiating dose

of dimethylbenzanthracene. The 2,4-DNP failed to promote skin tumors in mice in this experiment. In a similar experiment, Stenback and Garcia (1975) also examined the ability of 2,4-DNP to promote skin tumor formation in mice, and found no tumor promoting activity.

The existing review documents (U.S. EPA, 1984; ATSDR, 1995) and an updated literature search did not identify relevant studies regarding the carcinogenicity of 2,4-dinitrophenol in animals following oral exposure.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC ORAL RfD VALUES FOR 2,4-DINITROPHENOL

2,4-DNP is considered a classic uncoupler of oxidative phosphorylation and is widely used by biochemists to determine whether a given biochemical process is energy dependent. The toxic action of the dinitrophenol is generally attributed to their ability to uncouple oxidative phosphorylation. It prevents the utilization of the energy provided by cellular respiration and glycolysis by inhibiting the formation of high energy phosphate bonds. All energy dependent biochemical processes are therefore affected by the action of the compounds. The large number of clinical effects attributed to dinitrophenol toxicity result essentially from the shortcircuiting of metabolism in cells which absorb sufficient dinitrophenol. At concentrations higher than those necessary to uncouple oxidative phosphorylation, a number of inhibitory effects of the dinitrophenol isomers on certain enzymatic reactions may occur. The dinitrophenol may also act directly on the cell membrane, thus causing toxic effects on cells which do not depend on oxidative phosphorylation for their energy requirements. More detailed information on the mechanism of toxicity has been summarized by U.S. EPA (1980).

The database for 2,4-DNP toxicity is relatively comprehensive, and it covers human case studies and results from experimental exposure. In addition, the database also includes animals studies ranging from short-term studies, subchronic studies in dogs and rats, to a chronic study in rats, accompanied by developmental studies in mice and young rats, and an one-generation study in rats.

The toxicity of 2,4-DNP in humans ranges from mortality due to high dose exposure to minor effects such as gastrointestinal symptoms and cutaneous lesions due to therapeutic use of the compound as a weight reducing agent. The development of cataracts following the dinitrophenol therapy was first described by Horner et al. (1936), and similar response was also reported from treated animals. The length of time that 2,4-DNP was taken and the amount of the drug consumed varied widely among cataract victims. In 29 cases, the duration of treatment with the compound varied from 3 months to 24 months. Neither the length of treatment nor the total dose seemed to have any bearing on the occurrence of cataracts. The available data do not allow the calculation of a minimum effect level for 2,4-DNP-induced cataract formation in humans. Since cataractogenic activity in humans has been observed in a small proportion of patients receiving as little as 2 mg/kg-day dose of 2,4-DNP, this dose is considered as a LOAEL for cataract in humans after subchronic exposure to the chemical.

The formation of cataracts by acute exposure to 2,4-DNP has been reported in ducks and chickens treated with the compound. However, the experimental cataracts in the animals differ from DNP-induced human cataracts in that they can be formed in acute exposures and may appear in less than one hour. Furthermore, these lesions will disappear spontaneously in animals within 25 hours.

Subchronic studies in dogs and rats did not identify a specific target organ for 2,4-DNP, and a free-standing NOAEL of 8.6 mg/kg-day in dogs and a NOAEL of 18 mg/kg-day in rats based on decreased growth rate were identified (Spencer et al., 1948; Tainter et al., 1938). Similar to the subchronic studies, the chronic study in rats (Tainter et al., 1938) identified a NOAEL of 31 mg/kg-day based on significant decreases in body weight. There was no evidence of any toxic effect on the eyes in the rats treated chronically with 2,4-DNP at dose levels up to 187 mg/kg-day. Since the subchronic rat study (Spencer et al., 1948) did not include a dose at 30 mg/kg-day range, the subchronic NOAEL of 18 mg/kg-day from that study is considered consistent with the chronic NOAEL of 31 mg/kg-day (Tainter et al., 1938) because the latter study included smaller dose spacing in the experiment which allowed identification of a NOAEL higher than that from the subchronic study.

The toxicity of 2,4-DNP in developmental studies did not demonstrate more sensitive responses than the systemic effects observed in the subchronic or chronic studies. 2,4-DNP does not produce morphological defects in the offspring, but it could produce embryo toxicity at dose level of ≥ 13.6 mg/kg-day, and the same doses also produced overt toxic signs (hyperexcitability and hyperthermia) in the dams (Gibson, 1973). The developmental study provided a NOAEL of 7.7 mg/kg-day based on i.p. treatment dose. Short-term treatment (7 days) with 2,4-DNP in newborn rats resulted in decreased testis weight and body weight at the dose of 20 mg/kg-day, and the next lower dose of 10 mg/kg-day was identified as the NOAEL (Koizumi et al., 2001). 2,4-DNP treatment (28 days) in young rats (5-6 weeks old) resulted in decreases in locomotor activity and salivation at the dose level of ≥ 30 mg/kg-day (Koizumi et al., 2001), and this study identified a NOAEL of 20 mg/kg-day in young rats. Comparison of the effective doses in newborn and young rats suggested a less sensitivity to 2,4-DNP in young rats than newborn rats.

The one-generation reproductive study (Wulff et al., 1935) showed that 2,4-DNP treatment at dose level of 40 mg/kg-day resulted in increased stillbirth and mortality during lactation. It is not clear whether the mortality during lactation was due to toxicity in dams or fetuses exposed to the compound through milk. Therefore, this dose (40 mg/kg-day) is considered a free-standing LOAEL.

Based on all the data available, the cataracts developed in humans after therapeutic use of the compound as a weight reducing agent is considered the critical effect, and the estimated minimal dose of 2 mg/kg-day causing this effect is considered as the point of departure in deriving a provisional subchronic RfD. Using this point of departure is further supported by the relative rich data from animal studies. A **provisional subchronic RfD of 2×10^{-2} mg/kg-day** for 2,4-DNP is derived by applying a composite uncertainty factor of 100 (10 for the use of a LOAEL, and 10 to protect sensitive individuals) to the point of departure of 2 mg/kg-day. Because the database for 2,4-DNP included subchronic studies, chronic studies, developmental studies and an one-generation reproductive study, it is unlikely to identify more sensitive

responses from additional animal studies. Therefore, an uncertainty factor of 1 is used as the database factor. Because the critical effect in humans was observed after subchronic treatment of the compound, no extra uncertainty factor for exposure duration is needed for deriving a provisional subchronic RfD.

$$\begin{aligned}\text{subchronic p-RfD} &= \text{POD} / \text{UF} \\ &= (\text{subchronic LOAEL}) / (100) \\ &= 2 \text{ mg/kg-day} / 100 \\ &= 0.02 \text{ or } 2 \times 10^{-2} \text{ mg/kg-day}\end{aligned}$$

The current chronic RfD of 2×10^{-3} mg/kg-day on IRIS (U.S. EPA, 1991) was based on the same critical effect and point of departure with an extra uncertainty factor of 10 to cover the extrapolation from subchronic to chronic duration. A chronic RfD of 2×10^{-3} mg/kg-day for 2,4-DNP was derived by applying to the human LOAEL of 2 mg/kg-day a composite uncertainty factor of 1000 (10 for the use of a LOAEL, 10 for extrapolation from subchronic to chronic duration, and 10 to protect sensitive individuals). The chronic RfD on IRIS is still valid.

$$\begin{aligned}\text{Chronic RfD} &= \text{POD} / \text{UF} \\ &= (\text{subchronic LOAEL}) / (1000) \\ &= 2 \text{ mg/kg-day} / 1000 \\ &= 0.002 \text{ or } 2 \times 10^{-3} \text{ mg/kg-day}\end{aligned}$$

Confidence in the principal study is low. Higher study confidence is precluded because the principal study only describes anecdotal data which provides limited formation in the dosing and exposure duration, minimal data reporting, and the lack of reliable data on no effect levels for the critical effect in critical study. Confidence in the database is high because the database for 2,4-DNP toxicity not only includes experimental studies in humans, but also covers relative comprehensive studies in animals including short-term studies, subchronic studies in multiple species, a chronic study, developmental studies in pregnant mice and young rats, as well as a one-generation study. Therefore, it is unlikely to identify other more sensitive responses from additional animal studies. Overall confidence in the subchronic p-RfD values is medium, as the strengths in the database, particularly the supportive data from comprehensive animal studies, somewhat outweigh the low confidence in the principal human study. A chronic p-RfD based on the same point of departure with an additional uncertainty factor to cover the extrapolation from subchronic to chronic duration. The overall confidence for the chronic p-RfD would be the same as the one for subchronic p-RfD. The using of the subchronic data as the point of departure for chronic p-RfD is appropriate because neither the length of treatment nor the total dose seems to have any bearing on the occurrence of critical effect.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfC VALUES FOR 2,4- DINITROPHENOL

No data were located for the subchronic or chronic inhalation toxicity of 2,4-DNP in humans or animals. Due to the lack of data, no provisional RfC was derived for 2,4-DNP.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2,4- DINITROPHENOL

Weight-of-Evidence Descriptor

No studies were located examining associations between cancer and exposure of humans to 2,4-DNP. Thus, there were inadequate human data to assess the carcinogenicity of 2,4-DNP.

Data examining the potential for 2,4-DNP to produce cancer in animals were restricted to several mouse skin tumor assays that found no DNP-induced increases in incidence of skin tumors. U.S. EPA guidelines (2005) indicated that, in order to classify the compound as not likely to be carcinogenic to humans, no increased incidence of neoplasms should be found in at least two well designed and well-conducted animal studies of adequate power and dose in different species. Thus, the available animal data for 2,4-DNP were not sufficient to classify them as providing no evidence of carcinogenicity. Additional well-conducted testing in other animal species with long-term exposure, preferably via oral and inhalation exposure, is necessary to provide reasonable assurance as to whether 2,4-DNP may or may not be carcinogenic in animals or humans.

Mixed results in genotoxicity of 2,4-DNP were reported in several short-term mutagenesis assays in bacteria, and in *in vitro* and *in vivo* mammalian systems. The majority of the *in vitro* genotoxicity studies showed negative responses with some exceptions in DNA damage in mouse leukemia cells and human HeLa cells, although the positive findings in these studies probably reflects cytotoxicity rather than genotoxicity. An *in vivo* study produced chromosomal aberrations in bone marrow cells.

Following U.S. EPA (2005) guidelines for compounds with inadequate human data and inadequate animal data, 2,4-DNP was classified as having *inadequate information to assess carcinogenic potential*.

Quantitative Estimates of Carcinogenic Risk for 2,4-DNP

Due to inadequate information to assess carcinogenic potential, a quantitative cancer risk estimate for neither an oral slope factor nor an inhalation unit risk could be derived for 2,4-DNP.

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Provisional Peer Reviewed Toxicity Values for

2,6-Dinitrotoluene
(CASRN 606-20-2)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose

MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
2,6 -DINITROTOLUENE (CASRN 606-20-2)**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

The HEAST (U.S. EPA, 1997) lists subchronic and chronic oral RfD values of 1E-2 mg/kg-day and 1E-3 mg/kg-day, respectively, for 2,6-dinitrotoluene based on a 13-week gavage study in dogs (Lee et al., 1976). The assessment was based on a NOAEL of 4 mg/kg-day for neurotoxicity, blood alterations, and liver and kidney histopathology. An uncertainty factor of 3000 (10 for animal to human extrapolation, 10 for intraspecies diversity, 10 for less-than-lifetime study, and 3 for limited database, including lack of developmental and reproductive studies) was applied (Dourson and Stara, 1983). The source document for this derivation was a Health Advisory for 2,4- and 2,6-Dinitrotoluene (U.S. EPA, 1992). The RfD is included in the Drinking Water Standards and Health Advisories list (U.S. EPA, 2002). There is no RfD on IRIS (U.S. EPA, 2003). The HEAST does not include an RfC assessment for 2,6-dinitrotoluene due to lack of data, and no RfC assessment for 2,6-dinitrotoluene is available on IRIS (U.S. EPA, 2003). The HEAST (U.S. EPA, 1997) indicates that a quantitative cancer assessment for a

mixture of 2,4- and 2,6-dinitrotoluene is available on IRIS (U.S. EPA, 2003). The source document for the cancer assessment was a Health and Environmental Effects Profile (HEEP) for Dinitrotoluene (U.S. EPA, 1986) that assigned the 2,4 and 2,6-dinitrotoluene mixture to weight of evidence group B2 (probable human carcinogen) and derived an oral slope factor of $6.8\text{E-}1$ (mg/kg-day)⁻¹. In addition to the HEEP previously mentioned, a HEA (U.S. EPA, 1987) for 2,4- and 2,6-dinitrotoluene is included in the CARA list (U.S. EPA, 1991b, 1994).

A Toxicological Profile for 2,4- and 2,6-dinitrotoluene is available from ATSDR. ATSDR (1998) derived an intermediate-duration oral MRL of 0.004 mg/kg-day for 2,6-dinitrotoluene based on hematological effects in the dog study by Lee et al. (1976). The MRL was derived by applying an uncertainty factor of 1000 to 4 mg/kg-day , which was considered by ATSDR to be a LOAEL for mild extramedullary hematopoiesis in the spleen. Occupational exposure limits are available for dinitrotoluene (mixed isomers) that include a TLV-TWA of 0.2 mg/m^3 recommended by ACGIH (2002), a 8-hour PEL-TWA of 1.5 mg/m^3 promulgated by OSHA (2002), and a REL of also 1.5 mg/m^3 established by NIOSH (2002). These values are intended to protect against cardiovascular and reproductive effects of the dinitrotoluene mixture. IARC (1996) placed 2,6-dinitrotoluene in weight of evidence group 2B (possibly carcinogenic to humans) based on sufficient evidence in animals and inadequate evidence in humans. NTP (2002) has not studied 2,6-dinitrotoluene. No Environmental Health Criteria Document is available for 2,6-dinitrotoluene (WHO, 2002). Literature searches were conducted from 1985 through 2004 for studies relevant to the derivation of provisional toxicity values for 2,6-dinitrotoluene. Databases searched included: TOXLINE, MEDLINE, CANCERLIT, TSCATS, RTECS, CCRIS, DART, EMIC/ EMICBACK, HSDB, and GENETOX.

REVIEW OF PERTINENT DATA

Human Studies

No data are available regarding humans exposed solely to 2,6-dinitrotoluene. Most of the information available regarding exposure to dinitrotoluenes in humans involves occupational exposure to 2,4-dinitrotoluene or to technical grade dinitrotoluene (TGDNT, approximately 76% 2,4-dinitrotoluene and 19% 2,6-dinitrotoluene). In occupational settings, the routes of exposure are primarily inhalation and dermal contact. Acute exposure to high amounts of dinitrotoluenes is expected to produce methemoglobinemia, a characteristic feature of exposure to nitroaromatic compounds in general. Methemoglobin decreases the oxygen carrying capacity of the blood. A small amount of methemoglobin is normally present in the blood (approximately 1%), but it is constantly being reduced by enzymes in the red cell. Methemoglobin can reach up to 25% without giving apparent symptoms; levels of 35-50% result in mild symptoms such as dyspnea upon exertion and headaches, and levels exceeding 70% are probably lethal.

The U.S. EPA (1986) summarized early data on subjects occupationally exposed mainly to 2,4-dinitrotoluene and stated that a number of signs and symptoms were reported, including headache, vertigo, fatigue, dizziness, weakness, nausea, vomiting, dyspnea, drowsiness, arthralgia, insomnia, tremor, paralysis, unconsciousness, chest pain, shortness of breath, palpitation, anorexia, weight loss, and an unpleasant metallic taste in the mouth. Medical examination of the workers showed impaired reflexes, nystagmus, tremors, pallor, cyanosis, anemia, leukocytosis, hypertension, dermatitis, leukopenia and hepatitis or jaundice.

Studies of men occupationally exposed to TGDNT at dinitrotoluene or toluene diamine plants showed no significant differences in sperm counts or morphology, follicle stimulating hormone levels, or fertility of the workers, or incidence of miscarriage in their wives (ATSDR, 1998). A retrospective cohort mortality study of munition workers who were exposed to either 2,4-dinitrotoluene or TGDNT at two different manufacturing plants reported significant increases in death rates due to ischemic heart disease and residual disease of the circulatory system (congestive heart failure, cardiac arrest, and arteriosclerosis) (Levine et al., 1986). Such increases occurred more than 15 years after employment started. Furthermore, there appeared to be an association between heart disease and intensity of exposure to dinitrotoluene. No significant increases in mortality from malignant neoplasms as a group or from particular cancers were noted in this study. An additional study conducted at one of these plants reported an excess of cancer of the biliary tract, liver, and gall-bladder in workers exposed to a mixture of 98% 2,4-dinitrotoluene and 2% 2,6-dinitrotoluene in comparison with both the United States population and an internal unexposed group (Stayner et al., 1993). However, this finding was based on only 6 cases, and an exposure duration-response analysis could not be done because few workers had more than five years of exposure to dinitrotoluene. A more recent study of miners from the former German Democratic Republic exposed to TGDNT found a significant increase in urothelial cancers confined predominantly to a high-exposure category group (Brüning et al., 1999).

Animal Studies

Characteristic signs of intoxication with dinitrotoluene in animals include central nervous system depression, respiratory depression, and ataxia (U.S. EPA, 1986). U.S. EPA (1986) and ATSDR (1998) list the following oral LD₅₀s for 2,6-dinitrotoluene from various studies: 535 and 795 mg/kg for male and female CD rats, respectively; 180 mg/kg for male Sprague-Dawley rats; 621 and 807 mg/kg for male and female CD mice, respectively; and, 1000 mg/kg for CF-1 mice.

Few longer-term studies with 2,6-dinitrotoluene were located in the available literature, and some of them were designed primarily to evaluate the carcinogenic potential of 2,6-dinitrotoluene. The oral RfD listed in the HEAST (U.S. EPA, 1997) is based on a study by Lee et al. (1976) in dogs. In that study, groups of beagle dogs (4/sex/dose level) were given 2,6-dinitrotoluene (>99% pure) in gelatin capsules at doses of 0, 4, 20, or 100 mg/kg-day for 4 or 13 weeks. Endpoints monitored included clinical signs, body weight, food consumption,

hematology and clinical chemistry parameters and serum electrolytes, organ weights, and gross and microscopic evaluation of tissues and organs. Some dogs were removed from treatment at weeks 4 and 13 and placed on a control diet for 4 weeks to evaluate reversibility of the effects. All dogs in the high-dose group died between weeks 2 and 8. The signs exhibited by these dogs consisted of listlessness, incoordination, lack of balance, pale gums, dark urine, and weakness, particularly of the hind limbs; tremors were seen occasionally. Terminal signs seen in some dogs included yellow gums and darkened sclera. Gross necropsy showed little body fat, dehydration, and jaundice. Two female dogs from the mid-dose group died on week 9. A Fisher Exact test conducted by SRC comparing death in the control and mid-dose group yielded a *p* value of 0.233, indicating a non-statistically significant difference. However, the deaths may have been compound related, as gross necropsy showed emaciation and jaundice, and group sizes were too small for the statistical test to have much power to detect an effect. In general, the dogs in the mid-dose group showed signs similar to those seen in the high-dose group, but in milder form. In addition, the signs of toxicity in the mid-dose group were not seen until week 4.

No significant treatment-related effects occurred in the low-dose group other than mild splenic hematopoiesis in some dogs; however, animals at the mid-dose and high-dose levels showed clear signs of toxicity (neurological, hematological and liver histopathology) and the incidence and severity of the effects were dose-related. The extramedullary hematopoiesis observed at the low-dose appeared to be reversible even at the higher doses depending upon the length of exposure and post-exposure recovery period. Therefore, based on the reversibility of the mild effects with cessation of exposure, the low-dose of 4 mg/kg-day is designated the NOAEL.

No significant alterations in body weight were seen in control and low-dose animals, but dogs in the mid-dose group began to lose weight on week 4 or 5, which correlated with the toxicity signs mentioned above (Lee et al., 1976). The high-dose dogs lost weight from the first week of treatment. Food consumption correlated with weight changes. Control and low-dose group dogs showed mild fluctuations in hematology and clinical chemistry parameters which were considered to be of no toxicological significance by the authors. However, mid-dose animals showed significant effects, including anemia characterized by decreases in hematocrit and hemoglobin with a compensatory reticulocytosis. Small amounts of methemoglobin were seen at week 8 and Heinz bodies at week 13. Serum alanine aminotransferase (ALT) activity was increased at weeks 8 and 13. One of the females that died in week 9 was severely anemic, with large amounts of Heinz bodies and methemoglobin, and elevated serum ALT, aspartate aminotransferase (AST) and alkaline phosphatase (AP). Blood analysis done on week 2 in high-dose dogs showed severe effects, including a 66% reduction in RBC and signs of immature erythrocytes. Also evident was leukocytosis, with increased percentage of neutrophils and decreased percentage of lymphocytes, and increased serum AP and ALT activities. Laboratory data from dogs in the mid-dose group treated for 4 or 13 weeks showed recovery after 4 weeks, but high-dose dogs treated for 4 weeks did not recover until week 19.

No significant alterations in organ weights were seen in the low-dose groups compared to the control group (Lee et al., 1976). Treatment-related histological alterations in the mid- and high-dose groups after 4 weeks of treatment included extramedullary hematopoiesis in the liver and spleen, bile duct hyperplasia, degeneration and/or subacute inflammation in the liver, and degeneration and/or depression of spermatogenesis in the testes. The incidence and severity of these lesions were generally dose-related. Lymphoid depletion in the spleen and lymph node, and involution of the thymus were also seen in high-dose animals. A female dog from the low-dose group had several graafian follicles, but no corpora lutea. This female also had mild extramedullary hematopoiesis in the spleen. Since this was seen in dogs treated for 13 weeks at this dose level and at higher dose levels, these alterations were considered compound-related. Treatment for 13 weeks with the mid or high dose of 2,6-dinitrotoluene caused similar lesions in the liver and spleen. It also caused kidney effects consisting of dilated tubules, foci of inflammation, degeneration, yellow pigment and /or casts in the tubules. The high dose also caused lesions in the testes, lymph nodes and thymus. The effects at 13 weeks were usually more numerous and more severe than those seen at 4 weeks. Also compound-related was the finding of mild extramedullary hematopoiesis and lymphoid depletion in the spleen of some dogs from the low dose group. In dogs treated for 4 weeks and allowed to recover there were lesser amounts of extramedullary hematopoiesis and testicular toxicity. Two dogs given the high dose and allowed to recover for 19 weeks showed complete recovery. Dogs treated for 13 weeks did not show full recovery, as one dog in the mid-dose group still had various lesions in the liver, kidney, and testes and a low-dose female dog still had minimal bile duct hyperplasia. To determine whether treatment with 2,6-dinitrotoluene causes an allergic reaction, the authors measured its effects on serum IgE levels. The results revealed no apparent change in serum IgE concentration. Based on the effects on body weight, hematological and neurological effects, and histopathology, the dose level of 4 mg/kg-day is considered the study NOAEL. Mild splenic hematopoiesis seen in some dogs at this dose level was not considered to be adverse. The 20 mg/kg-day dose level is the LOAEL, but it could also be considered a FEL for the death of two female dogs on week 9. The signs of emaciation and jaundice seen in these dogs upon necropsy are consistent with those seen in the high-dose dogs and are likely compound-related.

Lee et al. (1976) also conducted a similar study in rats. Groups of CD rats (16/sex/dose level) were fed diets containing 2,6-dinitrotoluene at 0, 0.01, 0.05, or 0.25% for up to 13 weeks. According to the investigators, the diet provided 0, 7, 35, or 145 mg of test material/kg-day to male rats and 0, 7, 37, or 155 mg/kg-day to female rats. Treatment with 2,6-dinitrotoluene did not cause overt neuromuscular signs as in dogs, but high-dose rats were less active. They also had rough coats and signs of malnutrition. There were no unscheduled deaths. Body weights were markedly and consistently reduced in the mid- and high-dose males and females throughout the exposure period. At 13 weeks, body weights were 20-30% lower than controls in mid-dose rats and 30-55% lower than controls in high-dose rats. Body weights were also mildly reduced in low-dose males and females for much of the study, but the difference from controls was not considered to be toxicologically significant (generally less than 10%). The decreases in body weight were associated with corresponding decreases in food consumption. No significant

and/or consistent treatment-related effects were seen in the low-dose rats. Mid-dose rats showed extramedullary hematopoietic activity in the spleen and/or liver, bile duct hyperplasia, and/or depression of spermatogenesis and atrophy of the testes, and some rats had elevated serum ALT. In addition to these changes, the high-dose rats exhibited methemoglobinemia, Heinz bodies, anemia, and compensatory reticulocytosis. In general, the effects in the high-dose rats were more severe and occurred earlier than in the mid-dose rats. There appeared to be some adaptation in the high-dose group during the treatment period, as judged by the increasing amounts of food consumption and decreasing degree of anemia from week 4 to week 13, but the tissue lesions, particularly in the testes, worsened during the course of treatment. Only partial recovery of the tissue lesions was seen after the 4-week recovery periods. As seen in dogs, 2,6-dinitrotoluene did not increase serum IgE concentrations. Based on the changes in body weight, hematology, and histopathological lesions, the dose level of 7 mg/kg-day can be considered a NOAEL and 35 mg/kg-day the study LOAEL.

In another study of similar design, Lee et al. (1976) fed albino Swiss mice (16/sex/dose level) a diet containing 0, 0.01, 0.05, or 0.25% 2,6-dinitrotoluene for up to 13 weeks. According to the authors, the corresponding intakes of test material were 0, 11, 51, or 289 mg/kg-day for males and 0, 11, 55, or 299 mg/kg-day for females. No compound-related effects were observed in the low-dose group. Several unscheduled deaths occurred during the study, including 2 among the controls (week 12), 3 in the low-dose group (weeks 1, 3, and 13), 9 in the mid-dose group, and 14 in the high-dose group; all high-dose males died before week 9. The authors stated that in the mid- and high-dose groups most of the deaths were due to 2,6-dinitrotoluene. The exact cause of death was not discussed, but most dead mice were usually of low body weight, frequently with significant weight losses a week or two before death. In the mid- and high-dose groups, food consumption was lower than in controls. Blood analyses in the mid- and high-dose groups revealed a number of statistically significant changes relative to the controls at the respective time intervals; however, the authors stated that the changes were mild, inconsistent, and not related to 2,6-dinitrotoluene. Marked aspermatogenesis was observed in all males from the high-dose group, and depressed spermatogenesis was seen in one male from the mid-dose group treated for 4 weeks and in two males from the low-dose group treated for 4 or 13 weeks. Bile duct hyperplasia occurred in the only mouse that survived treatment with the high dose of 2,6-dinitrotoluene for 13 weeks and in two mice fed the mid-dose for 13 weeks. Bile duct hyperplasia was also present in two high-dose mice treated for 4 weeks and allowed to recover for 4 weeks, suggesting that this lesion develops slowly. The investigators also indicate that extramedullary hematopoiesis in the liver and spleen was seen more often in mice treated with 2,6-dinitrotoluene than in the controls and that generally, the incidence and severity were dose-related. No testicular lesions were observed in mice treated for 4 weeks and allowed to recover for 4 weeks. Whether this would also happen following 13 weeks of treatment is unknown, since no high-dose males survived longer than 8 weeks. There was partial recovery of the bile duct hyperplasia after the 4-week recovery period, but extramedullary hematopoiesis continued to occur in the liver and/or spleen. Based on the testicular effects in male mice, decreased food consumption, and incidence and severity of extramedullary hematopoiesis in

mid- and high-dose group, the dose of 11 mg/kg-day can be considered a NOAEL and 51 mg/kg-day a LOAEL.

Goldsworthy et al. (1986) conducted a study to evaluate the influence of various diets on the hepatocarcinogenicity of 2,6-dinitrotoluene. Limited information regarding non neoplastic effects is available from this study. Groups of Fischer 344/CrlBR male rats (30/group) were fed diets that provided 0, 0.6-0.7, or 3.0-3.5 mg 2,6-dinitrotoluene/kg-day for up to 12 months (the test material was 99.9% pure). Interim sacrifices were conducted at various times for evaluation of liver weight and neoplastic nodules in the liver. All groups receiving the high-dose of 2,6-dinitrotoluene gained approximately 10% less weight than their respective controls. Liver weights were not significantly altered throughout the study with respect to the control groups, except for the high-dose group, which showed multiple gross lesions (tumors) at 12 months. No further information was provided regarding non neoplastic effects. Hence, this study cannot be used to derive any non-cancer benchmarks.

In another carcinogenicity study, groups of male Fischer 344/CrlBR rats (28/dose level) were fed a diet that provided 0, 7, or 14 mg 2,6-dinitrotoluene/kg-day for 52 weeks (Leonard et al., 1987). The authors stated that purified 2,6-dinitrotoluene was used, but the actual purity was not specified. Body weights were checked every two weeks throughout the study. Some hepatic microsomal and cytosolic enzyme activities, phenotypic markers of neoplastic nodules, were measured from animals killed after 4 and 26 weeks of treatment. At the end of the treatment period, the liver and lungs were removed and weighed, and the liver was prepared for histological evaluation. Serum ALT and γ -glutamyl transferase (GGT) activities were also determined. At week 26 of treatment, terminal body weight in the low- and high-dose rats was decreased by 5% and 10%, respectively, relative to controls. Absolute liver weight was increased by 9.3% and 18.7%, and relative liver weight was increased by 15% and 49%, in the low- and high-dose groups, respectively. Serum ALT activity was reduced, although not significantly, whereas serum GGT activity was increased 6-fold and 18-fold in the low- and high-dose groups, respectively. Similar, but much more pronounced effects were seen after 52 weeks of treatment. At this time, body weight was reduced by 18% and 32% in the low- and high-dose groups, respectively, relative to controls, whereas absolute liver weight in the same groups was increased 2-fold and 4-fold. Microscopic evaluation of the liver revealed hepatocyte degeneration and vacuolation in the majority of the treated animals, but the effects did not appear to be dose-related. These changes were only occasionally seen in controls. Over 90% of the treated animals had acidophilic and basophilic foci; no foci were apparent in controls. No specific mention was made of non neoplastic effects in the lungs. No NOAEL can be defined in this study. Based on the changes in body weight, liver weight, and liver pathology, the study LOAEL is 7 mg/kg-day.

Other Studies

Information summarized by IARC (1996) indicates that 2,6-dinitrotoluene is weakly mutagenic in *Salmonella typhimurium* strain TA98 without metabolic activation and in TA1535 and TA1537 with rat liver S9. 2,6-Dinitrotoluene did not induce morphological transformation in Syrian hamster embryo cells, but induced DNA strand breaks in rat hepatocytes *in vitro*. IARC (1996) also states that 2,6-dinitrotoluene did not induce gene mutation to 6-thioguanine resistance in Chinese hamster ovary cells with or without metabolic activation, and that administration of 2,6-dinitrotoluene to rats *in vivo* induced unscheduled DNA synthesis in hepatocytes. Lee et al. (1976) reported that dosing rats with 35-37 mg of 2,6-dinitrotoluene/kg-day for either 6 or 13 weeks caused an increased number of chromatid breaks and gaps in peripheral lymphocytes and the same dose for 13 weeks also caused an increase in tetraploids in kidney cultures. Lee et al. (1976) also reported that 2,6-dinitrotoluene was not mutagenic in the specific locus mutation test using Chinese hamster ovaries.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC ORAL RfD VALUES FOR 2,6-DINITROTOLUENE

Lee et al. (1976) conducted a 13-week subchronic oral study with 2,6-dinitrotoluene in dogs administered the test compound daily in gelatin capsules; the doses tested were 0, 4, 20, and 100 mg/kg-day. No significant treatment-related effects occurred in the low-dose group other than mild splenic hematopoiesis in some dogs. Animals given the mid- and high-dose levels showed clear signs of toxicity (neurological, hematological, and liver histopathology) and the incidence and severity of the effects were dose-related. All dogs from the high-dose group died between weeks 2 and 8 and two females from the mid-dose group died on week 9. A comparison of the incidence of death between the mid-dose group and control group (2/8 vs. 0/8; includes males and females) using the Fisher Exact test yielded a *p* value of 0.233, which is not statistically significant, but the group sizes may have been too small for the test to detect an effect. However, the toxic effects which may have led to the death of the two females, particularly emaciation, suggest that lethality was compound-related and that the dose level of 20 mg/kg-day should be considered a FEL. NOAELs of 7 and 11 mg/kg-day were defined in similar studies in rats and mice, respectively, also conducted by Lee et al. (1976). The corresponding LOAELs were 35 and 51 mg/kg-day. Limited additional information on non cancer endpoints is available from two 12-month oral studies of the carcinogenicity of 2,6-dinitrotoluene (Goldsworthy et al., 1986; Leonard et al., 1987). In the former, doses of 2,6-dinitrotoluene of 3.0-3.5 mg/kg-day, but not 0.6-0.7 mg/kg-day, produced an approximately 10% reduction in body weight gain and induced liver tumors. In the Leonard et al. (1987) study, the lowest dose tested, 7 mg/kg-day, significantly reduced body weight gain and increased absolute liver weight, and induced histological changes in the liver indicative of pre-neoplastic effects. No developmental studies of 2,6-dinitrotoluene were located. The only information regarding reproductive effects of 2,6-dinitrotoluene is that from Lee et al. (1976), who reported

adverse testicular effects in dogs, rats, and mice in 13-week studies. The highest reliable NOAEL in the literature reviewed is 4 mg/kg-day from the Lee et al. (1976) study in dogs and can serve as basis for derivation of a provisional subchronic and chronic RfD.

A **provisional subchronic RfD of 0.01 mg/kg-day** is derived by applying an uncertainty factor of 300 (10 to extrapolate from dogs to humans, 10 to protect sensitive individuals and 3 for database limitations, including lack of reproductive or developmental studies) to the NOAEL of 4 mg/kg-day, as follows:

$$\begin{aligned}\text{subchronic p-RfD} &= \text{NOAEL} / \text{UF} \\ &= 4 \text{ mg/kg-day} / 300 \\ &= 0.01 \text{ mg/kg-day or } 1\text{E-}2 \text{ mg/kg-day}\end{aligned}$$

A **provisional chronic RfD of 0.001 mg/kg-day** is similarly derived by applying an uncertainty factor of 3000 (10 each for intraspecies variability, interspecies extrapolation, extrapolation from subchronic to chronic data, and 3 for a deficient database, including lack of reproductive or developmental studies) to the NOAEL of 4 mg/kg-day as follows:

$$\begin{aligned}\text{p-RfD} &= \text{NOAEL} / \text{UF} \\ &= 4 \text{ mg/kg-day} / 3000 \\ &= 0.001 \text{ mg/kg-day or } 1\text{E-}3 \text{ mg/kg-day}\end{aligned}$$

Confidence in the key study is medium because although a variety of endpoints were evaluated (clinical signs, hematology, gross and histological pathology), the number of animals per group (4) constitute a small sample size. Confidence in the database is medium because there are supporting subchronic studies in rats and mice, but, at the same time, there is a lack of lifetime and developmental/reproductive studies. Reproductive studies would be especially relevant since the subchronic studies found testicular and sperm effects in dogs, rats, and mice. Medium confidence in the provisional subchronic and chronic RfD values follows.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfC VALUES FOR 2,6-DINITROTOLUENE

No information was located that could be used to derive provisional inhalation RfC values for 2,6-dinitrotoluene. No relevant studies were found in animals. Workers with occupational exposure to 2,6-dinitrotoluene generally were exposed to the technical grade mixture, which consists primarily of the 2,4-dinitrotoluene isomer, had dermal as well as inhalation exposure, and did not have their exposure to 2,6-dinitrotoluene quantified.

DERIVATION OF A PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2,6-DINITROTOLUENE

A cancer assessment, including derivation of an oral slope factor, is available for a mixture of 2,4-dinitrotoluene (98% by weight) and 2,6-dinitrotoluene (2% by weight) on IRIS (U.S. EPA, 2002b). Such a slope factor derived from a mixture of these two isomers cannot be used to extrapolate to the carcinogenicity of either individual isomer, due to the fact that the potency of the isomers may be extremely different. In the absence of carcinogenicity data (potency) on pure 2,6-DNT, one cannot develop a provisional oral slope factor.

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7-3-2007

Provisional Peer Reviewed Toxicity Values for
2-Chlorophenol
(CASRN 95-57-8)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value

RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2-CHLOROPHENOL (CASRN 95-57-8)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A chronic reference dose (RfD) value of 5E-3 mg/kg-day is available for 2-chlorophenol on IRIS (U.S. EPA, 1988a) and in the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). The HEAST (U.S. EPA, 1997) lists a subchronic RfD for 2-chlorophenol of 5E-2 mg/kg-day. Both RfD values were based on a no-observed-adverse-effect level (NOAEL) of 5 mg/kg-day for reproductive effects in a drinking water study that exposed rats to 2-chlorophenol for 10 weeks prior to mating and during mating, gestation and weaning (Exon and Koller, 1982). Uncertainty factors of 100 and 1000 were used to derive the subchronic and chronic RfDs, respectively. The source documents for the RfD assessments included a Drinking Water Criteria Document (DWCD) (U.S. EPA, 1986a), a Health Effects Assessment (HEA) (U.S. EPA, 1987a), and two Health and Environmental Effects Documents (HEEDs) (U.S. EPA, 1987b, 1990). The Chemical Assessments and Related Activities (CARA) lists (U.S. EPA, 1991, 1994) do not include any additional relevant EPA documents. The Agency for Toxic Substances and Disease Registry (ATSDR, 1999) and the World Health Organization (WHO, 1989) have assessed the health effects of chlorophenols, but did not derive any oral risk assessment values specifically for 2-chlorophenol.

An RfC for 2-chlorophenol is not available on IRIS (U.S. EPA, 1988a) nor in the HEAST (U.S. EPA, 1997). The Agency for Toxic Substances and Disease Registry (ATSDR) and the World Health Organization (WHO) have not derived any inhalation risk assessment values for 2-chlorophenol. Occupational exposure limits for 2-chlorophenol have not been derived by the American Conference for Governmental Industrial Hygienists (ACGIH), the National Institute

for Occupational Safety and Health (NIOSH) or the Occupational Safety and Health Administration (OSHA).

A cancer assessment for 2-chlorophenol is not available on IRIS (U.S. EPA, 1988a). The HEEDs (U.S. EPA, 1987b, 1990) assigned 2-chlorophenol to U.S. EPA (1986b) Cancer Group D (not classifiable as to human carcinogenicity); this classification is also included in the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). The carcinogenicity of 2-chlorophenol has not been assessed by NTP or IARC.

Literature searches were conducted from the 1960's through August, 2006 for studies relevant to the derivation of provisional toxicity values for 2-chlorophenol. Data bases searched included: TOXLINE/TOXCENTER (including BIOSIS, NTIS and Chemical Abstracts subfiles), MEDLINE (including PubMed cancer subset), TSCATS/TSCATS 2, CCRIS, DART/ETIC, GENETOX, HSDB, RTECS, and Current Contents.

REVIEW OF PERTINENT DATA

Human Studies

Relevant information regarding the toxicity of 2-chlorophenol in humans was not located.

Animal Studies

Oral Exposure. In a 14-day study performed in conjunction with EPA, groups of 12 male and 12 adult female CD-1 ICR mice were administered 2-chlorophenol in corn oil by gavage in doses of 0, 35, 69 or 175 mg/kg-day (Borzelleca, 1983; Borzelleca et al., 1985). The highest dose level was approximately 50% of the acute oral LD₅₀ of 347 and 345 mg/kg in male and female CD-1 mice, respectively. Endpoints evaluated during the study included clinical observations, body weight (days 1, 8 and 15), and food and water intake. Endpoints evaluated at the end of the treatment period included hematology [red blood cells (RBC), total and differential white blood cells (WBC), platelets, hematocrit (Hct), hemoglobin (Hgb) and coagulation], serum chemistry [lactate dehydrogenase (LDH), alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), blood urea nitrogen (BUN), bilirubin, protein, glucose, cholesterol, albumin/globulin, phosphorus, potassium, calcium, sodium and chloride], hepatic microsomal activities (cytochrome P₄₅₀, cytochrome b₅, protein, aminopyrine demethylase, aniline hydroxylase, and arylhydrocarbon hydroxylase), immune response and behavioral measurements. The earlier report of the study (Borzelleca, 1983) implies that the immunology endpoints included cell-mediated response (Delayed-type hypersensitivity (DTH) response to sheep RBC, response to concanavalin A), humoral response [splenic Immunoglobulin mu (IgM) antibody forming cells (AFC) to sheep RBC, serum antibody levels to sheep RBC, lymphocyte response to lipopolysaccharide (LPS)], and reticuloendothelial system (RES) function (vascular clearance and uptake of ⁵¹Cr sheep RBC). The Borzelleca (1983) report also implies that the behavioral endpoints included inverted screen test, swimming endurance, locomotor activity, pain sensitivity, olfactory sensitivity, passive avoidance learning, and forepaw grip strength. Other endpoints included sister-chromatid exchange (bone marrow

and/or testes, not otherwise specified), *in vitro* fertilization capability (penetration of ova, fertilization, blastula formation), absolute and relative organ weights, and gross pathology. Histopathological examinations were not performed. The results of this study are qualitatively reported in tabular summaries. Effects included 100% mortality at 175 mg/kg-day, hyperactivity at 35 and 69 mg/kg-day, reduced body weight at 69 mg/kg-day, and reduced brain, liver and spleen weights (effect levels not indicated); additional information on these effects was not reported. No biologically or statistically significant compound-related adverse effects were reported for the other endpoints as indicated by the authors. The 100% mortality in the high-dose animals indicates that 175 mg/kg-day was a FEL for short-term repeated gavage exposures in mice. The authors (Borzelleca et al., 1985) referred to the effects at the lower doses as “slight toxic effects”, but apparently concluded that they were not biologically significant, indicating that 69 mg/kg-day was a NOAEL. Results of acute studies reported by Borzelleca et al. (1985) include an ED₅₀ of 63 mg/kg for reversible motor impairment in mice exposed to a single oral dose of 2-chlorophenol; additional information was not provided.

Gavage studies (10-day and 90-day) of 2-chlorophenol in Sprague-Dawley rats were conducted by the EPA (Daniel et al., 1993). In the 10-day study, groups of 10 male and 10 female 8-week-old Sprague-Dawley rats were administered 2-chlorophenol in corn oil by daily gavage at doses of 0, 13, 64, 129 or 257 mg/kg-day. The highest dose level was approximately 38% of the reported acute LD₅₀ of 670 mg/kg for a rat. Endpoints evaluated during the study included clinical signs (observed for physiological and behavioral responses and mortality), body weight and food and water consumption. Evaluations at the end of the exposure period included hematology [RBC, WBC, Hct, Hgb and mean corpuscular volume (MCV)], serum chemistry (ALP, AST, ALT, LDH, cholesterol, BUN, creatinine, glucose, and calcium), absolute and relative organ weights (brain, liver, spleen, lungs, thymus, kidneys, adrenal glands, heart, and gonads), and gross pathology. Comprehensive histological examinations were performed in the control and high-dose groups; target organs were also histologically evaluated at the lower dose levels. Tissues that were examined included liver, kidneys, urinary bladder, heart, aorta, skin, skeletal muscle, bone, sciatic nerve, spleen, thymus, lymph nodes, respiratory tract (nasal turbinates, trachea, lung with bronchi), gastrointestinal tract (esophagus, stomach, duodenum, jejunum, ileum, cecum, colon, rectum), endocrine system (adrenals, pancreas, pituitary, thyroid/parathyroid), and reproductive system (testes, epididymis, seminal vesicles, prostate, preputial gland, ovaries, uterus, clitoral gland).

There were no treatment-related deaths, significant clinical observations or significant changes in food or water consumption or body weight gain (Daniel et al., 1993). The hematology evaluations found significantly ($p \leq 0.05$) increased RBC count (12% higher than controls) and Hct (28% higher than controls) in the high-dose (257 mg/kg-day) males; these effects were not clearly dose-related and there were no significant changes in hematologic values in females. Serum chemistry changes that were statistically significant included increased glucose levels in females at 129 and 257 mg/kg-day (45 and 42% higher than controls, respectively) and males at 257 mg/kg-day (21% higher than controls); decreased ALP in females at 129 and 257 mg/kg-day (15 and 16% lower than controls); and decreased AST, cholesterol, and LDH in males at 257 mg/kg-day (25, 27 and 55% lower than controls, respectively). Serum LDH values were significantly decreased in females at 64 and 129 mg/kg-day, but not at 257 mg/kg-day. The only serum chemistry changes that appeared to be dose-related were the

increased glucose and decreased ALP in female rats, but the authors reported that these values were within the normal ranges for laboratory rats. Statistically significant organ weight changes consisted of decreases in absolute kidney and heart weights in females at 129 mg/kg-day, but not at other dose levels, and decreases in absolute and relative lung weights in females at all dose levels; quantitative data were not reported. Necropsy findings included enlarged mandibular lymph nodes, reddened lungs and reduced thymus size in all groups of both sexes; these were minimal to mild changes not considered to be treatment-related by the authors. The histological examinations similarly showed lymphoid hyperplasia, mild congestion of the lungs, and mild thymic atrophy in all groups; these effects did not appear to be treatment-related to the authors because they were not significant in severity or incidence (data not reported). Histopathological changes in kidneys, heart, lungs or other tissues were not reported. The lack of any clear treatment-related or biologically significant hematology, clinical chemistry, organ weight or pathological changes indicates that the highest dose level, 257 mg/kg-day, is a NOAEL for 10-day gavage exposure in male and female rats although it is difficult to ascertain the significance of the reported effects due to a lack of data reporting.

In the 90-day study, groups of 10 male and 10 female 8-week-old Sprague-Dawley rats were administered 2-chlorophenol in corn oil by daily gavage at doses of 0, 17, 50, or 150 mg/kg-day (Daniel et al., 1993). Study endpoints were the same as in the 10-day study summarized above; evaluations included clinical signs, body weight, food and water consumption, hematology, serum chemistry (with the addition of triglycerides, total protein, albumin and globulin), organ weights and gross pathology in all groups, and histopathology in the control and high-dose groups. There were no clinical signs of toxicity, unscheduled deaths, or significant changes in food or water consumption or body weight gain. Hematology changes that were statistically significant included increased RBC count in females at 17 and 150 mg/kg-day (3 and 6% higher controls), but not at 50 mg/kg-day; increased Hct in females at 150 mg/kg-day (5% higher than controls); and increased MCV in males at 150 mg/kg-day (3% higher than controls). Serum chemistry changes that were statistically significant included decreased ALP in males at 50 and 150 mg/kg-day (31 and 28% less than controls), decreased AST in males at 50 and 150 mg/kg-day (22 and 19% less than controls), decreased ALT in males at 50 and 150 mg/kg-day (18 and 18% less than controls), and increased glucose at 50 mg/kg-day (16% higher than controls; similar increases occurred at 17 and 150 mg/kg-day but were not statistically significant). Although statistically significant changes were observed for these and several other hematology and clinical chemistry indices, no responses were clearly dose-related, consistent between sexes or, according to the authors, outside normal ranges or biologically significant. There were no clear effects on organ weights; the only statistically significant changes were increased relative liver weight in females at 17 mg/kg-day, increased absolute spleen weight in males at 17 and 50 mg/kg-day, and increased absolute brain weight in males at 50 mg/kg-day; quantitative data were not reported. There were no gross or histopathological changes in either sex. The lack of any clear treatment-related or biologically significant hematology, clinical chemistry or organ weight changes, as well as the lack of any pathological effects, indicates that the highest dose level, 150 mg/kg-day, is a NOAEL for 90-day gavage exposure in rats.

The oral toxicity of 2-chlorophenol was also assessed in 18-day studies with preweanling rats and in 14- and 28-day studies with juvenile rats (Hasegawa et al., 2005). Preweanling Sprague-Dawley SPF rats were administered 2-chlorophenol in olive oil by gavage on postnatal

days (PNDs) 4-21 in dose-finding and main studies. In the 18-day dose-finding study with preweanling rats, groups of 4 males and 4 females were exposed to dose levels of 0, 20, 100 or 500 mg/kg-day. General behavior and body weight were evaluated during the study, and hematology, blood chemistry, gross pathology and organ weights were evaluated on PND 22; histopathology was not assessed. Although not specifically reported, it is assumed that the scope of these evaluations was the same as in the main study with newborn rats summarized below. Effects were limited to 100% mortality by the 9th day of dosing at 500 mg/kg-day; clinical signs were not observed at 20 and 100 mg/kg-day, and no other results were reported. This study identified a FEL of 500 mg/kg-day for lethality in preweanling rats. The next lowest dose level of 100 mg/kg-day is a NOAEL based on the lack of clinical signs and systemic effects, but confidence in this effect level is low due to the small numbers of animals and lack of histological examinations.

In the main 18-day study with preweanling rats, groups of 12 male and 12 female Sprague-Dawley SPF rats were administered 2-chlorophenol in olive oil by gavage in doses of 0, 8, 50 or 300 mg/kg-day on PNDs 4-21 (Hasegawa et al., 2005). Half of the animals were sacrificed on PND 22, and the remaining 6 rats/sex/group were observed without treatment for the following 9 weeks and then sacrificed (on PND 85). Endpoints evaluated during the study included general behavior, body weight and postnatal developmental parameters, including surface righting and visual placing reflex for reflex ontogeny, fur appearance, incisor eruption and eye opening for external development, and preputial separation, vaginal opening and estrous cycle for sexual development. Comprehensive hematology and blood biochemistry evaluations were conducted at the end of the treatment period on PND 22 (6 rats/sex/dose) and end of the observation period on PND 85 (6 rats/sex/dose). Hematology indices included RBC, Hct, Hgb, MCV, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, total and differential WBC, platelet count, and reticulocyte count. Blood biochemistry indices included total protein, albumin, albumin/globulin ratio, glucose, total cholesterol, triglycerides, phospholipid, total bilirubin, BUN, creatinine, AST, ALT, ALP, γ -glutamyl transpeptidase, calcium, inorganic phosphorus, sodium, potassium and chloride. Prothrombin time, activated thromboplastin time, and urine indices (color, pH, occult blood, protein, glucose, ketone bodies, bilirubin, urobilinogen, sediment, volume and osmotic pressure) were evaluated only at the end of the observation period. Organ weights (brain, pituitary, thymus, thyroids, heart, lungs, liver, spleen, kidneys, adrenals, testes, epididymides, ovaries and uterus) and histopathology (organs that were weighed as well as macroscopically abnormal organs) were evaluated on PND 22 (6 rats/sex/dose); it was not indicated if these evaluations were performed on PND 85.

Effects included tremors in 11/12 males and 12/12 females at 300 mg/kg-day; the tremors appeared within 5 minutes of dosing and disappeared within 4 hours in most animals. At 50 mg/kg-day, 1/12 females showed tremors once from 15-30 minutes following dosing on treatment day 9. No tremors were observed in males at 50 mg/kg-day or in either sex at 0 or 8 mg/kg-day. The only other reported effects occurred at 300 mg/kg-day; these consisted of other signs of neurotoxicity (hypoactivity in 2/12 males and 3/12 females and abnormal gait in 1/12 males and 1/12 females), transiently decreased body weight in both sexes (additional information not reported), and histological changes in the kidneys (slight to moderate basophilic renal tubules in 4/6 males and 5/6 females) with increases in relative kidney weight (8% in males and 4% in females). The biological significance of the basophilic renal tubular changes was not discussed.

No results were reported for the 9-week observation period. The 300 mg/kg-day dose is a FEL for preweanling rats based on the occurrence of tremors in 23/24 of the exposed males and females; other signs of neurotoxicity (hypoactivity and abnormal gait) were also observed at this dose level. The next lowest dose of 50 mg/kg-day is a NOAEL because tremors were only observed in 1/12 females once on exposure day 9; the incidence is not statistically different from the control group (0/12) and the occurrence was isolated. Additionally, there were no clinical signs of neurotoxicity in the males exposed to 50 mg/kg-day, or in the 4 males and 4 females exposed to 100 mg/kg-day in the dose-finding study summarized above.

The studies with juvenile rats included a 14-day dose-finding study and a 28-day main study (Hasegawa et al., 2005). In the 14-day dose-finding study, 5-week-old male and female Sprague-Dawley SPF rats were administered 2-chlorophenol in olive oil by gavage in doses of 0, 100, 200 or 500 mg/kg-day; group sizes were 3 per sex at 500 mg/kg-day and were not reported for the other dose levels. General behavior, body weight and food consumption were evaluated during the study, and hematology, blood chemistry, gross pathology and organ weights were evaluated the day after the last treatment; histopathology was not assessed. Although not specifically reported, it is assumed that the scope of these evaluations was the same as in the study with newborn rats summarized above. The only information regarding the results is a statement that no toxic signs were observed, indicating that 500 mg/kg-day is a NOAEL in juvenile rats. Confidence in this effect level is low due to the apparent small numbers of animals and lack of histological examinations.

In the 28-day main study, groups of 12 male and 12 female 5- to 6-week old Sprague-Dawley SPF rats were exposed to 2-chlorophenol in olive oil by gavage in doses of 0, 8, 40, 200 or 1000 mg/kg-day (Hasegawa et al., 2005). It appears that half of the animals were sacrificed following the last treatment and the remaining 6 rats/sex/group were observed without treatment for the following 2 weeks and then sacrificed. Evaluations included general behavior, body weight, food consumption, urinalysis, hematology, blood biochemistry, gross pathology, organ weights and histopathology. Although not specifically reported, it is implied that the scope and schedule of these evaluations are the same as in the 18-day study with preweanling rats summarized above. The only effects in this study were clinical signs of neurotoxicity and histological changes in the liver in most animals only at 1000 mg/kg-day. The clinical signs occurred sporadically in both sexes within 3 hours of dosing and included tremors (4/12 males and 5/12 females), hypoactivity (8/12 males and 5/12 females) and abnormal gait (4/12 males and 7/12 females). The liver effects consisted of slight centrilobular hypertrophy of hepatocytes (6/6 males and 5/6 females); the authors indicated that this suggested a compensatory response for hepatic metabolism. None of the animals showed basophilic renal tubules as observed in the preweanling rats exposed to 300 mg/kg-day on PNDs 4-21 (see above). No results were reported for the 2-week observation period. This study identified a FEL of 1000 mg/kg-day based on the clinical signs of neurotoxicity; the NOAEL is 200 mg/kg-day.

Additional information on effects of repeated oral exposures to 2-chlorophenol is available from a series of reproductive toxicity, immunotoxicity and carcinogenicity studies in Sprague-Dawley rats that were exposed prenatally, postnatally, or both pre- and postnatally to concentrations of 0, 5, 50 or 500 ppm 2-chlorophenol in drinking water (Exon and Koller, 1982, 1983a,b, 1985). Offspring produced in the reproductive study were used in the immunotoxicity

and carcinogenicity studies. In the reproductive study, groups of 12-14 females were exposed to the treated drinking water from 3 weeks of age through breeding (to untreated males) at 90 days of age and subsequently until 3 weeks post-parturition (Exon and Koller, 1982, 1983b, 1985). Table 1 shows the statistically significant reproductive endpoints that were reported by Exon and Koller (1982). The values found in Exon and Koller (1983b, 1985) agree with each other but are slightly different from those found in Exon and Koller (1982), and differ in their statistical evaluation. The reason for the differences is unknown. Maternal and pup weight, percent conception, litter size, and number of stillbirths were evaluated at parturition. Pup survival, body weight and hematology (red and white cell counts, hemoglobin, packed cell volume, and mean corpuscular volume) were evaluated at weaning.

Table 1. Reproductive effects of 2-Chlorophenol in Rats				
Effect	Dose (ppm)			
	0	5	50	500
Litter Size (mean \pm SD)	11.4 \pm 1.2 n=12	11.7 \pm 3.5 n=12	10.1 \pm 2.3 n=12	9.2 \pm 4.3 ^b n=14
Stillborn (incidence)	0/91	2/105	0/91	6/110 ^b
^a Female rats were exposed to 2-chlorophenol in drinking water from 3 weeks of age through mating at 90 days of age and subsequently through pregnancy and lactation.				
^b Significantly different from control group (p \leq 0.05).				
Source: Exon and Koller (1982)				

Statistically significant (p \leq 0.05) changes included 19% reduced mean litter size (live and stillborn pups) at 500 ppm (9.2 \pm 4.3 compared to 11.4 \pm 1.2 in controls) and 5% increased incidence of stillbirths at 500 ppm (6/110 compared to 0/91 in controls) (Exon and Koller, 1982).

Based on the evidence of decreased litter size and an increase in stillbirth incidence, this study identified a NOAEL of 50 ppm and a LOAEL of 500 ppm for reproductive toxicity. The conversion factor for converting the amount of 2-chlorophenol ingested in drinking water (ppm) to a dose (mg/kg-day) was calculated by dividing the reference water consumption of 0.031 L/day for female Sprague Dawley rats in a subchronic study by the corresponding reference body weight in female Sprague Dawley rats (0.031 L/day/0.204 kg = 0.15 L/kg-day) (U.S. EPA, 1988b). Thus, the 5, 50 and 500 ppm doses correspond to estimated drinking water doses of 0.75, 7.5 and 75 mg/kg-day, respectively, and the NOAEL and LOAEL correspond to 7.5 and 75 mg/kg-day, respectively.

In the immunotoxicity studies, offspring from female rats described in the above studies that were exposed to 0, 5, 50 or 500 ppm 2-chlorophenol in drinking water from 3 weeks of age through mating at 90 days until 3 weeks post-parturition were continued on treatment for 10 weeks (Exon and Koller, 1983a) or 15 weeks (Exon and Koller, 1985), at which time immune responses were evaluated. Tests were conducted for humoral immunity (measured as the ratio of serum Immunoglobulin gamma (IgG) antibody levels to bovine serum albumin or keyhole limpet hemocyanin), cell-mediated immunity (measured as delayed-type hypersensitivity response in ears injected with oxazolone), and macrophage function (measured as the ability of peritoneal exudate cells to phagocytize sheep red blood cells *in vitro*) in 4 male and 4 female offspring from each exposure group. Body, liver, spleen, and thymus weights were also evaluated in these

offspring. There were no statistically significant ($p \leq 0.05$) differences between the treated and control groups for any of the immune responses or other end points, indicating that a NOAEL of 500 ppm was identified. Using conversion factors of 0.14 and 0.15 L/kg-day based on subchronic values for water consumption and body weight in male and female Sprague Dawley rats (U.S. EPA, 1988b), respectively, the NOAEL of 500 ppm identified in these studies corresponds to estimated drinking water doses of 70 mg/kg-day in males and 75 mg/kg-day in females.

In the carcinogenicity studies (Exon and Koller, 1983b, 1985), groups of 24-32 male and 24-28 female rats received combined pre- and postnatal exposures to 0, 5, 50 or 500 ppm of 2-chlorophenol in drinking water. Three-week-old females were exposed continuously through mating (90 days of age), pregnancy and lactation, and the offspring received treated drinking water from weaning for 24 months. All rats were observed daily for gross signs of morbidity, and moribund or tumor-bearing rats were sacrificed. Body weight was measured monthly in all rats, and hematology (RBC, WBC, Hct, Hgb and MCV) was evaluated every 2 weeks (Exon and Koller, 1983b) or every 2 months (Exon and Koller, 1985) in 5 males and 5 females per group. Gross and microscopic examinations of major organs and tumor tissues were conducted in all animals. There were no effects on body weight at 15 weeks (Exon and Koller, 1985) or 7 months (Exon and Koller 1983b), the only times for which data were reported. A significant decrease in body weight ($p \leq 0.10$) was observed at 7 months in females at doses of 5 and 500 ppm (7.6 and 5.2% less than controls respectively). Exon and Koller (1985) noted that red blood cell count, packed cell volume and blood hemoglobin concentrations were “generally increased” in both sexes at 500 ppm. These effects were most evident after 14 months of exposure, when the RBC, packed cell volume (PCV) and hemoglobin values were 15, 19 and 16% higher than controls ($p \leq 0.05$), respectively; no other quantitative hematology data were reported. In an earlier report of interim (15-month) findings, however, Exon and Koller (1983b) indicated that 2-chlorophenol did not affect any of the measured hematology parameters. Noncancer histopathologic observations were not reported. Although there were no clear treatment-related or biologically significant body weight or hematology changes, the lack of noncancer histopathology data precludes identification of a NOAEL or LOAEL for chronic toxicity. There were no statistically significant ($p \leq 0.10$) differences between exposed and control groups in tumor incidence, latency or type in either sex. Incidences of total tumors in the 0, 5, 50 and 500 ppm groups were 13, 17, 8 and 18% in males, and 5, 0, 13, and 18% in females, respectively; no other incidence data were reported.

Inhalation Exposure. Relevant information regarding the inhalation toxicity of 2-chlorophenol in animals was not located.

Other Studies

Co-carcinogenicity and Tumor Promotion. In a co-carcinogenicity study (Exon and Koller, 1983b, 1985), groups of 24-32 male and 24-28 female Sprague-Dawley rats were exposed prenatally, postnatally, or both pre- and postnatally to 0, 5, 50 or 500 ppm 2-chlorophenol in drinking water, with prenatal exposure to the known carcinogen ethylnitrosourea (ENU). Comparison groups received prenatal exposure to ENU alone; comparisons were not made to offspring unexposed to ENU or 2-chlorophenol. Rats were exposed to ENU as its precursors,

ethylurea (0.316% in feed) and sodium nitrite (1 ppm in drinking water), on days 14-21 of gestation. Prenatal exposure to 2-chlorophenol involved exposing 3-week-old females through mating (90 days of age) and pregnancy; the dams were not exposed during lactation, and the offspring were observed without treatment from weaning for 24 months. Postnatal exposure to 2-chlorophenol involved exposing offspring from unexposed dams to the treated water from weaning for 24 months. Combined pre- and postnatal exposure to 2-chlorophenol involved exposing 3-week-old females continuously through mating (90 days of age), pregnancy and lactation, and subsequent exposure of the offspring to the treated water from weaning for 24 months. Histological examinations were performed on major organs and grossly observed tumors, but data were only reported for total tumors.

Male offspring of rats treated with ENU and combined pre- and postnatal exposure to 2-chlorophenol, at all treatment levels, had significantly ($p \leq 0.10$) increased incidences of total tumors when compared to the group exposed to ENU alone (Table 2). Significantly higher incidences of total tumors also occurred in male offspring exposed to ENU and 2-chlorophenol given prenatally at 5 and 500 ppm (but not 50 ppm), male offspring exposed to ENU and 2-chlorophenol given postnatally at 5 ppm, and female offspring exposed to ENU and 2-chlorophenol given prenatally or postnatally at 500 ppm. Tumor latency (mean days to tumor) was significantly decreased in rats exposed to ENU with combined pre- and postnatal exposure to 2-chlorophenol at all treatment levels when compared to the group exposed to ENU alone. Although total tumor incidence was increased and time-to-tumor latency was decreased in all groups of male rats with combined pre- and postnatal exposure to 2-chlorophenol compared with those exposed to ENU alone, interpretation of the findings is complicated by a high tumor incidence in the group exposed to ENU alone, lack of a dose-response relationship, and lack of similar effects in females (Table 2). The authors concluded that the results suggest that 2-chlorophenol may act as a co-carcinogen or promoter of carcinogenesis.

Table 2. Tumor Incidence and Latency in Rats Exposed Pre- and Postnatally to 2-Chlorophenol with Prenatal Exposure to ENU (Exon and Koller, 1983b)						
2-Chlorophenol (ppm) (Pre-and Postnatal + ENU)	Total Tumor Incidence (%)			No. Rats/Group		Days to Tumor (mean \pm SE)
	Total	Male	Female	Male	Female	
Unexposed	3	7	0	30	30	422 \pm 40
ENU only	58	54	63	28	24	302 \pm 16
5	85 ^a	92 ^a	79	24	24	245 \pm 14 ^a
50	63	75 ^a	50	24	24	256 \pm 17 ^a
500	68	77 ^a	60	30	30	259 \pm 14 ^a
^a $p \leq 0.10$ compared to ENU positive control group by chi-square test (incidence data) or analysis of variance (least-square means) (latency data).						

The skin tumor-promoting ability of 2-chlorophenol was assessed in 2- to 3-month old female albino Sutter mice (Boutwell and Bosch, 1959). When 25 μ l of a 20% solution of 2-chlorophenol in benzene was applied to shaved back skin twice weekly for 15 weeks following initiation with a single 25 μ l application of 0.3% DMBA (9,10-dimethyl-1,2-benz[a]anthracene) in benzene, 31/35 mice survived compared to 15/20 similarly initiated vehicle control mice. Of the survivors, 61% had skin papillomas compared to 7% in controls, and 10% had skin

carcinomas compared to 0% in controls. When 2-chlorophenol was applied as a 20% solution in dioxane to uninitiated mice twice weekly for 12 weeks, 28/30 mice survived; 46% of the survivors had papillomas and 0% developed carcinomas. A dioxane-treated vehicle control group was not reported.

Genotoxicity. A limited amount of information is available on the genotoxicity of 2-chlorophenol. 2-Chlorophenol did not induce reverse mutations in *Salmonella typhimurium* strains TA98, TA100, TA1535 and TA1537 when tested with or without exogenous metabolic activation (Haworth et al., 1983). 2-Chlorophenol did not induce DNA-repairing genes (*umuDC*) in *S. typhimurium* TA1535/pSK1002 (Ono et al., 1992), or DNA damage in *Escherichia coli* as shown by the induction of prophage lambda (DeMarini et al., 1990), when tested with or without exogenous metabolic activation. Sister-chromatid exchanges were not increased in mice that were exposed to 2-chlorophenol in corn oil by gavage in doses of 35-175 mg/kg-day for 14 days (Borzelleca et al., 1985); bone marrow and testicular cells (specific cell types not indicated) were examined.

DERIVATION OF A PROVISIONAL SUBCHRONIC ORAL RfD FOR 2-CHLOROPHENOL

Subchronic RfD

Information relevant to the derivation of a subchronic oral RfD for 2-chlorophenol is available from one 14-day study in mice (Borzelleca, 1983; Borzelleca et al., 1985) and several studies in rats ranging in exposure duration from 10 days to approximately 16-21 weeks (Daniel et al., 1993; Exon and Koller, 1982, 1983a, 1985; Hasegawa et al., 2005). The preponderance of these studies used gavage exposure and showed frank toxic effects, particularly mortality and clinical signs of neurotoxicity, as summarized in Table 3. The gavage studies identified FELs of 175 mg/kg-day for mortality in mice exposed for 14 days (Borzelleca, 1983; Borzelleca et al., 1985), 300 mg/kg-day for overt neurotoxicity (tremors) and 500 mg/kg-day for mortality in preweanling rats exposed for 18 days on PNDs 4-21 (Hasegawa et al., 2005), and 1000 mg/kg-day for overt neurotoxicity (tremors, hypoactivity and abnormal gait) in rats exposed for 28 days (Hasegawa et al., 2005). Although these are generally well-designed studies with comprehensive evaluations that included clinical signs, body weight, hematology, clinical chemistry, organ weights, histology and, in the study with preweanling rats, postnatal developmental indices, they did not identify more subtle indicators of toxicity and actual data were not supplied in some instances. NOAELs in the gavage studies were 69 mg/kg-day in mice exposed for 14 days (Borzelleca, 1983; Borzelleca et al., 1985), 50 and 100 mg/kg-day in preweanling rats exposed for 18 days on PNDs 4-21 (Hasegawa et al., 2005), 150 mg/kg-day in rats exposed for 90 days (Daniel et al., 1993), and 200 mg/kg-day in rats exposed for 28 days (Hasegawa et al., 2005).

Table 3. Summary of Effect Levels from Oral Toxicity Studies of 2-Chlorophenol

Species	Exposure Duration	NOAEL ^a	LOAEL ^a	FEL ^a	Effects	Reference
mouse	14 days (gavage)	69	ND	175	100% mortality at 175 mg/kg-day. No biologically significant effects at 69 mg/kg-day ^{b,c} .	Borzelleca, 1983; Borzelleca et al., 1985
rat	10 days (gavage)	257	ND	ND	No clear treatment-related or biologically significant effects ^b .	Daniel et al., 1993
rat	90 days (gavage)	150	ND	ND	No clear treatment-related or biologically significant effects ^{b,d} .	Daniel et al., 1993
rat	18 days (PND 4-21) (gavage)	100	ND	500	100% mortality at 500 mg/kg-day. No effects at 100 mg/kg-day but small numbers of rats were tested. Dose-finding study with no histology ^b .	Hasegawa et al., 2005
rat	18 days (PND 4-21) (gavage)	50 ^e	ND	300	Tremors in 23/24 males and females at 300 mg/kg-day. No clear treatment-related effects at 50 mg/kg-day ^{b,d} .	Hasegawa et al., 2005
rat	14 days (gavage)	500	ND	ND	No clinical signs or other effects but small numbers of rats were tested. Dose-finding study with no histology ^b .	Hasegawa et al., 2005
rat	28 days (gavage)	200	ND	1000	Tremors, hypoactivity, abnormal gait and centrilobular hepatocellular hypertrophy at 1000 mg/kg-day. No reported effects at 200 mg/kg-day ^{b,d} .	Hasegawa et al., 2005
rat	16 weeks ^f (drinking water)	7.5	75	ND	Reduced litter size (19%) and increased incidence of stillbirths.	Exon and Koller, 1982, 1985
rat	16-21 weeks ^g (drinking water)	75	ND	ND	No effects on immune responses ^h or body, liver, spleen or thymus weights. Other endpoints not evaluated.	Exon and Koller, 1983a, 1985

ND = not determined

^amg/kg-day^bEndpoints included clinical signs, body weight, hematology, serum chemistry, organ weights and gross pathology.^cEndpoints included immune responses and behavioral tests.^dEndpoints included histopathology.^eThe only reported effect was tremors in 1/12 females that occurred once on treatment day 9.^fFemale rats were exposed from 3 weeks of age through mating to untreated males at 90 days of age and subsequently through pregnancy and lactation.^gOffspring of female rats that were exposed from 3 weeks of age through mating to untreated males at 90 days of age and subsequently through pregnancy and lactation were continued on treatment for 10-15 weeks.^hTests for humoral immunity, cell-mediated immunity and macrophage function were conducted.

Drinking water studies (Exon and Koller, 1982, 1983a and b, 1985) investigated reproductive and immunological toxicity in rats. There were no effects on immune function in rats that were exposed to 75 mg/kg-day via maternal drinking water during gestation and lactation and subsequently by direct consumption for 10-15 weeks (Exon and Koller, 1983a, 1985). Exposure to 75 mg/kg-day in drinking water during pregnancy and lactation significantly ($p \leq 0.05$) affected litter size (19% reduced) and stillbirths (5% increased) in rats (Exon and Koller 1982, 1985); no effects on litter size occurred at 7.5 mg/kg-day. Therefore, reproductive toxicity as evidenced by decreased litter size and an increase incidence in stillbirths was chosen for the development of the subchronic RfD for 2-chlorophenol based on a NOAEL of 7.5 mg/kg-day (Exon and Koller, 1982).

The NOAEL of 7.5 mg/kg-day is divided by a composite uncertainty factor of 1000 to derive a provisional **subchronic RfD of 8E-3 mg/kg-day**, as follows:

$$\begin{aligned} \text{sRfD} &= \text{NOAEL} / \text{UF} \\ &= 7.5 \text{ mg/kg-day} / 1000 \\ &= \mathbf{0.0075 \text{ or } 8\text{E-3 mg/kg-day}} \end{aligned}$$

The composite UF of 1000 includes a factor of 10 for animal-to-human extrapolation, 10 for interindividual variability and 10 for database deficiencies.

The animal-to-human UF of 10 reflects a factor of three ($10^{1/2}$) for pharmacokinetic differences across species and a factor of three ($10^{1/2}$) for pharmacodynamic considerations.

The intraspecies UF of 10 is used to account for variation in sensitivity within human populations because there is limited information on the degree to which humans of varying gender, age, health status or genetic makeup might vary in the disposition of, or response to, the chemical.

An UF for extrapolation from a LOAEL to a NOAEL is not necessary because a NOAEL was chosen for the point of departure for the derivation for the sRfD.

The UF of 10 for database deficiencies is applied due to the lack of comprehensive reproductive and developmental toxicity studies, including a two-generation reproductive toxicity study and a subchronic study in mice (see below).

Confidence in the key study is low because a limited number of reproductive/developmental endpoints (maternal and pup weight, percent conception, litter size and number of stillborn) were evaluated and the adequacy of the reporting is marginal. Confidence in the database is also low. The database includes 18-day, 28-day and 90-day studies in rats that assessed systemic toxicity and postnatal developmental toxicity at doses that include the range of those tested in the key study. Deficiencies in the database include the lack of comprehensive reproductive and developmental toxicity studies (especially important because reproductive effects have been identified as critical for this chemical) and a subchronic toxicity study longer than 14 days in duration in mice, which appeared to be more sensitive than rats to the subchronic effects of the chemical. In addition, a two-generation reproductive toxicity study is not

available. Considering the levels of confidence in the key study and data base and the lack of supporting data for the critical effects, confidence in the provisional RfD is low.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfC VALUES FOR 2-CHLOROPHENOL

No information is available on the subchronic or chronic inhalation toxicity of 2-chlorophenol, precluding derivation of RfC values for this chemical.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2-CHLOROPHENOL

Weight-of-evidence Classification

Information regarding the carcinogenicity of 2-chlorophenol mainly consists of the negative results of a drinking water study in which rats were exposed to 0, 5, 50 or 500 ppm via maternal consumption during pregnancy and lactation and subsequently by direct consumption for 24 months (Exon and Koller 1983b, 1985). There were no significant increases in tumor incidence, latency or type in either sex, but a definitive conclusion regarding carcinogenicity is precluded by the use of marginal numbers of animals for a cancer bioassay (24-32/sex/dose level) and the apparent lack of a MTD, because the only observed effects (body weight and hematology changes) were not clearly treatment-related or biologically significant.

The ability of 2-chlorophenol to act as a promoter or co-carcinogen was investigated in a study with the known carcinogen ENU (Exon and Koller 1983b, 1985). Male rats that were exposed to 0, 5, 50 or 500 ppm of 2-chlorophenol in drinking water via maternal consumption during pregnancy and lactation and subsequently by direct consumption for 24 months, combined with prenatal exposure to ENU, had increased total tumor incidences and decreased time-to-tumor latencies compared to rats exposed to ENU alone. Another study found that dermal application of 2-chlorophenol promoted the formation of DMBA-initiated skin tumors in mice (Boutwell and Bosch, 1959).

2-Chlorophenol has been studied in several short term *in vitro* and *in vivo* animal studies. 2-Chlorophenol did not induce reverse mutations or DNA-repair in *S. typhimurium* (Haworth et al., 1983; Ono et al., 1992), DNA damage in *E. coli* (DeMarini et al., 1990), or sister-chromatid exchanges in orally-exposed mice (Borzelleca et al., 1985).

In accordance with current EPA cancer guidelines (U.S. EPA, 2005), the available data are inadequate for an assessment of human carcinogenic potential.

Quantitative Estimates of Carcinogenic Risk

Derivation of quantitative estimates of cancer risk for 2-chlorophenol is precluded by the lack of data demonstrating carcinogenicity associated with 2-chlorophenol exposure.

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9-18-2007

Provisional Peer Reviewed Toxicity Values for
2-Methylnaphthalene
(CASRN 91-57-6)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose

PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2-METHYLNAPHTHALENE (CASRN 91-57-6)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

The U.S. Environmental Protection Agency's (EPA) Integrated Risk Information System (IRIS) (U.S. EPA, 2003) for 2-Methylnaphthalene (2-MN) included an RfD of 4×10^{-3} mg/kg-day based on a BMDL₀₅ of 3.5 mg/kg-day and an uncertainty factor of 1000. IRIS also included a carcinogenicity assessment that concluded data were inadequate to assess human carcinogenic potential. ATSDR (2005) derived a chronic MRL of 4×10^{-2} mg/kg-day based on a BMDL₀₅ of 4.3 mg/kg-day in mice (Murata et al., 1997) and an uncertainty factor of 100. ATSDR also had derived an MRL of 7×10^{-2} mg/kg-day for chronic duration oral exposure to 1-MN based on a LOAEL of 71.6 mg/kg-day for increased incidence of alveolar proteinosis in mice (Murata et al., 1993) and an uncertainty factor of 1000.

Updated literature searches for oral noncancer data were conducted from 1983 to 2007. The databases searched were TOXLINE, MEDLINE, CANCERLIT, CCRIS, TSCATS, HSDB, RTECS, GENETOX, DART/ETICBACK, and EMIC/EMICBACK.

This document has passed the STSC quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No relevant human studies were found in the literature.

Animal Studies

Lifetime Exposure

The chronic toxicity of 2-MN was investigated in mice by Murata et al. (1997). Groups of 50 male and 50 female B6C3F1 mice were given diets containing 0, 0.075, or 0.15% 2-MN for 81 weeks. Food consumption and body weight were recorded throughout the experimental period. At necropsy organ weights were recorded for brain, liver, kidney, heart, spleen, lungs, testes, pancreas, thymus, and salivary glands. Gross pathology and histopathology were conducted for these tissues and for adrenals, trachea, stomach, large and small intestines, seminal vesicles, ovaries, uterus, vagina, mammary glands, skeletal muscle, eye, Harderian glands, spinal cord, bone, skin, and other tissues with abnormal appearance. A complete clinical chemistry examination was performed, including hematology, serology, and enzyme analysis. Incidence data were statistically evaluated using a Fisher's exact test and analysis of variance. Continuous endpoints (organ weights, blood, and serum parameters) were evaluated using a multiple comparison post-test with the Dunnett procedure.

Average 2-MN intakes of 50.3 (males) and 54.3 (females) mg/kg-day were calculated from food consumption data reported in Murata et al. (1997) for the 0.075% 2-MN dose group. Similarly, 2-MN intakes for the 0.15% 2-MN dose group were 107.6 (males) and 113.8 (females) mg/kg-day.

Animals at the high dose exhibited slight growth retardation over the entire experimental period. Compared with control animals, final body weights were reduced by 4.5% for females and 7.5% for males. Only the male body weight reductions were statistically significant ($p < 0.01$). Survival was not affected by 2-MN treatment. Pulmonary alveolar proteinosis (PAP) was reported in both treatment groups. PAP was characterized by the appearance of foamy cells in the alveoli and the accumulation of protein and lipid in the lungs. On gross examination, the protenosis appeared as white nodules, 1-5 mm in diameter. Microscopically, the alveolar lumens contained acidophilic amorphous material, foamy cells, and cholesterol crystals. The incidence of PAP was 42.9% among males at 50.3 mg/kg-day and 55.1% among females at 54.3 mg/kg-day; in the high-dose group, the incidence was 46.9% for males and 45.8% for females. The fraction of lung volume affected for individual treated or control animals was not reported. Incidence of this effect in the control animals was 8.2% for males and 10% for females; the effects in control animals were less pronounced than those in the treatment groups. The authors stated that this effect had not been observed previously in more than 5000 B6C3F1 mice housed in the same room and speculated that the control mice may have been exposed to volatilized 1-MN and 2-MN from the treatment groups housed in the same room for this experiment. The authors also concluded that 2-MN was not carcinogenic in this study, although some results were equivocal. In particular, there appeared to be an increase in the incidence of lung tumors. Incidence of lung adenomas and adenocarcinomas, combined, was significantly increased (10/49 vs. 2/49 controls; $p < 0.05$) in male mice at 50.3 mg/kg-day; the increase (6/49) was not significant at the 107.6 mg/kg-day dose. Because they noted association between tumorigenesis and PAP, the authors concluded that PAP was not a risk factor for carcinogenesis in the mouse.

A study by the same group (Murata et al., 1993) investigated the effect of 1-MN in the same strain of mice. Although the study results were published four years apart, the two studies were conducted at the same time and utilized the same control group. The nominal dose groups and endpoints were the same in both studies. The actual exposure levels were somewhat higher for 1-MN (approximately 73 mg/kg-day at 0.075% and 142 mg/kg-day at 0.15%). Results for 1-MN were similar to those for 2-MN, with PAP occurring in both treatment groups. The incidence, however, was slightly lower in the 1-MN treated mice (46% in both males and females at 0.075% 1-MN, and 38 and 35% in males and females, respectively, at 0.15% 1-MN). Unlike 2-MN, this study concluded 1-MN was a lung carcinogen (adenomas and adenocarcinomas) for B6C3F1 mice.

Less-than-Lifetime Exposure

A subchronic 2-MN dietary study in B6C3F1 mice was briefly reported in Murata et al. (1997). This study was preliminary to the chronic study to determine the chronic dosing regimen. Groups of ten mice of each sex each were fed 2-MN for 13 weeks at dietary concentrations of 0, 0.0163, 0.049, 0.147, 0.44, or 1.33%. Estimated doses were: 0, 29.4, 88.4, 265, 794, or 2400 mg/kg-day for males and 0, 31.8, 95.6, 287, 859, or 2600 mg/kg-day for females, respectively. Approximate average doses (across genders) were 0, 31, 92, 276, 827, or 2500 mg/kg-day, respectively (U.S. EPA, 2003). Growth retardation was reported at the three highest dose levels, but was attributed to food refusal. The authors reported no histopathological lesions in any organs of the control or treated animals, although it was unclear whether the lungs were examined. Based on this study, a subchronic NOAEL of 2500 mg/kg-day could be established, because growth retardation accompanied by reduced food consumption in the absence of other effects was not considered an adverse effect.

In a number of studies (Reid et al., 1973; Mahvi et al., 1977; Tong et al., 1981; Griffin et al., 1981, 1982; Warren et al., 1982), intraperitoneal (IP) injection of 2-MN or naphthalene resulted in lung lesions (Clara cell necrosis) similar to those observed in the long-term dietary studies of Murata et al. (1993, 1997).

PAP also was observed in mice following dermal exposure to a mixture of 1-MN and 2-MN for 30 weeks (Murata et al., 1992). A 100% incidence of PAP was observed in 15 B6C3F1 female mice treated dermally with 119 mg MN/kg twice a week. No lesions were observed in the control animals, which were exposed to the acetone vehicle only.

Developmental/Reproductive

No relevant reproductive or developmental data were found in the literature.

Toxicokinetics and Toxicodynamics

Some evidence suggested that mice may be a sensitive species for the type of lung toxicity induced by the methylnaphthalenes. Mice were far more sensitive than rats to acute lung effects arising from exposure to dichloroethylene (Chieco et al., 1981; Krijgheld et al., 1984), bromobenzene (Reid et al., 1973), butylated hydroxytoluene (Kehrer & Witschi, 1980),

naphthalene (Reid et al., 1973; O'Brien et al., 1985), and 2-MN (Griffin et al., 1982). For naphthalene, Buckpitt and Franklin (1989) suggested that the selective lung cytotoxicity in mice may be a result of the high degree of stereoselectivity with which naphthalene is epoxidated in the mouse lung (in vitro microsomal incubations). Rats and humans did not show the same stereoselectivity (Buckpitt and Bahnson, 1986). Together, these data suggested that mice exposed to naphthalene might be more sensitive than humans for this particular endpoint. This conclusion should be considered somewhat speculative, however, particularly because subchronic oral naphthalene studies in mice did not produce lung effects at dose levels producing other adverse effects (142 mg/kg-day for decreased body weight, 286 mg/kg-day for mortality [BCL, 1980b]; 133 mg/kg-day for decreased organ weights [Shopp et al., 1984]). There was, however, a suggestion that tolerance could be developed for this effect in mice (Shopp et al., 1984). Also, the role of metabolic activation in the toxicity of the methylnaphthalenes was less clear than for naphthalene (Griffin and Franklin, 1982; Buckpitt et al., 1984; Buckpitt and Franklin, 1989). Much less was known about species differences in the metabolism of methylnaphthalenes, so no firm conclusions could be made regarding the potential for unique susceptibility mouse to 2-MN-induced lung toxicity. More extensive discussions of the metabolism of naphthalene and the methylnaphthalenes were found in the Toxicological Review of Naphthalene on IRIS (U.S. EPA, 2003) and in Buckpitt and Franklin (1989).

DERIVATION OF PROVISIONAL SUBCHRONIC OR CHRONIC ORAL RfD VALUES FOR 2-METHYLNAPHTHALENE

Fitzhugh and Buschke (1949) evaluated the ability of 2-methylnaphthalene to induce cataract formation in rats. While no cataracts were found in a group of 5 weanling F344 rats fed a diet of 2% 2-MN (equivalent to 2000 mg/kg-day) for at least 2 months, cataracts were detected in rats fed an equivalent concentration of naphthalene. Evaluation of this study was limited by the lack of experimental details. In this study, 2000 mg/kg-day was an apparent NOAEL for cataract formation.

Evaluation of the Murata et al. (1997) subchronic data was limited by inadequate reporting of study results. It appeared that very few potential endpoints were considered. In its evaluation of these data, IRIS (U.S. EPA, 2003) concluded that 92 mg/kg-day and 276 mg/kg-day (averaged between genders) were the NOAEL and LOAEL, respectively, for reduced weight gain in rats, apparently rejecting the study authors' attribution of these effects to food refusal. However, the study report did not clearly identify what organs were examined or other potential effects were considered. This raised the possibility that other effects might have resulted from subchronic dosing that were not observed. Because very few details of the data or methods for the subchronic study were reported by Murata et al. (1997) and because it was unclear whether the reduced weight gain resulted from treatment with 2-MN, these data were considered inadequate for derivation of a subchronic p-RfD. As a result, the chronic RfD of 4×10^{-3} mg/kg-day on IRIS was selected as the subchronic p-RfD.

The IRIS chronic RfD (U.S. EPA, 2003) was based on a BMDL₀₅ of 3.5 mg/kg-day for 5% extra risk of pulmonary alveolar proteinosis in male and female mice exposed to 2-MN in the diet for 81 weeks (Murata et al., 1997). A total UF of 1000 was applied to this effect level: 10

for interspecies differences (UF_A : animal to human); 10 for intraspecies variation (UF_H : human variability); and 10 for deficiencies in the database (UF_D).

The subchronic p-RfD for 2-MN was calculated using the same factors as follows:

$$\begin{aligned}\text{subchronic p-RfD} &= \text{BMDL}_{05} \div UF \\ &= 3.5 \text{ mg/kg-day} \div 1000 \\ &= 0.004 \text{ mg/kg-day} = 4 \times 10^{-3} \text{ mg/kg-day}\end{aligned}$$

In the derivation of the chronic RfD, IRIS (U.S. EPA, 2003) noted that, in addition to the uncertainties noted above, there was model uncertainty owing to the lack of actual dose-response information or mode of action information near a dose where the point of departure was estimated. The responses in 2-MN exposed animals suggested a continuation of the plateau into the lower exposure region, so using a linear model might have provided a higher benchmark dose than was appropriate. In addition, while BMDS was used to generate a lower bound on the estimated benchmark dose, the lower bound probably described too narrow a confidence limit on the benchmark dose. This was because the uncertainty in the data set could not be adequately described without the high dose responses.

CONFIDENCE IN THE SUBCHRONIC ORAL RfD

The principal study for the p-RfD (Murata et al., 1997) examined a comprehensive number of endpoints, including extensive histopathology, and tested two dietary dose levels using sufficient numbers (50/gender/group) of B6C3F1 mice. Confidence in the study was medium because there was potential confounding from possible inhalation exposure of controls to volatilized 2-MN and 1-MN. This added some uncertainty to the dose-response relationship between oral exposure to 2-MN and pulmonary alveolar proteinosis described by the results. Confidence in the oral toxicity database was low. No epidemiology studies or case reports were located which examined the potential effects of human exposure to 2-MN. Only mice had been examined in adequate animal studies on toxicity from repeated exposure to 2-MN. No assays of developmental toxicity, reproductive toxicity, or neurotoxicity following oral exposure to 2-MN were available. Confidence in the oral RfD was low, principally due to the low confidence in the database.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC OR CHRONIC INHALATION RfC VALUES FOR 2-METHYLNAPHTHALENE

A provisional inhalation RfC could not be derived for 2-MN because data on adverse health effects following inhalation exposure were lacking for humans and animals. Without sufficient pharmacokinetic data and information to rule out portal-of-entry effects, there was no basis to support a route-to-route extrapolation from the oral data, even if they otherwise were considered sufficient.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2-METHYLNAPHTHALENE

Weight-of-Evidence Descriptor

Using the draft revised guidelines for carcinogen risk assessment (U.S. EPA, 1999), the IRIS assessment (U.S. EPA, 2003) concluded the data were inadequate for an assessment of human carcinogenic potential of 2-MN. This conclusion was based on the absence of data concerning the carcinogenic potential of 2-MN in humans, by any route of exposure, and limited, equivocal oral evidence in animals. Updated literature searches for this assessment identified no relevant data other than those already considered for the IRIS assessment. Based on the revised guidelines for carcinogen risk assessment (U.S. EPA, 2005), the equivalent carcinogenicity descriptor would be “*Inadequate Information to Assess Carcinogenic Potential.*”

Quantitative Estimates of Carcinogenic Risk

Quantitative estimates of cancer risk for 2-MN could not be derived because no data demonstrating carcinogenicity associated with 2-MN exposure were identified.

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Provisional Peer Reviewed Toxicity Values for

2-Nitrophenol
(CASRN 88-75-5)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
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U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR 2-NITROPHENOL (CASRN 88-75-5)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Neither a reference dose (RfD), reference concentration (RfC), nor carcinogenicity assessment is available for 2-nitrophenol in the Integrated Risk Information System (IRIS) database (U.S. EPA, 2007), the Health Effects Assessment Summary Table (HEAST) (U.S. EPA, 1997), or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2006). The Chemical Assessments and Related Activities (CARA) database (U.S. EPA, 1991, 1994a) lists a Health Effects Assessment (HEA) (U.S. EPA, 1987) and a Health and Environmental Effects Profile (HEEP) (U.S. EPA, 1985) for Nitrophenols in which limited toxicity data for 2-nitrophenol are available. An Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Nitrophenols (2-Nitrophenol and 4-Nitrophenol) (ATSDR, 1992) also includes only limited toxicity data for 2-nitrophenol. Neither the American Conference of Governmental Industrial Hygienists (ACGIH, 2006), the National Institute of Occupational Safety and Health (NIOSH, 2006) nor the Occupational Safety and Health Administration (OSHA, 2006) has adopted occupational exposure limits for 2-nitrophenol. Health assessments for 2-nitrophenol are not available from CalEPA (2006) or the International Agency for Research on Cancer (IARC, 2006). Pertinent data was found for 2-nitrophenol after examining the Concise International Chemical Assessment Document (CICAD) for mononitrophenols (WHO, 2000). Relevant information for 2-nitrophenol from the National Toxicology Program (NTP, 2006) is limited to genotoxicity assays.

Literature searches covering the time period 1960's to August, 2006 were conducted in PUBMED, TOXLINE, and DART/ETIC to identify information relevant to 2-nitrophenol. TOXCENTER was searched for the time period August, 2001 to August 2006. Databases

searched without date limitations included TSCATS/TSCATS2, CCRIS, GENETOX, HSDB and RTECS. Search of Current Contents encompassed May to August, 2006.

REVIEW OF PERTINENT DATA

Human Studies

No data were located regarding the toxicity or carcinogenicity of 2-nitrophenol in humans following oral or inhalation exposure.

Animal Studies

Oral Exposure. Available repeated-dose oral studies consist of two limited 28-day gavage studies (Andrae et al., 1981; Koerdel et al., 1981; both in German) performed to evaluate OECD guideline 407 and a range-finding developmental toxicity study (IRDC, 1990).

Andrae et al. (1981) administered 2-nitrophenol to groups of Sprague-Dawley rats (10/sex/dose) at gavage doses of 0, 70, 210 or 630 mg/kg-day for 28 days. Because the original German report of this study was not available, information from the CICAD for mononitrophenols (WHO, 2000) was used to summarize the findings. Mid- and high-dose animals exhibited what was described by the WHO (2000) as locomotor inhibition for approximately 2 hours postdosing. Mortality rates were 1/10 in mid-dose males and 4/10 and 6/10 in high-dose males and females, respectively. Gross and histopathological examinations revealed pale liver in 7/20 low-dose rats (not reported by sex), hydropic liver cell swelling in 4/10 and 0/10 high-dose males and females, respectively, and vascular congestion of the liver in all high-dose male and female rats that died prior to terminal sacrifice. Fatty degeneration of the liver was noted in 6/20 control animals, 14/20 low-dose and 13/20 mid-dose rats, but not in high-dose rats. Other treatment-related effects, noted only at the highest dose level, included significantly increased alanine aminotransferase activity in males (data not reported), increased nephrosis in 2 and 5 males and females, respectively, testicular atrophy (1 male) and decreased spermatogenesis (2 males), and follicular atresia (4 females). This report did not contain information on hematological effects. WHO (2000) concluded that a NOAEL could not be determined for this study due to “unclear effects in the liver.”

Koerdel et al. (1981) administered 2-nitrophenol to groups of rats (5/sex/dose) at gavage doses of 0, 22, 67 or 200 mg/kg-day for 28 days. The summary from WHO (2000) was used as the source of study details because the original study was not available. Reported treatment-related effects included decreased food intake in high-dose males and mid- and high-dose females, non-significantly depressed final body weight in all dosed animals, decreased absolute liver and kidney weights in mid-dose groups, increased relative testes weight in low- and mid-dose males (decreased in high-dose males) and increased absolute and relative adrenal weight in all dosed groups. Hematology, clinical chemistry and histopathological examinations gave no indication of treatment-related effects. The study did not show a clear dose-response relationship for any of the endpoints examined.

In a range-finding developmental toxicity study, groups of Charles River COBS CD rats (5 dams/group) were administered 2-nitrophenol (in corn oil) at gavage doses of 0, 50, 125, 250, 500, or 1000 mg/kg-day on days 6-15 of gestation (IRDC, 1990). Body weights were determined during the treatment period and clinical signs were noted. Uterine examinations were performed on gestation day 20. A single high-dose dam died, but cause of death was not determined. Excessive salivation was observed in two high-dose dams. Mean maternal body weight gains in the 0, 50, 125, 250, 500 and 1000 mg/kg-day dose groups were 8, 7, 5, 6, 1 and -8 grams, respectively, for the initial 4 days of treatment (gestation days 6-9) and 52, 56, 54, 55, 45 and 39 grams, respectively, for the entire treatment period (gestation days 6-15). The appearance and behavior of the 50 mg/kg-day group of dams were comparable to the control group. Dose-related increases in the incidence of yellow staining around the nose, mouth and anogenital area were observed at doses ≥ 125 mg/kg-day. Dose-related increases in the incidence of darkly colored urine (probably due to the presence of the test chemical) occurred at doses ≥ 250 mg/kg-day. An increase in the number of early resorptions was observed in the highest dose group (2.3 versus 1.2 in controls), resulting in mean postimplantation loss of 13.8% compared to 8.2% in controls (statistical significance not reported). Among dams surviving until necropsy, no biologically significant treatment-related effects were seen. There were no biologically significant treatment-related effects on mean number of viable fetuses, implantations or *corpora lutea*. No data on hematological parameters were included in this study. This study assessed a limited number of potential adverse endpoints and is therefore of limited usefulness for risk assessment.

Inhalation Exposure. Available information for repeated inhalation exposure is restricted to results of a single 28-day study (Hazleton Laboratories, 1984). Groups of 7-week-old Sprague-Dawley rats (15/sex/group) were exposed to 2-nitrophenol vapors at target concentrations of 0, 5, 30 or 60 mg/m³ for 6 hours/day, 5 days/week for 4 weeks. All rats were subjected to ophthalmoscopic examinations prior to initiation of exposures and immediately preceding terminal sacrifice. Each animal was observed twice daily (pre- and postexposure during the week; morning and afternoon on weekends) for mortality and morbidity. Clinical signs and body weights and weight gains were assessed throughout the study. Following the 11th and 20th exposures, blood was collected by orbital sinus puncture from 10 rats/sex/group and analyzed for methemoglobin concentrations. At termination of the study (day 29), blood was collected via the abdominal aorta from 10 anesthetized rats/sex/group for hematology and serum chemistry. At necropsy, all rats were subjected to comprehensive gross examinations and organ weights were recorded. Comprehensive histopathological examinations were performed on 10 rats/sex in the 0 and 60 mg/m³ exposure groups. Nasal turbinates were examined histopathologically in 10 rats/sex of each exposure group.

Overall mean analytical concentrations deviated from the target concentrations by 0.0, +8.3 and +2.5% for the 5, 30 and 60 mg/m³ exposure groups, respectively (Hazleton Laboratories, 1984). The aerosol content of the exposure chambers was not significantly different from that present in room air. No significant exposure-related ocular lesions were apparent in any of the rats. No animals died during the study. No apparent exposure-related trends in clinical signs were apparent with the exception of yellow stains on the fur of all 2-nitrophenol exposed animals. There were no statistically significant exposure-related effects on mean body weight or weight gain. A statistically significant increase in methemoglobin

levels was noted in male and female rats of the 5 mg/m³ group analyzed on day 15 of the study. However, when animals were analyzed on day 28, the methemoglobin levels were similar to controls. No statistically significant increases were found in the higher dose groups. The change, compared with controls, in methemoglobin levels in treated animals of the low dose groups, while exhibited statistical significance, was not considered biologically significant. Hematology and clinical chemistry findings were unremarkable. Gross pathology revealed no consistent exposure-related trends. Small increases in liver weight, liver/brain weight ratio and spleen/brain weight ratio were seen in the 5 mg/m³ group females, but were not observed in females at higher doses or in any of the treated males. Histopathological examinations revealed squamous metaplasia in epithelium of the nasoturbinates and maxilloturbinates in 1/10, 0/10, 10/10 and 10/10 male rats and 1/10, 1/10, 9/10 and 10/10 female rats of the 0, 5, 30 and 60 mg/m³ exposure groups, respectively. No other apparent exposure-related effects were observed. On the basis of the nasal lesions, this study identified a NOAEL of 5 mg/m³ and a LOAEL of 30 mg/m³ for 2-nitrophenol in rats.

Other Studies

Limited genotoxicity data are available for 2-nitrophenol. The chemical produced negative results in the Ames test with *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 both in the presence and absence of rat liver S9 metabolic activation (Chiu et al., 1978; Dellarco and Prival, 1989; Haworth et al., 1983; Kawai et al., 1987; Koerdel et al., 1981; Massey et al., 1994; Shimizu and Yano, 1986; Suzuki et al., 1983). 2-Nitrophenol did not induce DNA breakage in λ phage DNA (Yamada et al., 1987) or increase reversions from streptomycin dependence to independence in *Escherichia coli* strain Sd-4-73 (Szybalski, 1958). Negative results were reported for mutagenic activity in post-meiotic and meiotic germ cells of male *Drosophila melanogaster* exposed to 2-nitrophenol via feeding (400-500 ppm) or injection (2500 or 5000 ppm) (Foureman et al., 1994).

2-Nitrophenol did not exhibit skin tumor-promoting action in mice receiving dermal applications of a 20% solution twice weekly for 12 weeks (Boutwell and Bosch, 1959).

In rats and mice administered single oral doses of 2-nitrophenol, calculated LD₅₀ values were 2830 and 1300 mg/kg, respectively (Vernot et al., 1977). No information was located regarding the toxicity of 2-nitrophenol following acute inhalation exposure.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC RfD VALUES FOR 2-NITROPHENOL

Oral studies of 2-nitrophenol are limited to two 28-day studies from the German literature available only as brief summaries in WHO (2000) and a range-finding developmental toxicity study. None of these studies appear to have been adequate to derive NOAEL or LOAEL values. The lack of adequate oral data for humans or animals precludes the derivation of a provisional subchronic or chronic RfD for 2-nitrophenol.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfC VALUES FOR 2-NITROPHENOL

Subchronic p-RfC

Results of the only available repeated exposure (28-day) inhalation study of 2-nitrophenol (Hazleton Laboratory, 1984) provide marginally adequate information in rats to derive a provisional subchronic RfC for 2-nitrophenol. This study identified significantly increased incidences of squamous metaplasia of the nasal epithelium in rats as the critical effect following 4 weeks of exposure to 2-nitrophenol vapors for 6 hours/day, 5 days/week. The lowest concentration of 2-nitrophenol associated with squamous metaplasia of the nasal epithelium was 30 mg/m³ in both male and female rats; the associated NOAEL was 5 mg/m³. Because the NOAEL and LOAEL represent essentially 0 and 100% response, respectively, it is not feasible to apply meaningful benchmark dose analysis to the data set. Therefore, the NOAEL of 5 mg/m³ was selected as the point of departure for deriving a subchronic RfC for 2-nitrophenol.

The NOAEL of 5 mg/m³ from intermittent exposure was adjusted to account for a continuous exposure scenario as follows:

$$\begin{aligned}\text{NOAEL}_{[\text{ADJ}]} &= \text{NOAEL} \times 6 \text{ hours}/24 \text{ hours} \times 5 \text{ days}/7 \text{ days} \\ \text{NOAEL}_{[\text{ADJ}]} &= 5 \text{ mg/m}^3 \times 6/24 \times 5/7 = 0.89 \text{ mg/m}^3\end{aligned}$$

According to U.S. EPA (1994b) methodology for respiratory effects of a category 1 gas (a systemic toxicant without significant portal of entry (lung) effects), such as 2-nitrophenol the NOAEL_[HEC] (human equivalent concentration) is calculated by multiplying the NOAEL_[ADJ] for upper respiratory effects by the regional gas dose ratio for extrathoracic effects (RGDR_{ET}). The default RGDR_{ET} is calculated according to the following equation:

$$\text{RGDR}_{\text{ET}} = \frac{\left[\frac{\dot{V}_E}{\text{SA}_{\text{ET}}} \right]_A}{\left[\frac{\dot{V}_E}{\text{SA}_{\text{ET}}} \right]_H} \quad (\text{Equation 4-18; U.S. EPA 1994b})$$

where:

\dot{V}_E = minute volume (cm³/minute)

SA_{ET} = surface area of the extrathoracic region (cm²), and

A, H = subscripts denoting laboratory animal and human, respectively.

Default surface area values for the extrathoracic respiratory region are 15 cm² for the rat and 200 cm² for the human (U.S. EPA (1994b). For the male Sprague-Dawley rat, a reference inhalation rate of 0.27 m³/day (270,000 cm³/day; U.S. EPA, 1988, standard default) produces a minute volume of 187.5 cm³/min (270,000 cm³/day ÷ 1440 min/day). The default minute volume for the human is 13,800 cm³/min (13.8 L/min or 20 m³/day; U.S. EPA, 1994b). Therefore:

$$RGDR_{PU} = \frac{\left[\frac{187.5}{15} \right]_A}{\left[\frac{13,800}{200} \right]_H} = 0.1812$$

The NOAEL_[HEC] is derived as follows:

$$\text{NOAEL}_{[\text{HEC}]} = \text{NOAEL}_{[\text{ADJ}]} \times \text{RGDR}_{\text{ET}} = 0.89 \text{ mg/m}^3 \times 0.1812 = 0.1613 \text{ mg/m}^3$$

The **subchronic p-RfC of 5E-4 mg/m³** based on squamous metaplasia of the nasal epithelium in rats (Hazleton Laboratories, 1984) is derived by dividing the NOAEL_[HEC] of 0.16 mg/m³ by a composite uncertainty factor (UF) of 300, which includes factors of 3 for interspecies extrapolation, 10 for interindividual human variability and 10 for data base deficiencies.

A 3-fold UF is used to account for uncertainty in extrapolating from laboratory animals to humans (i.e., interspecies variability). No information is available regarding the toxicity of 2-nitrophenol in humans. No comparative information is available regarding the toxicokinetics or toxicodynamics of 2-nitrophenol in animals and humans. However, the default dosimetric calculation for deriving an HEC accounts for the uncertainty in the variability in toxicokinetics of humans and rats. A 3-fold UF is applied to account for uncertainty in species differences for toxicodynamics (U.S. EPA, 1994b).

A 10-fold UF is used to account for variation in sensitivity among members of the human population (i.e., interindividual variability). This UF was not reduced due to the lack of human inhalation exposure data.

A 10-fold UF is used to account for uncertainty associated with data base deficiencies. A single 28-day inhalation toxicity study in one animal species (rat) is available (Hazleton Laboratories, 1984). The data base lacks studies of subchronic and chronic toxicity, inhalation neurotoxicity, developmental toxicity and reproductive toxicity (including 2-generation reproductive toxicity). Although the principal study (Hazleton Laboratories, 1984) was only a 28-day study (less than subchronic duration), the minor nature of the effects observed suggests that the 10-fold database UF is adequate to capture the uncertainties associated with use of the less-than-subchronic study in this instance.

Confidence in the principal study (Hazleton Laboratories, 1984) is low-to-medium. The study included comprehensive gross and histopathologic assessments. A major limitation of this study is the less-than-subchronic study duration of 28 days. Confidence in the data base is low because the data base lacks studies of subchronic and chronic toxicity, inhalation neurotoxicity, and developmental and reproductive toxicity (including 2-generation reproductive toxicity). Reflecting low-to-medium confidence in the principal study and low confidence in the data base, confidence in the provisional subchronic RfC is low.

Chronic p-RfC

The lack of adequate subchronic or chronic inhalation data for humans or animals precludes the derivation of a provisional chronic RfC for 2-nitrophenol. Use of the 28-day study (Hazleton Laboratories, 1984) was rejected because of uncertainties in exposure duration and toxicokinetics and dynamics in humans, and a lack of reproduction/developmental studies and which would result in five areas of uncertainties. According to the uncertainty in hematological effects which could become apparent in a chronic study, the database is insufficient to support derivation of chronic p-RfC (U.S. EPA, 1994b).

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR 2-NITROPHENOL

Weight-of-Evidence Descriptor

No information was located regarding the carcinogenicity of 2-nitrophenol in humans. No lifetime assessments were located regarding the carcinogenicity of inhaled or ingested 2-nitrophenol in animals. 2-Nitrophenol did not exhibit skin tumor-promoting action in mice receiving dermal applications twice weekly for 12 weeks (Boutwell and Bosch, 1959). Available genotoxicity assays of 2-nitrophenol indicate that the chemical is not genotoxic (Chiu et al., 1978; Dellarco and Prival, 1989; Foureman et al., 1994; Haworth et al., 1983; Kawai et al., 1987; Koerdel et al., 1981; Massey et al., 1994; Shimizu and Yano, 1986; Suzuki et al., 1983; Szybalski, 1958; Yamada et al., 1987). In accordance with U.S. EPA (2005) cancer guidelines, there is *inadequate information to assess carcinogenic potential* for 2-nitrophenol, based on the lack of human or animal carcinogenicity data.

Quantitative Estimates of Carcinogenic Risk

There are no human or animal data from which to derive an oral slope factor or inhalation unit risk for 2-nitrophenol.

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Hierarchy of toxicity information: IRIS, PPRTV, ATSDR, Cal EPA, and HEAST		Blue text = PPRTV attached	— = No info			
Chemical	CASRN	RfD	RfC	CSF _o	CSF _i	Notes
Trimethylbenzene, 1,2,4-	95-63-6	PPRTV: no value		PPRTV: no value	—	IUR - PPRTV: no value
Trimethylbenzene, 1,3,5-	108-67-8	—	—	—	—	
Dichlorobenzene, 1,4-	106-46-7	ATSDR		CalEPA	CalEPA	
Chlorophenol, 2-	95-57-8		PPRTV: no value		—	IUR - PPRTV: no value
Hexanone, 2-	591-78-6	—	—		—	
Methylnaphthalene, 2-	91-57-6		IRIS Discussion			
Methylphenol, 2-	95-48-7		IRIS Message			
Nitrophenol, 2-	88-75-5	PPRTV: no value	Decision pending		—	IUR - PPRTV: no value
DDT, 4,4- (Dichlorodiphenyltrichloroethane, 4,4-)	50-29-3		—			
Chloroaniline, 4-	106-47-8		—		—	
Methylphenol, 4-	106-44-5	—	—			
Nitrophenol, 4-	100-02-7	—	IRIS Message		—	
Acenaphthylene	208-96-8	—	—			
Acetone	67-64-1		ATSDR			
Aluminum	7429-90-5			PPRTV: no value	—	IUR - PPRTV: no value
Arsenic	7440-38-2		CalEPA			
Barium	7440-39-3		HEAST			
Benzo(a)pyrene	50-32-8				CalEPA	
Bis(2-ethylhexyl)phthalate	117-81-7		—			
Bromodichloromethane	75-27-4		—		CalEPA	
Cadmium	7440-43-9		—			
Chloroform	67-66-3		ATSDR			
Copper	7440-50-8	HEAST	—			
Cyanide	57-12-5		—			
Delta-BHC (delta-Hexachlorocyclohexane; delta-HCH)	319-86-8	—	—			
Dibenzofuran	132-64-9	PPRTV: Appendix	PPRTV: no value			
Dieldrin	60-57-1		—			
Endosulfan Sulfate	1031-07-8	—		PPRTV: no value	—	
Endrin Aldehyde	7421-93-4		—	—	—	

Endrin Ketone	53494-70-5		—	—	—	
Endosulfan I (alpha-Endosulfan; Thiodan I)	959-98-8		—	—	—	
Endosulfan II (beta-Endosulfan; Thiodan II)	33213-65-9		—	—	—	
Heptachlor	76-44-8		—			
Heptachlor epoxide	1024-57-3		—			
Hexachlorobenzene	118-74-1		IRIS Message			
Manganese	7439-96-5			—		
Methylene chloride	75-09-2		ATSDR			
Molybdenum	7439-98-7		—			
Naphthalene	91-20-3				CalEPA	
Nickel	7440-02-0		ATSDR	—	CalEPA	
n-Nitroso-di-n-propylamine	621-64-7	—	—		CalEPA	
Phenanthrene	85-01-8	—	—			
Phenol	108-95-2		CalEPA			
Silver	7440-22-4		—			
Strontium	7440-24-6		—		—	
Styrene	100-42-5				—	
Tetrachlorethylene	127-18-4		ATSDR			
Thallium	7440-28-0		—		—	
Vanadium	7440-62-2		—	—	—	
Zinc	7440-66-6		—			



Superfund Technical Support Center

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U.S. Environmental Protection Agency

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April 16, 2008

Michael Sivak
U.S. EPA, Region 2

ASSISTANCE REQUESTED: PPRTVs for 4,4'-DDD, Acenaphthene, Aldrin, Aluminum, Ammonia, and Benzo(a)anthracene (Onondaga Lake)

ENCLOSED INFORMATION: Attachment 1: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR p,p'-DICHLORODIPHENYLDICHLOROETHANE (p,p'-DDD) (CASRN 72-54-8)**

Attachment 2: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR ACENAPHTHENE (CASRN 83-32-9) Derivation of a Chronic Inhalation RfC**

Attachment 3: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR ACENAPHTHENE (CASRN 83-32-9) Derivation of an Oral Slope Factor**

Attachment 4: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR ACENAPHTHENE (CASRN 83-32-9) Derivation of a Provisional Inhalation Unit Risk**

Attachment 5: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR ALDRIN (CASRN 309-00-2)**

Attachment 6: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR ALUMINUM (CASRN 7429-90-5)**

Attachment 7: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR AMMONIA (CASRN 7664-41-7)**

Attachment 8: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR BENZ[a]ANTHRACENE (CASRN
56-55-3)**

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (8)

cc: STSC Files

Provisional Peer Reviewed Toxicity Values for
p,p'-Dichlorodiphenyldichloroethane (*p,p'*-DDD)
(CASRN 72-54-8)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose

PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
p,p'-DICHLORODIPHENYLDICHLOROETHANE (*p,p'*-DDD) (CASRN 72-54-8)**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

No verified chronic reference dose (RfD) or reference concentration (RfC) for *p,p'*-dichlorodiphenyldichloroethane (*p,p'*-DDD) is available on the U.S. Environmental Protection Agency's (EPA) Integrated Risk Information System (IRIS) (U.S. EPA, 2007), Drinking Water Standards and Health Advisories list (U.S. EPA, 2006) or Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997). The U.S. EPA's Chemical Assessments and Related Activities (CARA) list (U.S. EPA, 1991, 1994) does not indicate any documents relating to the noncancer health effects of *p,p'*-DDD. The Agency for Toxic Substances Disease and Registry (ATSDR, 2002) prepared a toxicological profile for dichlorodiphenyltrichloroethane (DDT), dichlorodiphenyldichloroethylene (DDE) and DDD. ATSDR did not develop any Minimal Risk Levels (MRLs) for *p,p'*-DDD, but no explanation was provided. The American Conference of Governmental Industrial Hygienist (ACGIH, 2006), Occupational Safety and Health Administration (OSHA, 2006) and National Institute for Occupational Safety and Health (NIOSH, 2006) have not adopted occupational exposure limits for *p,p'*-DDD. A NIOSH Special Occupational Hazard Review document, two International Agency for Research on Cancer monographs (IARC, 1974, 1991), the National Toxicology Program status report (NTP, 2006) and two World Health Organization documents (WHO, 1979, 1989) were consulted for relevant information.

A cancer weight-of-evidence classification and an oral slope factor for *p,p'*-DDD are available on IRIS (U.S. EPA, 2007). The cancer assessment, verified in 1988, classifies *p,p'*-DDD in category B2 (probable human carcinogen) under U.S. EPA (1986) Guidelines for Carcinogen Assessment, based on lung tumors in female mice, lung and liver tumors in male

mice, and thyroid tumors in male rats after dietary exposure. IRIS (U.S. EPA, 2007) reports an oral slope factor of 0.24 per mg/kg-day, and drinking water unit risk of 6.9 E-6 per µg/L, based on liver tumors in male mice exposed via the diet by Tomatis et al. (1974). The IRIS carcinogenicity assessment for *p,p'*-DDD is derived from the Hazard Assessment Report on DDT, DDD and DDE (U.S. EPA, 1980) and Carcinogen Assessment Group's Calculation of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE, and DDD (TDE) (U.S. EPA, 1985a). IRIS does not report an inhalation unit risk for *p,p'*-DDD. *p,p'*-DDD is not included in the NTP's 11th Report on Carcinogens (NTP, 2005). IARC (1991) classifies DDT and associated compounds (including *p,p'*-DDD) in Group 2B (possibly carcinogenic to humans), citing inadequate evidence in humans, but sufficient evidence in animals for the carcinogenicity of DDT. The present document does not include a cancer assessment for *p,p'*-DDD, as one is available on IRIS.

To identify toxicological information pertinent to the derivation of provisional toxicity values for *p,p'*-DDD, references from the 2002 ATSDR Toxicological Profile for DDT, DDE and DDD were screened for publications pertinent to the toxicity of *p,p'*-DDD. Update searches were conducted in January, 2007 for literature dating from 2001 to 2007 using the following databases: MEDLINE, TOXLINE Special, and DART/ETIC (2001-2007); BIOSIS (2000-2007); TSCATS, CCRIS, GENETOX, HSDB, RTECS (not date limited); and Current Contents (previous 6 months).

REVIEW OF PERTINENT DATA

Human Studies

Human studies of *p,p'*-DDD include one subchronic study with a human volunteer (Morgan and Roan, 1971), several studies of occupational exposure to technical grade DDT, which contains *p,p'*-DDD (Kolmodin et al., 1969; Laws et al., 1967, 1973; Morgan and Lin, 1978; Morgan et al., 1980; Ortelee, 1958; Poland et al., 1970; Wong et al., 1984), and several investigations of associations between reproductive effects and *p,p'*-DDD levels in biological fluids (Saxena et al., 1980, 1981, 1983; Pines et al., 1987; Dalvie et al., 2004; Pant et al., 2004; Perry et al., 2006). Due to the low number of study subjects, concurrent exposures to other chemicals, and difficulty in distinguishing between biological levels of *p,p'*-DDD resulting from exposure and levels resulting from human metabolism of DDT or DDE, data from the available human studies were not considered useful for derivation of provisional toxicity values.

In a study of the toxicokinetics of DDT and its metabolites (including *p,p'*-DDD), an adult male volunteer ingested 5 mg/day of *p,p'*-DDD for 81 days (Morgan and Roan, 1971). The pesticide was mixed with vegetable oil, emulsified with gum arabic and water and taken with meals (no further detail on dosing was provided). Assuming a reference body weight of 70 kg (U.S. EPA, 1988a), the intake of *p,p'*-DDD was 0.071 mg/kg-day. Before, during and after the treatment period, the man was given a battery of hematological and clinical biochemical tests (frequency and nature of testing not reported). No abnormalities were detected. Serum and adipose levels of *p,p'*-DDD rose steadily during the exposure period, peaking at exposure termination at almost 80 ppb in serum and more than 4 ppm in adipose (based on visual

examination of data presented graphically). After exposure was withdrawn, levels in both serum and adipose declined rapidly. Measurements taken 180 days after exposure termination showed no detectable *p,p'*-DDD in serum and levels reduced to almost 1 ppm in adipose. Although no adverse effects on hematological and clinical chemistry endpoints were observed, details of the test endpoints, frequency, and results were not reported, and other endpoints were not assessed; thus the administered dose cannot be considered a NOAEL. Furthermore, the study was conducted on only one volunteer, limiting the usefulness of the data.

Several epidemiology studies of workers exposed to technical grade DDT were located (Kolmodin et al., 1969; Laws et al., 1967, 1973; Morgan and Lin, 1978; Morgan et al., 1980; Ortelee, 1958; Poland et al., 1970; Wong et al., 1984). Technical grade DDT consists of a mixture of *p,p'*-DDT (77.1%), *o,p'*-DDT (14.9%), *p,p'*-DDE (4.0%), *p,p'*-DDD (0.3%), *o,p'*-DDE (0.1%), *o,p'*-DDD (0.1%) and unidentified compounds (3.5%) (U.S. EPA, 1980). Exposure was primarily via the inhalation and dermal routes, but some oral exposure probably occurred as well. In most of the studies, workers were exposed to a variety of other compounds in addition to technical grade DDT. Because of the mixed exposures, these studies do not provide any useful information on health effects of *p,p'*-DDD in humans.

Measurements of *p,p'*-DDD in biological fluids have been used to evaluate potential effects on female reproductive function. Saxena et al. (1980, 1981, 1983) studied the levels of organochlorine insecticides in maternal blood and placenta in cases of stillbirth, premature labor and delivery, spontaneous abortion, and normal full-term delivery among patients in India. The levels of *p,p'*-DDD in maternal blood, placentas and cord blood of stillbirths were not significantly different from the levels in normal full-term deliveries (Saxena et al., 1983); however, there were few participants in this study (9 stillbirths and 27 full-term deliveries). Maternal blood and placental levels of *p,p'*-DDD were significantly ($p < 0.001$) higher in cases of preterm labor and spontaneous abortion when compared with full-term deliveries; levels of *p,p'*-DDT, *p,p'*-DDE, lindane, and aldrin were also significantly higher (Saxena et al., 1980, 1981). However, due to the small numbers of study participants (< 25 cases and ≤ 25 controls) and the confounding role of other pesticides, a causal relationship between *p,p'*-DDD and reproductive effects cannot be established from these data. As reported in an abstract, Perry et al. (2006) evaluated the association between serum levels of DDT and its metabolites (not specified) with urinary levels of progesterone and estrogen, and menstrual cycle characteristics in 287 newly-married women who were trying to conceive. Data were collected from each woman for 1 year or until conception. After adjustment for potential confounders, increased serum *p,p'*-DDD levels were associated with decreased urinary levels of pregnanediol-3-glucuronide across all menstrual cycle days; however, the authors did not present statistical analysis of the results. No other associations with *p,p'*-DDD were reported in the abstract. In these studies (Saxena et al., 1980, 1981, 1983; Perry et al., 2006), it is not known whether the *p,p'*-DDD detected in the subjects was derived from direct exposure to *p,p'*-DDD or from metabolism of DDT or DDE. In addition, since the subjects in these studies also had detectable levels of other compounds (including DDT and its other metabolites), the degree to which the observed effects can be attributed to *p,p'*-DDD is uncertain.

Because there are indications that *p,p'*-DDD may have antiandrogenic effects, several studies have examined the association between male reproductive function and *p,p'*-DDD in

biological fluids. Pines et al. (1987) studied the possible associations between organochlorine insecticide exposures and reproductive function in men by comparing concentrations of these compounds in the serum of 29 infertile and 14 fertile men from the general Israeli population. Serum concentrations of *p,p'*-DDD alone or in combination with *p,p'*-DDT and *p,p'*-DDE were statistically significantly ($p \leq 0.05$) higher in infertile men than in fertile men. Correlations between semen characteristics (sperm count, motility, morphology) and the serum concentrations of these compounds, however, were not significant. Dalvie et al. (2004a) evaluated the effects of DDT and its metabolites on semen, fertility and sexual function in a cross-sectional study of 60 anti-malaria workers in South Africa. There were no statistically significant associations between serum levels of *p,p'*-DDD and sperm count, density or morphology; self-reported problems with sexual function; prevalence of genital abnormalities on physical examination; or number of pregnancies fathered. In a companion study, Dalvie et al. (2004b) reported that levels of estradiol and testosterone were significantly ($p \leq 0.05$) increased with higher serum levels of *p,p'*-DDD. Pant et al. (2004) compared levels of *p,p'*-DDD and other chlorinated pesticides in the semen of 45 fertile and 45 infertile men in India. Levels of *p,p'*-DDD, *p,p'*-DDE, total DDT and various isomers of hexachlorocyclohexane (HCH) were significantly ($p < 0.05$) higher in the semen of infertile than fertile men. Semen levels of *p,p'*-DDD were 78% higher in infertile men. Correlation analysis showed that *p,p'*-DDD levels in semen of infertile men were significantly correlated with higher levels of fructose (a marker for seminal vesicle secretion). Infertile men had higher levels of fructose than fertile men and the authors suggested that the higher fructose was indicative of underutilization of fructose due to biochemical defects. As with other studies using biological levels of *p,p'*-DDD as a measure of exposure, it is not possible to associate any of the observed effects on male reproductive function with exposure to *p,p'*-DDD.

In summary, the available human studies do not provide conclusive evidence for an association between *p,p'*-DDD exposure and reproductive or hormonal effects. In all of these studies, the participants had measurable levels of other chlorinated compounds, including DDE and DDT. Further, when *p,p'*-DDD levels in biological fluids are used as a surrogate for exposure, it is not possible to determine whether the levels result from direct exposure to *p,p'*-DDD or from metabolism of DDT and/or DDE. As a consequence, none of the human studies is suitable for use in deriving provisional toxicity values.

Animal Studies

Oral Exposure

Subchronic Exposure — In preparation for a chronic cancer bioassay, NCI (1978) conducted a range-finding dietary toxicity study of DDD in Osborne-Mendel rats and B6C3F1 mice. Technical grade DDD (60% *p,p'*-DDD) in corn oil was mixed with feed and administered *ad libitum* to groups of 5 male and 5 female rats per concentration for 6 weeks, followed by a 2-week observation period. The test material contained 19 impurities contributing 40% of the total dose; none of the impurities were identified. The major analytical peak comprising 60% of the test material was assumed to be *p,p'*-DDD. Diets containing 0, 562, 1000, 1780, 3160 or 5620 ppm technical grade DDD were fed to rats (corresponding to *p,p'*-DDD doses of 0, 29, 52, 93,

166 or 295 mg/kg-day in males, and 0, 32, 57, 101, 179 or 319 mg/kg-day in females¹ after adjustment for 60% purity). Only mortality and body weight changes were evaluated; no animals were necropsied.

No deaths were observed in rats exposed to *p,p'*-DDD concentrations up to 3160 ppm; no information was reported on mortality at 5620 ppm (NCI, 1978). Mean body weights were reduced in male rats exposed to 1780 ppm (9% lower than controls) and 3160 ppm (10% lower), and in female rats exposed to 1000 ppm (39% lower) and 1780 ppm (4% lower); neither statistical analysis nor raw data were presented. No data on body weight changes at other doses were reported. This study did not provide sufficient information to establish effect levels.

Groups of 5 male and 5 female mice were exposed to dietary *p,p'*-DDD for 6 weeks, followed by a 2-week observation period (NCI, 1978); test material and study protocol were as described above for rats (NCI, 1978). Mice received diets containing 0, 251, 398, 631, 1000 or 1590 ppm (0, 27, 43, 68, 108 or 172 mg/kg-day *p,p'*-DDD in males, and 0, 29, 47, 74, 117 or 186 mg/kg-day *p,p'*-DDD in females² after adjustment for 60% purity). Mortality was observed in male mice of all but the 631 ppm exposure group (data and details not reported); no deaths occurred among control males (NCI, 1978). Mortality was also observed in female mice exposed to 1000 and 1590 ppm but not in other groups (data not reported). *p,p'*-DDD did not affect body weights in the exposed mice; mean body weight gain in male and female mice exposed to concentrations up to 631 ppm exceeded weight gain in controls (details not reported). This study did not provide sufficient information to establish effect levels.

Banerjee et al. (1996) evaluated the effects of dietary *p,p'*-DDD exposure on humoral and cell-mediated immune response in Wistar rats. Groups of 8-12 male rats were given either the control diet or a diet containing 200 ppm *p,p'*-DDD (99% pure) for 6 weeks (equivalent to about 18 mg/kg-day³), during which general condition, food consumption and body weights were recorded weekly. Half of each group was immunized by subcutaneous administration of 3 mg ovalbumin three weeks before the end of the exposure period; the other half was left unstimulated. At the end of the exposure period, rats were sacrificed and blood samples collected. The liver, spleen and thymus from each animal were removed and weighed. The humoral immune response was quantified by measuring immunoglobulin levels (IgM and IgG), estimating the albumin/globulin ratio and measuring the ovalbumin antibody titer by ELISA. Cell-mediated response was assessed *in vivo*, by quantifying the delayed type hypersensitivity reaction (measuring footpad thickness after ovalbumin challenge) and *in vitro* by measuring leukocyte and macrophage migration inhibition. The latter tests assess whether chemical exposure results in suppression of lymphokine production.

Exposure to *p,p'*-DDD had no effect on mortality, food intake, body weight, or relative liver or thymus weights, but significantly ($p < 0.05$) reduced relative spleen weight by 14%; absolute spleen weights were not reported (Banerjee et al., 1996). With regard to humoral

¹ Based on reference values for food consumption and body weight (U.S. EPA, 1988a); doses given are for pure *p,p'*-DDD after adjustment for 60% purity.

² Based on reference values for food consumption and body weight (U.S. EPA, 1988a); doses given are for pure *p,p'*-DDD after adjustment for 60% purity.

³ Based on reference values for food consumption and body weight (U.S. EPA, 1988a).

immune responses, treatment with *p,p'*-DDD had no effect on the serum albumin/globulin ratio, but significantly ($p < 0.05$) reduced the levels of IgG, IgM and the titer of anti-ovalbumin antibody in serum by 15, 24 and 35%, respectively, compared to controls. Treatment with *p,p'*-DDD significantly reduced cell-mediated immune responses; delayed type hypersensitivity reactions (increase in footpad thickness) and tests of inhibition of migration of leucocytes and macrophages were suppressed by 24%, 24% and 25% (respectively) compared to controls. In this study, the only dose tested (18 mg/kg-day) is a minimal LOAEL for evidence of immunosuppression and potential effects on spleen weight in rats; no NOAEL can be identified from these data. The LOAEL is considered minimal because the impact of the observed changes on immune function is not certain.

Chronic Exposure — Tomatis et al. (1974) evaluated the carcinogenicity of *p,p'*-DDD (and *p,p'*-DDE separately) in CF-1 mice treated via the diet for a lifetime. The authors administered *p,p'*-DDD in the diet (250 ppm) to 60 male and 60 female mice (6-7 weeks old) for up to 123 weeks; 101 male and 97 female mice were maintained on a control diet. The test compound was 99% pure and was dissolved in acetone prior to being mixed with powdered food and converted to pellets. It is not clear whether the control diet contained acetone. A dietary concentration of 250 ppm corresponds to an estimated *p,p'*-DDD dose of about 43 mg/kg-day (for both males and females) based on reference values for food consumption and body weight of mice (U.S. EPA, 1988a). Groups of four animals (sex not specified) were sacrificed either between weeks 65 and 74 of treatment or between weeks 94 and 118 of treatment for analysis of *p,p'*-DDD levels in the liver and interscapular fat (and sometimes in liver tumors and kidney; details not provided). All animals dying spontaneously or killed humanely were necropsied; remaining animals were sacrificed at 130 weeks of age. Histopathology evaluation was restricted to the lungs, heart, thymus, liver, kidneys, spleen, brain and any organs with gross abnormalities.

Survival was not affected by *p,p'*-DDD (Tomatis et al., 1974). Survival to 90 weeks was 76 and 72% in treated males and females, compared with 67 and 73% in control males and females, respectively. There were no clinical signs of toxicity among mice treated with *p,p'*-DDD. The authors reported neither a statistical comparison of body weights nor raw data; however, based on visual evaluation of body weight curves (covering the period from the 3rd through 14th month of age), body weights of the treated males were depressed by more than 10% relative to controls over the entire period of observation; body weights of treated females were unaffected by treatment. The only other possible effect was a 5-fold increase in the incidence of myocardial necrosis in males, although the overall incidence was small (3/59 in treated animals vs. 1/98 in controls). No statistical analysis was presented by the authors. A post-hoc Fisher's Exact test was performed on the response data with a p-value of 0.15. Although not statistically significant by standard definitions, the 5-fold increase is still suggestive of an effect. The only dose of *p,p'*-DDD tested, 43 mg/kg-day, is a LOAEL for body weight depression and suggestive of myocardial necrosis in male mice in this study.

The authors noted that the incidence of lung tumors was increased over controls in *p,p'*-DDD-exposed mice of both sexes; in addition, the incidence of hepatomas was increased in male mice (Tomatis et al., 1974). This study was used in the derivation of the oral slope factor for *p,p'*-DDD (U.S. EPA, 2007).

NCI (1978) conducted a carcinogenicity bioassay of *p,p'*-DDD in Osborne-Mendel rats and B6C3F1 mice. Technical grade DDD (60% *p,p'*-DDD) in corn oil was mixed with feed at varying concentrations and administered *ad libitum*. The test material contained 19 impurities, contributing 40% of the total dose; none of the impurities were identified. Nominal concentrations, durations of exposure at these concentrations, and weighted average concentration and dose estimates are given in Table 1. As the table indicates, the exposure concentration was increased once in rats and twice in mice, as the animals tolerated the exposures well. Rats were observed for 34 or 35 weeks after exposure termination and prior to sacrifice. Mice were observed for 13 to 15 weeks after the 78-week exposure period and prior to sacrifice. Weighted average exposure concentrations shown in Table 1 are averaged over the 78-week exposure period and do not take into account the post-exposure observation period. Weighted average dose estimates shown in the table are doses of *p,p'*-DDD after adjustment for purity.

Body weight and food consumption measurements, clinical observations and palpations for masses were conducted weekly for 10 weeks and monthly thereafter; mortality checks were performed daily (NCI, 1978). Necropsy was performed on all animals, but organ weights were not recorded. Histopathologic examination was initially limited to control animals, animals with visible tumors and at least 10 males and females with no gross pathological findings from each group. Later in the study, the protocol was altered to include tissues from other animals; however, the authors did not indicate how the other animals were selected, how many were included or when the protocol change was initiated. Nearly 30 tissues were subjected to microscopic examination. The authors noted that tissues were not examined from some animals that died early and that some animals were missing, cannibalized or in an advanced state of autolysis, precluding histopathologic examination. Incidence of lesions was reported using the number of animals for which that specific tissue was examined as the number at risk, except where lesions were observed grossly or could appear at multiple sites (e.g., lymphoma), in which cases the number of animals necropsied was used.

The authors reported that, beginning during week 30 and continuing through termination of the exposure period, treated rats exhibited a slightly greater incidence of clinical signs of toxicity (hunched appearance and urine staining; data not reported) (NCI, 1978). Prior to 30 weeks and during the recovery period, there was no treatment-related effect on the incidence of clinical signs (data not reported), according to the authors. *p,p'*-DDD treatment did not significantly affect probability of survival in either sex. There were clear treatment-related reductions in body weight, but the authors did not present statistical comparisons of group mean body weights or raw data. Based on graphical presentation of the data, the greatest differences from control weights occurred between weeks 60 and 75, when the mean body weights were about 10% and 20% lower than controls in low- and high-dose males (respectively) and about 20% and 30% lower in low- and high-dose females. Treatment with *p,p'*-DDD had no significant effect on the incidence of nonneoplastic lesions in rats in any tissue examined. A NOAEL cannot be determined from this study. The low dose (39 mg/kg-day in females) is a LOAEL for depression of body weight gain and clinical signs of toxicity. The LOAEL is for the mixture. A LOAEL for *p,p'*-DDD cannot be established from this study.

Table 1. Group Sizes, Dietary Concentrations and Dose Estimates for NCI (1978) Cancer Bioassay for <i>p,p'</i>-DDD						
Group	Group Size	Nominal Concentration (mg/kg)	Duration at this Concentration (weeks)	Untreated Duration (weeks)	Weighted Average Concentration Technical grade DDD^a (mg/kg)	Weighted Average Daily Dose <i>p,p'</i>-DDD^b (after adjustment for purity) (mg/kg-day)
Male Rats						
Control	20	0		111		0
Low Dose	50	1400 1750 0	23 55	34	1647	69
High Dose	50	2800 3500 0	23 55	35	3294	138
Female Rats						
Control	20	0		111		0
Low Dose	50	850 0	78 35	35	850	39
High Dose	50	1700 0	78 35	35	1700	79
Male Mice						
Control	20	0		90		0
Low Dose	50	315 375 425 0	5 11 62	13	411	42
High Dose	50	630 750 850 0	5 11 62	14	822	85
Female Mice						
Control	20	0		90		0
Low Dose	50	315 375 425 0	5 11 62	14	411	43
High Dose	50	630 750 850 0	5 11 62	15	822	85
^a Calculated by the authors as the sum of concentration x time averaged over 78 weeks. ^b Calculated using weighted average concentration and reference values for body weight and food consumption from U.S. EPA (1988a); doses adjusted for 60% purity. Source: NCI, 1978.						

The authors reported treatment-related increases in the incidence of thyroid follicular-cell neoplasms in rats treated with *p,p'*-DDD (NCI, 1978). No other treatment-related effects on neoplasm frequency were observed. This study was evaluated as part of the IRIS cancer assessment, but was not used in deriving the oral slope factor.

In mice, *p,p'*-DDD treatment had no significant effect on probability of survival in either sex. Clinical signs occurred with the same frequency in treated and control animals. Exposure to *p,p'*-DDD had no effect on male body weight throughout the treatment period, but dose-related depression of body weight was observed in female mice after week 30. The authors did not present statistical comparisons of group mean body weights or raw data. Based on graphical presentation of the data, the body weight reduction peaked at about 14% in the high-dose group between weeks 60 and 75; in the low-dose group, body weight decrements appeared to be less than 10% throughout the study. Treatment did not significantly increase the incidence of neoplastic or nonneoplastic lesions in any tissue in either sex. The low dose of 42 mg/kg-day *p,p'*-DDD is a NOAEL and the high dose of 85 mg/kg-day *p,p'*-DDD is a LOAEL for body weight depression in female mice.

Inhalation Exposure

There are no data on the effects in laboratory animals of *p,p'*-DDD exposure via inhalation.

Other Studies

Adrenal Effects — Cueto and Brown (1958) fractionated technical grade DDD and tested the fractions and isolates, delivered in gelatin capsules, for adrenocorticolytic activity in male dogs (breed not specified). A single dog received 80 mg/kg-day of purified *p,p'*-DDD for 29 days and another the same dose for 80 days; a third dog was treated with 200 mg/kg-day for 30 days and a fourth dog was left untreated for 100 days as a control. The endpoints examined included general appearance, periodic tests of adrenal activity and, after necropsy, examination of adrenal histopathology. No other organ system was evaluated. Treatment with *p,p'*-DDD at either dose level had no effect on the physical state of the dogs. In tests of adrenal activity administered after 4 and 20 days of treatment, the dog treated with 200 mg/kg-day of *p,p'*-DDD and the control dog exhibited the same effects in response to an injection of adrenocorticotrophic hormone: there were similar decreases in the eosinophil count and similar increases in the plasma level of 17-hydroxycorticosteroids. At termination, no treated dogs showed evidence of adrenal histopathology.

In a similar study, Powers et al. (1974) fed technical grade DDD (characterized by the authors as 90% *p,p'*-DDD and 5-8% *o,p'*-DDD, other impurities unspecified) dissolved in corn oil and administered in gelatin capsules to mixed groups of mongrel and purebred beagle dogs. The dogs were given doses of either 100 or 200 mg/kg for varying time periods up to 30 days. Control groups (mongrels and beagles) of various sizes were maintained. Upon sacrifice, the adrenal glands were weighed (in some cases) and/or examined with light and electron microscopy. The authors reported histopathology findings in the adrenals of treated dogs, including degenerative vacuolation, especially in the inner cortex, mitochondrial swelling,

cellular necrosis and dilatation of smooth endoplasmic reticulum. Because the test material in this study included *o,p'*-DDD and potentially other contaminants, it is not possible to determine whether any of the adrenal affects are attributable to *p,p'*-DDD exposure.

Mechanistic — A number of studies have investigated the hormonal activities of DDT and related compounds. When Gellert et al. (1972) injected groups of 11 or 12 mature ovariectomized Sprague-Dawley rats with 0.1 or 10 mg/day of *p,p'*-DDD in DMSO for 7 days, there was no effect on uterine weight, uterine histology, cytology of vaginal smears or serum levels of luteinizing hormone or follicle stimulating hormone. In castrated male Brl Han: WIST Jcl (GALAS) rats treated with 8, 40 or 200 mg/kg-day *p,p'*-DDD via gavage for 10 days, either with or without testosterone propionate, treatment with 200 mg/kg *p,p'*-DDD and testosterone propionate resulted in significant decreases in seminal vesicle and bulbocavernosus/levator ani muscles, indicating antiandrogenic activity (Yamasaki et al., 2004). In *in vitro* assays, *p,p'*-DDD did not competitively inhibit binding of 17β -estradiol to the estrogen receptor, but competitively inhibited binding of a synthetic androgen (R1881) to the rat androgen receptor (Kelce et al., 1995). In *in vitro* assays using yeast reporter gene systems, *p,p'*-DDD was unable to activate expression of the estrogen receptor gene or the androgen receptor gene at concentrations below 10^{-4} M (Gaido et al., 1997). Using an *in vitro* human hepatoma cell reporter gene system, Maness et al. (1998) found that *p,p'*-DDD did not stimulate expression of the human androgen receptor (hAR) gene, but did inhibit androgen-dependent expression of the hAR gene. *p,p'*-DDD gave positive results in an androgen receptor binding assay (Yamasaki et al., 2004). The results of these experiments suggest that *p,p'*-DDD has antiandrogenic activity, but no estrogenic activity.

Limited evidence suggests that *p,p'*-DDD binds to lung tissues and can be cytotoxic to lung cells. When Lund et al. (1989) intravenously injected radiolabeled *p,p'*-DDD into mice, autoradiography of solvent-extracted, whole-body sections revealed specific covalent binding in the alveoli of the lung, in the lateral nasal gland and the salivary glands. The results of the *in vivo* study suggest that pulmonary binding of *p,p'*-DDD can occur after intravenous exposure. An *in vitro* experiment in the same paper demonstrated that *p,p'*-DDD irreversibly bound to protein following incubation with S-9 fractions from murine lung or liver. The authors concluded that covalent binding of *p,p'*-DDD in the lung was the result of *in situ* bioactivation. In an *in vitro* study, Nichols et al. (1992) incubated lung cells isolated from rabbits with *p,p'*-DDD, with or without 1-aminobenzotriazole (1-ABT - a suicide substrate inhibitor of cytochrome P-450 monooxygenases). Cytotoxicity of *p,p'*-DDD to Clara cells especially and to alveolar type II cells and alveolar macrophages to a lesser degree, was dependent on the presence of functional cytochrome P-450. Subsequently, Nichols et al. (1995) evaluated potential mechanisms for bioactivation of *p,p'*-DDD in cultured Clara cells of rabbits and a transformed human bronchial epithelial cell line (BEAS-2B). Both cell types were vulnerable to *p,p'*-DDD-mediated cytotoxicity and were protected by co-incubation with 1-ABT, the inhibitor to cytochrome P-450. In another experiment, Nichols et al. (1995) found that cytotoxicity was reduced when human BEAS-2B cells, rabbit Clara cells, or rabbit pulmonary microsomes were incubated with *p,p'*-DDD that had a deuterium substitution at the C-1 position. The results indicated that the cytotoxicity of *p,p'*-DDD may be caused by its oxidation at C-1 mediated by cytochrome P-450 in the lung.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC ORAL RfD VALUES FOR *p,p'*-DDD

None of the human studies of *p,p'*-DDD are suitable for derivation of provisional oral RfD values. The database includes several epidemiological studies of workers exposed to technical grade DDT (a mixture that includes a small percentage of *p,p'*-DDD), as well as studies evaluating the potential association between biological measurements of *p,p'*-DDD and reproductive or hormonal effects. It is not possible to clearly attribute any effects reported in these studies to direct exposure to *p,p'*-DDD due to the confounding effects of concomitant exposure to other organochlorine compounds (especially DDT and its other metabolites), and because it is not possible to determine whether *p,p'*-DDD measured in biological tissues resulted from exposure to *p,p'*-DDD or from metabolism of DDT or DDE to *p,p'*-DDD in the human body.

There are no suitable long-term general toxicity animal studies for derivation of a provisional RfD. The Tomatis et al. (1974) study is designed primarily as a carcinogenicity bioassay, with very sparse detail on noncancer effects. Furthermore, the LOAEL of 43 mg/kg-day is very high compared to closely-related compounds. Chronic LOAELs for related compounds are 0.25 mg/kg-day (*p,p'*-DDT; U.S. EPA, 1985b), 4.0 mg/kg-day (Cueto and Brown, 1958) and 12 mg/kg-day (NCI, 1978), with the latter two being FELs (mortality). Given the low LOAELs and FELs for closely related compounds, the potential is high that a well-designed *p,p'*-DDT chronic study would produce a much lower LOAEL. As a result, the Tomatis study is judged to be inadequate for assessment of long-term noncancer toxicity.

Studies suitable for use in deriving provisional RfD values include a chronic study in mice (Tomatis et al., 1974) and a 6-week immunotoxicity study in rats (Banerjee et al., 1996). Summaries of these studies and comparisons with the LOAEL values from the chronic NCI (1978) study are shown in Table 2. The usefulness of data from the NCI (1978) subchronic and chronic feeding studies for p-RfD derivation is compromised by the low purity of the technical grade DDD tested. Only 60% of the product was *p,p'*-DDD and at least 19 impurities (unspecified) were present in the remaining 40%. The chronic data are further compromised by the substantial adjustments in administered dietary level during the study and by the long post-treatment observation period, during which recovery from or reversal of effects could have occurred. The two studies in dogs (Cueto and Brown, 1958; Powers et al., 1974) are not suitable for p-RfD derivation due to the small number of animals used, limited endpoints evaluated and, in some cases, post-treatment observation periods allowing for reversal of effects.

The only remaining study, Banerjee et al. (1996), is a 6-week immunotoxicity study that does not cover the required general toxicity endpoints. Although the study was adequate for its purpose and establishes the lowest LOAEL for *p,p'*-DDD, by itself, it does not qualify as the basis for either a subchronic or chronic p-RfD. The oral noncancer database is inadequate for derivation of p-RfDs. Neither of the two studies available for p-RfD derivation included more than one dose level, precluding benchmark dose modeling of the effects.

Table 2. Summary of Available Oral Noncancer Dose-Response Information Suitable for p-RfD Derivation and Comparison with LOAELs from NCI (1978) Chronic Studies								
Species	Sex	Dose (mg/kg-day)	Exposure Duration	NOAEL (mg/kg-day)	LOAEL (mg/kg-day)	Responses	Comments	Reference
Rats	M	0, 18 mg/kg-day	6 weeks	NA	18	Immunosuppression (reduced humoral and cell-mediated immunity) and decreased relative spleen weight. Minimal LOAEL.	Endpoints included clinical signs, body weight, selected organ weights, and immunotoxicity parameters.	Banerjee et al., 1996
Mice	M,F	0, 43 mg/kg-day	123 weeks	NA	43	Body weight depression in males.	Endpoints included survival, clinical signs, body weight, and histopathology of selected organs.	Tomatis, 1974
Rats	M,F	0, 69, 138 mg/kg-day (M) or 0, 39, 79 mg/kg-day (F)	78 weeks, followed by 34-35 weeks observation	NA	39	Depression of body weight gain and clinical signs of toxicity in females.	Test article only 60% pure. Prolonged observation period may have allowed for recovery from toxic effects. Not suitable for p-RfD derivation.	NCI, 1978
Mice	M,F	0, 42, 85 mg/kg-day (M) or 0, 43, 85 mg/kg-day (F)	78 weeks, followed by 13-15 weeks observation	42	85	Depression of body weight gain in females.	Test article only 60% pure. Prolonged observation period may have allowed for recovery from toxic effects. Not suitable for p-RfD derivation.	NCI, 1978

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION p-RfC VALUES FOR *p,p'*-DDD

No studies specifically investigating the effects of inhaled *p,p'*-DDD in humans or animals were located. Thus, provisional RfCs were not derived for *p,p'*-DDD.

DERIVATION OF A PROVISIONAL CARCINOGENICITY ASSESSMENT FOR *p,p'*-DDD

A cancer assessment, including an oral slope factor, is available for *p,p'*-DDD on IRIS (U.S. EPA, 1988b).

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11-30-2001

Provisional Peer Reviewed Toxicity Values for

Acenaphthene
(CASRN 83-32-9)

Derivation of an Oral Slope Factor

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR ACENAPHTHENE (CASRN 83-32-9)
Derivation of an Oral Slope Factor**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional

Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

No oral slope factor for acenaphthene is listed on IRIS (U.S. EPA, 2001), in the HEAST (U.S. EPA, 1997), or in the Drinking Water Standards or Health Advisories List (U.S. EPA, 2000). The CRAVE workgroup (U.S. EPA, 1993, 1995) assigned acenaphthene to Group D, not classifiable as to human carcinogenicity, based on no human data and no animal studies pertinent to carcinogenicity; accordingly, no oral slope factor was derived. The CARA lists (U.S. EPA, 1991a, 1994) include an Ambient Water Quality Criteria document (AWQCD) for acenaphthene (U.S. EPA, 1980), an AWQCD addendum (U.S. EPA, 1989), and a Health Effects Assessment document (HEA) for polycyclic aromatic hydrocarbons (U.S. EPA, 1984). In addition, a Health Effects Assessment document for acenaphthene (U.S. EPA, 1987) is available (although not listed on CARA). All four documents reported a lack of data regarding chronic exposure or carcinogenicity for acenaphthene. Acenaphthene was not discussed in a Drinking Water Criteria Document for polycyclic aromatic hydrocarbons (U.S. EPA, 1991b). A Toxicological Profile for polycyclic aromatic hydrocarbons (ATSDR, 1995) contained no data regarding carcinogenicity for acenaphthene, and no data are listed by IARC (2001). An NTP status report (NTP, 2001), an Environmental Health Criteria document on polycyclic aromatic hydrocarbons (WHO, 1998), a review of health effects of aromatic hydrocarbons (Cavender, 1994), and a Multimedia Document for Polycyclic Aromatic Hydrocarbons produced by SRC for EPA (SRC, 1992) were consulted for relevant information. Literature searches were conducted from 1989 to December 2000 for studies relevant to the derivation of an oral slope factor for acenaphthene. The

databases searched were: TOXLINE, MEDLINE, CANCERLIT, RTECS, GENETOX, HSDB, CCRIS, TSCATS, EMIC/EMICBACK, and DART/ETICBACK.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No studies were located in the literature search regarding carcinogenicity in humans following oral exposure to acenaphthene.

Animal Studies

Previously reviewed studies of oral exposure to acenaphthene, being ≤ 90 days, are not of sufficient duration to evaluate potential carcinogenicity (U.S. EPA, 1980, 1984, 1987, 1989, 2001). No additional studies were located in the literature search regarding carcinogenicity in animals following oral exposure to acenaphthene.

Other Studies

Genotoxicity studies previously reviewed by the U.S. EPA (1980, 1984, 1987, 1989, 1993, 2001) or the WHO (1998) reported inconsistent, but mostly negative, results for acenaphthene. Updated literature searches identified additional relevant genotoxicity studies for acenaphthene. Acenaphthene tested negative in studies using *Salmonella typhimurium* strains TA98 and TA100 without S-9 activation (Kangsadalampai et al., 1996; Sasaki et al., 1995) and strains TA97, TA98, TA100, TA1535, and TA1537 with S-9 activation (Zeigler et al., 1992). Acenaphthene was negative with S-9 metabolic activation in the SOS chromotest, a genotoxicity assay using *Escherichia coli* strain PQ37 (Mersch-Sunderman et al., 1993).

Chaloupka et al. (1994) evaluated hepatic enzyme induction, as methoxyresorufin *O*-demethylase (MROD) activity, following acenaphthene injection in 15-day-old nursing male B6C3F1 mouse pups. Acenaphthene did not bind to the aryl hydrocarbon (Ah) receptor or to the 4S carcinogen-binding protein, suggesting that the induction of hepatic Cyp1a2 gene expression by tricyclic PAHs, such as acenaphthene, is independent of the Ah receptor pathway.

DERIVATION OF A PROVISIONAL ORAL SLOPE FACTOR FOR ACENAPHTHENE

Human and animal data are inadequate to evaluate the potential carcinogenicity of acenaphthene by the oral route. Acenaphthene is categorized in Group D, not classifiable as to human carcinogenicity, which precludes derivation of an oral slope factor.

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11-30-2001

Provisional Peer Reviewed Toxicity Values for

Acenaphthene
(CASRN 83-32-9)

Derivation of an Inhalation Unit Risk

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR ACENAPHTHENE (CASRN 83-32-9)
Derivation of an Inhalation Unit Risk**

Background

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 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
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A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided

in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

No inhalation unit risk for acenaphthene is listed on IRIS (U.S. EPA, 2001) or in the HEAST (U.S. EPA, 1997). The CRAVE workgroup (U.S. EPA, 1993, 1995) assigned acenaphthene to Group D, not classifiable as to human carcinogenicity, based on no human data and no animal studies pertinent to carcinogenicity; accordingly, no inhalation unit risk was derived. The CARA lists (U.S. EPA, 1991, 1994) include a Health Effects Assessment document (HEA) for polycyclic aromatic hydrocarbons (U.S. EPA, 1984). In addition, a Health Effects Assessment document for acenaphthene (U.S. EPA, 1987) is available (although not listed on CARA). Both HEAs indicate that no chronic inhalation or carcinogenicity data are available for acenaphthene. No information regarding carcinogenicity of acenaphthene is listed by the ACGIH (2000), NIOSH (2000), OSHA (1999), or IARC (2001). A Toxicological Profile for polycyclic aromatic hydrocarbons (ATSDR, 1995) contained no data regarding inhalation exposure or carcinogenicity for acenaphthene. An NTP status report (NTP, 2001), an Environmental Health Criteria document (WHO, 1998), a review on the health effects of aromatic hydrocarbons (Cavender, 1994), and a Multimedia Document for Polycyclic Aromatic Hydrocarbons produced by SRC for EPA (SRC, 1992) were consulted for relevant information. Literature searches were conducted from 1989 to December 2000 for studies relevant to the derivation of an inhalation unit risk for acenaphthene. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, RTECS, GENETOX, HSDB, CCRIS, TSCATS, EMIC/EMICBACK, and DART/ETICBACK.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No studies were located in the literature search regarding health effects in humans following inhalation exposure to acenaphthene.

Animal Studies

A subchronic rat inhalation study by Reshettiuk et al. (1970) was considered inadequate for cancer assessment because of incomplete reporting of methods and results (U.S. EPA, 1984, 1987). Reshettiuk et al. (1970) exposed male white rats (number, strain, controls not reported) to $12 \pm 1.5 \text{ mg/m}^3$ of acenaphthene vapors 4 hours/day, 6 days/week for 5 months. Chronic aspecific pneumonia, including circulatory alterations (undefined), was observed and described: altered reflexes in the upper airways, desquamation of alveolar epithelium, and focal bronchitis accompanied by hyperplasia and metaplasia. An increase in the concentration of nucleic acids in the liver was observed. No further details of this study were reported.

No additional inhalation studies were located in the literature search.

Other Studies

Genotoxicity studies previously reviewed by the U.S. EPA (1984, 1987, 1993, 2001) or the WHO (1998) reported inconsistent, but mostly negative, results for acenaphthene. Updated literature searches identified additional relevant genotoxicity studies for acenaphthene. Acenaphthene tested negative in studies using *Salmonella typhimurium* strains TA98 and TA100 without S-9 activation (Kangsadalampai et al., 1996; Sasaki et al., 1995) and strains TA97, TA98, TA100, TA1535, and TA1537 with S-9 activation (Zeigler et al., 1992). Acenaphthene was negative with S-9 metabolic activation in the SOS chromotest, a genotoxicity assay using *Escherichia coli* strain PQ37 (Mersch-Sunderman et al., 1993).

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DERIVATION OF A PROVISIONAL INHALATION UNIT RISK FOR ACENAPHTHENE

Human and animal data are inadequate to evaluate the potential carcinogenicity of acenaphthene by the inhalation route. Acenaphthene is categorized in Group D, not classifiable as to human carcinogenicity, which precludes derivation of an inhalation unit risk.

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11-30-2001

Provisional Peer Reviewed Toxicity Values for

Acenaphthene
(CASRN 83-32-9)

Derivation of a Chronic Inhalation RfC

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR ACENAPHTHENE (CASRN 83-32-9)
Derivation of a Chronic Inhalation RfC**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional

Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

No RfC for acenaphthene is listed on IRIS (U.S. EPA, 2001) or in the HEAST (U.S. EPA, 1997). An RfC for acenaphthene has not been reviewed by the RfD/RfC workgroup (U.S. EPA, 1995). The CARA lists (U.S. EPA, 1991, 1994) include a Health Effects Assessment document (HEA) for polycyclic aromatic hydrocarbons (U.S. EPA, 1984). In addition, a Health Effects Assessment document for acenaphthene (U.S. EPA, 1987) is available (although not listed on CARA). Both HEAs indicate that no chronic inhalation data are available for acenaphthene. A Toxicological Profile for polycyclic aromatic hydrocarbons does not derive any inhalation MRLs for acenaphthene (ATSDR, 1995, 2000). Exposure limits for acenaphthene are not listed by the ACGIH (2000), NIOSH (2000), or OSHA (1999). Acenaphthene is not listed by IARC (2001). An NTP status report (NTP, 2001), an Environmental Health Criteria document (WHO, 1998), a review on the health effects of aromatic hydrocarbons (Cavender, 1994), and a Multimedia Document for Polycyclic Aromatic Hydrocarbons produced by SRC for EPA (SRC, 1992) were consulted for relevant information. Literature searches were conducted from 1989 to December 2000 for studies relevant to the derivation of an RfC for acenaphthene. The databases searched were TOXLINE, MEDLINE, CANCERLIT, RTECS, GENETOX, HSDB, CCRIS, TSCATS, EMIC/EMICBACK, and DART/ETICBACK.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No studies were located regarding health effects in humans following inhalation exposure to acenaphthene.

Animal Studies

A subchronic rat inhalation study by Reshettiuk et al. (1970) is considered inadequate because of incomplete reporting of methods and results. Reshettiuk et al. (1970) exposed male white rats (number, strain, controls not reported) to 12 ± 1.5 mg/m³ of acenaphthene vapors 4 hours/day, 6 days/week for 5 months. Chronic aspecific pneumonia, including circulatory alterations (undefined), was observed and described: altered reflexes in the upper airways, desquamation of alveolar epithelium, and focal bronchitis accompanied by hyperplasia and metaplasia. An increase in the concentration of nucleic acids in the liver was observed. No further details of this study were reported.

No additional inhalation studies were located in the literature search.

Other Studies

Beach et al. (1992) evaluated the effect of acenaphthene on respiration in mitochondria isolated from beef heart. A dose-response relationship was observed for the inhibition of specific partial electron transport reactions, suggesting that impairment of cellular respiration may be the mechanism of cellular toxicity of acenaphthene.

DERIVATION OF A PROVISIONAL RfC FOR ACENAPHTHENE

No provisional RfC can be derived for acenaphthene because of a lack of appropriate human or animal data.

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3-14-2005

Provisional Peer Reviewed Toxicity Values for
Aldrin
(CASRN 309-00-2)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms

bw - body weight

cc - cubic centimeters

CD - Caesarean Delivered

CERCLA - Comprehensive Environmental Response, Compensation, and Liability Act of 1980

CNS - central nervous system

cu.m - cubic meter

DWEL - Drinking Water Equivalent Level

FEL - frank-effect level

FIFRA - Federal Insecticide, Fungicide, and Rodenticide Act

g - grams

GI - gastrointestinal

HEC - human equivalent concentration

Hgb - hemoglobin

i.m. - intramuscular

i.p. - intraperitoneal

i.v. - intravenous

IRIS - Integrated Risk Information System

IUR - Inhalation Unit Risk

kg - kilogram

L - liter

LEL - lowest-effect level

LOAEL - lowest-observed-adverse-effect level

LOAEL(ADJ) - LOAEL adjusted to continuous exposure duration

LOAEL(HEC) - LOAEL adjusted for dosimetric differences across species to a human

m - meter

MCL - maximum contaminant level

MCLG - maximum contaminant level goal

MF - modifying factor

mg - milligram

mg/kg - milligrams per kilogram

mg/L - milligrams per liter

MRL - minimal risk level

MTD - maximum tolerated dose

MTL - median threshold limit

NAAQS - National Ambient Air Quality Standards
NOAEL - no-observed-adverse-effect level
NOAEL(ADJ) - NOAEL adjusted to continuous exposure duration
NOAEL(HEC) - NOAEL adjusted for dosimetric differences across species to a human
NOEL - no-observed-effect level
OSF - Oral Slope Factor
p-RfD - provisional Oral Reference Dose
p-RfC - provisional Inhalation Reference Concentration
p-OSF - provisional Oral Slope Factor
p-IUR - provisional Inhalation Unit Risk
PBPK - physiologically based pharmacokinetic
ppb - parts per billion
ppm - parts per million
PPRTV - Provisional Peer Reviewed Toxicity Value
RBC - red blood cell(s)
RCRA - Resource Conservation and Recovery Act
RGDR - Regional deposited dose ratio (for the indicated lung region)
REL - relative exposure level
RGDR - Regional gas dose ratio (for the indicated lung region)
RfD - Oral Reference Dose
RfC - Inhalation Reference Concentration
s.c. - subcutaneous
SCE - sister chromatid exchange
SDWA - Safe Drinking Water Act
sq.cm. - square centimeters
TSCA - Toxic Substances Control Act
UF - uncertainty factor
ug - microgram
umol - micromoles
VOC - volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR ALDRIN (CASRN 309-00-2)

Background

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Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

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Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

The HEAST (U.S. EPA, 1997a) lists a subchronic RfD of $3E-5$ mg/kg-day for aldrin adopted from the chronic RfD of the same value listed on IRIS (U.S. EPA, 2003a). The chronic RfD was based on an estimated LOAEL of 0.025 mg/kg-day for liver toxicity (centrilobular histopathology and increased relative organ weight) in rats exposed to aldrin in the diet at a concentration of 0.5 ppm for two years (Fitzhugh et al., 1964). In this derivation, a total uncertainty factor of 1000 (10 to extrapolate from animals to humans, 10 to protect sensitive individuals, and 10 for the use of a LOAEL) was applied to the LOAEL. The chronic RfD is also included in the Drinking Water Standards and Health Advisories List (U.S. EPA, 2002) and the OPP reference dose tracking report (U.S. EPA, 1997b), which includes an identical OPP RfD for aldrin. A Health Effects Assessment (HEA) for aldrin did not derive subchronic or chronic oral RfDs for aldrin because the compound was classified as a carcinogen (U.S. EPA, 1987a).

No other relevant documents were included in the CARA list (U.S. EPA, 1991, 1994a). ATSDR (2002) calculated a chronic oral minimum risk level (MRL) of 3×10^{-5} mg/kg-day for aldrin also using the study of Fitzhugh et al. (1964) and the same uncertainty factors employed in the IRIS derivation. ATSDR (2002) did not derive an intermediate (subchronic) oral MRL because the available studies were not considered to be suitable.

An RfC for aldrin is not listed on the HEAST (1997a) or on IRIS (U.S. EPA, 2003a). The HEA (U.S. EPA, 1987a) indicated that inhalation data for aldrin, limited to monitoring studies in humans, were insufficient to derive an RfC. ATSDR (2002) did not derive inhalation MRLs for aldrin because no suitable quantitative data were available from the existing inhalation studies. The threshold limit value (TLV-TWA) established by ACGIH (2001, 2002), the recommended exposure limit (REL-TWA) set by NIOSH (2002) and the permissible exposure limit (PEL-TWA) established by OSHA (2002) for aldrin are each set at 0.25 mg/m^3 , with notations for skin absorption and carcinogenicity (TLV and REL only, see next paragraph); the levels are intended to protect against effects in the liver (increased organ weight, parenchymatous degeneration and necrosis observed in rats), central nervous system (ranging from headache to convulsions in humans), and kidney (parenchymatous degeneration observed in rats and dogs).

The HEAST (1997a) cites IRIS as the primary source for the carcinogenicity assessment of aldrin. On IRIS (U.S. EPA, 2003a), aldrin is classified as a Group B2, possible human carcinogen, based on inadequate evidence in humans and sufficient evidence in mice. A human oral q_1^* of $17 (\text{mg/kg-day})^{-1}$ for aldrin is presented based on data for hepatic carcinoma in mice; an inhalation unit risk of $4.9 \times 10^{-3} (\mu\text{g/m}^3)$ was derived by extrapolation from the oral data (U.S. EPA, 2003a). The source document for this assessment was a Carcinogenicity Assessment (U.S. EPA, 1987b). IARC (1974, 1987, 2002) determined that aldrin is not classifiable as to its carcinogenicity to humans (Group 3) based on an inadequate data in humans and limited data in animals (definitive positive results in mice but not in rats). ACGIH (2001, 2002) included an A3 notation in its TLV assessment for aldrin to indicate the compound's status as a confirmed animal carcinogen with unknown relevance to humans. The NIOSH (2002) REL also noted the carcinogenicity of aldrin to animals in the same organs: lungs, liver, thyroid and adrenal glands.

An Environmental Health Criteria document on aldrin and dieldrin (WHO, 1989), a Health Effects Support Document for Aldrin/Dieldrin (U.S. EPA, 2003b), a toxicity review on chlorinated hydrocarbon pesticides (Bus and Leber, 2001), a review on the long-term health effects of aldrin and dieldrin (de Jong, 1991), an occupational hazard review on aldrin and dieldrin (NIOSH, 1978) and the NTP (2002a, 2002b) management status report and health and safety report for aldrin were consulted for relevant information. Literature searches were conducted for the period from 1999 to October 2002 to identify data relevant for the derivation of a provisional RfD, RfC and cancer assessment for aldrin. The following databases were searched: TOXLINE, MEDLINE, CANCERLIT, NTIS/BIOSIS, RTECS, HSDB, GENETOX,

CCRIS, TSCATS, EMIC/EMICBACK, and DART/ETICBACK. Additional literature searches were conducted by NCEA-Cincinnati from 2002 through May 2004 using TOXLINE, MEDLINE, Chemical and Biological Abstracts databases.

REVIEW OF PERTINENT DATA

Human Studies

No data were located for chronic or subchronic toxicity in humans following quantified exposures by the oral route. Acute oral exposures to aldrin have been reported to result in neurological effects that include headache, dizziness, nausea, vomiting, malaise, myoclonic jerks of the limbs, clonic and tonic convulsions, and coma (NIOSH, 1978; U.S. EPA, 1987b; de Jong, 1991; ACGIH, 2001; Bus and Leber, 2001; U.S. EPA, 2003b). A 23-year-old male who ingested 25.6 mg/kg exhibited convulsions within 20 minutes, followed by hematuria and azotemia that lasted 18 days, restlessness, hypothermia and tachycardia that lasted 5 days, and abnormalities in electroencephalograms (generalized cerebral dysrhythmia) that lasted for six months. Estimates for the lowest human lethal dose of aldrin range between 1.25 and 10 mg/kg (de Jong, 1991; U.S. EPA, 2003b).

No data were located for chronic or subchronic toxicity in humans following quantified exposures by the inhalation route. Dermal absorption is presumed to account for the major fraction of the absorption following exposures of unprotected industrial or agricultural workers to aldrin dust, and undoubtedly some oral absorption also occurs under these conditions (de Jong, 1991; Bus and Leber, 2001).

Epidemiological studies have been conducted to evaluate the cancer risk following occupational exposure to aldrin. The IRIS document for aldrin (U.S. EPA, 2003a) indicated that two different epidemiological studies did not quantify exposure in workers and were limited in their ability to detect an excess of deaths from cancer (Van Raalte, 1977; Ditraglia et al., 1981); subjects in both studies had also been exposed to other pesticides in addition to aldrin. Increases (not statistically significant) in standard mortality ratios for a few cancer sites (rectum, esophagus, and lymphatic/hemopoietic cancer) identified in the study of Ditraglia et al. (1981) did not persist in subsequent follow-up studies of the cohort (Brown, 1992; Amoateng-Adjepong et al., 1995). The mortality in these studies was not associated with any cancer events in the rectum, esophagus or lymphatic/hematopoietic system. However, an increase in deaths in male workers from cancer of the liver and biliary tract that was observed in the study of Ditraglia et al. (1981) was found to be significant in the study of Brown (1992). Amoateng-Adjepong et al. (1995) reported that excess deaths from hepatobiliary cancer were significantly increased only for hourly paid workers, but were not correlated with increasing duration of employment.

de Jong (1991) conducted an epidemiological study in Dutch petrochemical workers exposed primarily by dermal contact and inhalation to aldrin and sometimes also to dieldrin. In this study, 570 workers (gender not identified) primarily exposed between January 1954 and January 1970 were followed up for mortality until January 1987. The study used extensive data for industrial hygiene and job histories to assess exposures to aldrin/dieldrin retrospectively. In addition, biological monitoring data, based on blood levels of dieldrin, a persistent metabolite of aldrin, were available for 343 of the workers for the period 1963-1970; data from these workers was used to estimate exposures in workers with similar job histories for which blood data were not available. Estimates of daily and total intakes of dieldrin were calculated for each member of the cohort and three levels of exposure (low, moderate and high) were identified; the average daily intake of dieldrin was calculated as 488 µg/day, ranging from 12 to 7000 µg/day. The standard mortality ratio for the exposed cohort was lower than expected based on the mortality of the general population. No increases were observed in the cancer mortality and no specific cancer sites predominated. In a follow-up study, de Jong et al. (1997) extended analysis of the cohort up to January 1993. Total mortality and total mortality from cancer were lower than expected. The number of deaths (6) from rectal cancer was significantly higher than expected (1.5) (SMR = 390.4 [95% CI:143-850]); however there was not a clear dose relationship (SMR 865 [95% CI: 174-2526], 201 [95% CI :3-1117], and 289 [95% CI: 32-1044] in the low-, moderate- and high-exposure groups, based on 3, 1, and 2 deaths, respectively). Two deaths (one each in the low- and moderate-exposure groups) from liver cancer were not significantly higher than expected (0.9) (SMR = 225 [95% CI: 27-813]). The authors concluded that the study does not provide support for the carcinogenicity of aldrin and dieldrin in exposed workers.

Animal Studies

Several subchronic and chronic oral toxicity studies have been conducted in rodents and subchronic studies in dogs exposed to aldrin in the diet (U.S. EPA, 1987a, 2003a,b; WHO, 1989; Bus and Leber, 2001). Hepatotoxicity (hepatomegaly, histopathological degenerative changes) and neurotoxicity (degenerative brain histopathology, hypersensitivity, twitching, tremors and convulsions) were the major effects observed in exposed rats, mice and dogs. Hepatic carcinogenicity was reported in oral studies in mice, but not in rats (U.S. EPA, 1987b, 2003a). The oral database for aldrin also includes developmental toxicity studies in dogs, hamsters, rats and mice, and reproductive toxicity studies in dogs, rats and mice (U.S. EPA, 1987a, 2003b; WHO, 1989; Bus and Leber, 2001). These studies reported reduced fetal survival and/or increases in malformations at doses that were maternally toxic.

Subchronic oral toxicity studies on aldrin examined a limited array of endpoints. Treon and Cleveland (1955) conducted intermediate mortality studies on dogs and rats exposed to aldrin (95% purity) in the diet. Groups of beagle dogs (2-3 male and 2 female) were given diets containing 1, 3, 10, 25 or 50 ppm aldrin for 5 or 6 days per week for up to 15.6 months. Other methods, such as vehicles to dissolve aldrin, were not described systematically, but are inferred

from the reported results. Body weight was recorded weekly. At termination, dogs were necropsied and organ weights were recorded for liver, kidneys, heart, brain, spleen and fat. The liver and kidneys were examined for histopathology. Hemocytology analyses were conducted, but endpoints were not specified. Doses between 0.9 and 9.1 mg/kg-day (10-50 ppm levels) were lethal to all dogs. All dogs that received doses between 0.9 and 1.8 mg/kg-day (10 ppm group) died within 6.7 months. No deaths occurred among dogs exposed to 3 ppm (0.12-0.25 mg/kg-day) or 1 ppm (0.043-0.091 mg/kg-day) for up to 15.6 months. Aldrin had no effect on body weight or hemocytology parameters (not specified). Treatment at ≥ 3 ppm increased the absolute and relative liver weights in both sexes. Histopathology of the liver (local hyaline droplet degeneration) and kidneys (renal tubular degeneration or vacuolization) was observed in male dogs at ≥ 3 ppm and female dogs at ≥ 1 ppm. Male and female dogs that died (that is, those exposed at ≥ 10 ppm or ≥ 0.9 mg/kg-day) had diffuse degenerative changes in the liver, kidneys and brain. The 1 ppm dietary level (0.043-0.091 mg/kg-day) was a LOAEL for renal histopathology in female dogs. A NOAEL was not identified.

NCI (1978) conducted subchronic range-finding feeding bioassays on aldrin (technical grade, $>85\%$ purity) to establish maximum tolerated doses for carcinogenicity studies in rats and mice. Groups of Osborne-Mendel rats (5/sex/group) were given diets containing 0, 40, 80, 160 or 320 ppm of aldrin for six weeks and observed for an additional two weeks. Using reference values for body weight and food consumption (U.S. EPA, 1988), doses are estimated as 0, 3, 7, 14 and 28 mg/kg-day for male and 0, 4, 8, 16 and 32 mg/kg-day for female rats. Dose-related mortality was observed in both sexes at ≥ 160 ppm. Body weight gain was depressed consistently in males at 320 ppm. No additional information was provided. No effects were observed at the 80 ppm dietary level (7 or 8 mg/kg-day in male or female rats respectively), based on limited evaluations.

In the subchronic range-finding assay in B6C3F₁ mice (5/sex/group), NCI (1978) provided diets containing 0, 2.5, 5, 10, 20, 40 or 80 ppm of aldrin technical grade, $>85\%$ purity) for six weeks and then control diets for two weeks. Using reference values for body weight and food consumption (U.S. EPA, 1988), doses are estimated as 0, 0.5, 1, 2, 4, 7 and 14 mg/kg-day in male and 0, 0.5, 1, 2, 4, 8 or 16 mg/kg-day in female mice. Dose-related increases in mortality were observed in both sexes at ≥ 20 ppm. Aldrin had no effect on body weight gain. No additional information was provided. No effects were observed at the 10 ppm dietary level (4 mg/kg-day) in male or female mice, based on limited evaluations.

A single new oral study was located in the literature search. Paul et al. (1992) evaluated the effect of subchronic oral exposure to aldrin or endosulfan on muscle coordination, learning and memory in rats. Groups of Wistar rats (10/sex/group) were given 0 or 1 mg/kg-day of aldrin by gavage in an aqueous suspension with tragacanth powder daily for 90 days. Motor coordination (balancing on a moving rod) was measured prior to exposure and on every 15th day of treatment. Unconditioned and conditioned avoidance tests were conducted after 90 days of

treatment. Rats were tested for their ability to learn the correct behavior (pole climbing) for avoiding a buzzer/shock in 15 trials; subsequently (the next day) the time for their conditioned response to the buzzer alone was measured in 15 trials. The authors reported that treatment with aldrin had no effect on growth or behavior but did not report any methods or data for these endpoints. Treatment reduced the ability of male and female rats to remain balanced on a moving rod, indicating impairment of motor coordination; male were more severely affected than females. Treated male and female rats demonstrated an inhibition in the ability to learn the correct avoidance behavior compared to controls; 6 trials were needed before all controls were successful, whereas 12 trials were needed before all treated rats were successful. The responding time of all rats to a conditioned stimulus declined with each repetition, reaching a constant value after the 7th trial; however, the response time was slightly slower (about four seconds longer) for treated rats compared to controls. This study identifies a LOAEL of 1 mg/kg-day for neurological impairment (reduced motor coordination, learning ability and delayed response to conditioned stimulus) in rats exposed by gavage for 90 days.

No additional chronic oral animal data were found in the literature search beyond those studies already reviewed by U.S. EPA (1987a, 2003a). Thus, there is no new information to challenge the basis for the chronic RfD on IRIS, namely, the estimated LOAEL of 0.025 mg/kg-day in the 2-year dietary study in rats (Fitzhugh et al., 1964).

Reproduction studies in dogs did not establish no-effect levels for aldrin (WHO, 1989; U.S. EPA, 2003b). Deichmann et al. (1971) administered aldrin (95% purity) by capsule to groups of beagles at doses of 0.15 mg/kg-day (4 females) or 0.3 mg/kg-day (4 males, 3 females) 5 days/week for 14 months. Dogs were mated and the viability of pups recorded. Estrous cycles were delayed 7 to 12 months in treated females and some males exhibited a depressed sexual drive. Mammary development and milk production were severely depressed in treated females. A dose-related decrease in pup survival at weaning was observed: 85, 75 and 44% in the control, low- and high-dose groups, respectively. The low dose of 0.15 mg/kg-day is a LOAEL for reproductive effects (reduced pup survival) in dogs.

In another reproduction study in dogs, Kitselman (1953) fed aldrin (99% purity) in dosed meatballs to groups of mongrel dogs (1-2/sex/group) at doses of 0, 0.2, 0.6 or 2.0 mg/kg-day for one year. Aldrin had no apparent effect on fertility or pregnancy rates. In all treated groups, pups were born with no obvious defects, but died within three days and exhibited degenerative changes in the liver and renal tubules. Treated females also exhibited degenerative changes in the liver. The low dose of 0.2 mg/kg-day in this study is a LOAEL for reproductive toxicity (hepatic and renal toxicity in pups exposed during gestation and hepatic toxicity in bitches).

Reproductive and developmental oral toxicity studies for aldrin were also conducted in rodents (WHO, 1989; U.S. EPA, 2003b). In a three-generation study, Treon and Cleveland (1955) fed groups of male and female Carworth rats (group sizes not reported) diets containing

0, 2.5, 12.5 or 25 ppm of aldrin. U.S. EPA (2003b) estimated doses as 0, 0.125, 0.624 and 1.25 mg/kg-day. Two litters were produced for each generation. Treatment had no effect on numbers of live pups per litter or pup weights at weaning (postnatal day 21), but reduced the viability of pups during lactation in a dose-specific manner. The low dose of 0.125 mg/kg-day is a LOAEL for reduced viability in rat pups.

In a six-generation study, Keplinger et al. (1970) fed Swiss white mice (4 males and 14 females per group) diets containing 0, 3, 5, 10 or 25 ppm of aldrin (purity not reported). U.S. EPA (2003b) estimated doses as 0, 0.45, 0.75, 1.5 or 3.75 mg/kg-day. The 3.75 mg/kg-day dose was discontinued because of high litter mortality. Reduced pup survival during lactation was observed in the 0.75 and 1.5 mg/kg-day groups. No effects on fertility, viability or gestation were observed in the 0.45 mg/kg-day group. The lowest dose of 0.45 mg/kg-day was a NOAEL and 0.75 mg/kg-day was a LOAEL for reduced pup survival during lactation in mice.

Ottolenghi et al. (1974) conducted developmental toxicity studies in which pregnant mice (groups of 10) or hamsters (groups of 41-43) were given a single gavage dose of aldrin (in corn oil) at half the median lethal dose during gestation; the studies included both untreated and vehicle-only controls. In pregnant CD-1 mice, treatment with aldrin at 25 mg/kg-day on gestational day (GD) 9, had no effect on fetal survival or body weight. However, the number of live fetuses with malformations (webbed feet, cleft palate and open eyes) was significantly increased (33%). Maternal toxicity was observed. In pregnant Syrian golden hamsters, treatment with 50 mg/kg-day on GD 7, 8 or 9 significantly reduced the number of live fetuses and fetal weight and increased the incidence of fetal abnormalities (webbed feet, cleft palate and open eyes).

No adequate inhalation toxicity studies in animals were found in the review documents or the literature search. According to ATSDR (2002), Treon et al. (1957) exposed animals to unknown concentrations of aldrin vapor/particles generated by sublimating aldrin at 200°C; however, these studies are confounded by the presence of uncharacterized thermal decomposition products.

Other Studies

Absorption of aldrin in humans or animals occurs following exposure by any route (ATSDR, 2002). Since aldrin is readily metabolized in both humans and animals, it is rarely detected in distribution studies; the metabolite dieldrin is the primary marker for aldrin exposure. Based on studies in volunteers and human cadavers, the relative steady-state distribution of dieldrin in human whole blood, brain gray matter, brain white matter, liver and adipose tissue is estimated as 1, 2.8, 4.2, 22.7 and 136, respectively (U.S. EPA, 2003b). Dieldrin persists in the body with a mean half-life of ~8 months (de Jong, 1991). In humans and rodents, dieldrin has

been detected in breast milk and has been shown to pass through the placenta (ATSDR, 2002; U.S. EPA, 2003b).

The biotransformation of aldrin to dieldrin has been detected in the liver, lung and skin, (ATSDR, 2002; U.S. EPA, 2003b). Mixed function oxidases (cytochrome P-450) are responsible for the epoxidation of aldrin to dieldrin in mammalian hepatocytes; rates of conversion are higher in male rats and mice than in females. *In vitro* studies suggest that the conversion of aldrin in tissues with a low cytochrome P-450 content is carried out by an arachadonic acid-dependent prostaglandin endoperoxide synthase pathway (ATSDR, 2002; U.S. EPA, 2003b). 9-Hydroxydieldrin has been identified as a fecal metabolite in humans and animals. Other metabolites include pentachloroketone, 6,7-trans-dihydroxydihydroaldrin (and its glucuronide conjugate), the glucuronide conjugate of 9-hydroxydieldrin, and aldrin dicarboxylic acid. The relative proportion of the metabolites varies by species, strain and sex (U.S. EPA, 2003b).

In humans and rodents, excretion following oral exposure is primarily in the feces via the bile, with smaller amounts in the urine (ATSDR, 2002). Excretion via lactation has also been described in for humans and rodents (ATSDR, 2002; U.S. EPA, 2003b).

Neurological effects of aldrin have been attributed to alterations in brain neurotransmitters, specifically GABA (γ -aminobutyric acid) (Ecobichon, 2002). Treatment of rats with single gavage doses between 2 and 10 mg/kg or with 2 mg/kg-day for 12 days altered GABA parameters in the brain and increased locomotor activity within two hours of administration (Jamaluddin and Poddar, 2001a,b). The increased locomotor activity was attributed to an activation of glutamate and concomitant inhibition of the GABA system in the cerebellum, hypothalamus and pons-medulla. In a biophysical study, aldrin was demonstrated to change the fluidity of model phospholipid bilayers, increasing the fluidity of bilayers enriched with cholesterol (Demétrio et al., 1998). The results of this study suggest that *in vivo*, intercalation of aldrin into the hydrophobic layer of neuronal cell membranes may contribute to the perturbed function of embedded proteins such as the GABA receptor.

Daily subcutaneous administration of aldrin to pregnant Wistar rats at a dose of 1 mg/kg-day throughout gestation had no teratogenic effect and no effect on body weights in pups, although subtle effects were observed in pups postnatally (Castro et al., 1992). Treated pups showed significant changes in the average timing of certain developmental landmarks; the appearance of incisor eruption was accelerated (on day 4.4 compared to 6.6 for controls), whereas the descent of the testes was delayed (day 31.5 compared to 21.0 for the controls). The timing of pinna detachment, fur development, ear opening and eye opening were not significantly affected. Treated pups also showed significantly higher scores in locomotor frequency tests conducted at 21 and 90 days. Histological examination of brain sections did not reveal overt changes in brain structure that could have contributed to the behavioral effects.

Genotoxicity assays for aldrin were primarily negative in bacteria, but were occasionally positive in mammalian systems, possibly reflecting a requirement for bioactivation. In several studies, aldrin did not induce reverse mutations in *Salmonella typhimurium* (strains TA98, TA100, TA1535, TA1537 or TA1538), *Escherichia coli* or *Bacillus subtilis*, with or without metabolic activation (U.S. EPA, 1987b, 2003a,b; ATSDR, 2002). With or without metabolic activation, aldrin did not cause gene conversion, but did induce reverse mutations in *Saccharomyces cerevisiae* (U.S. EPA, 2003b). In a mouse dominant lethal mutation assay, aldrin reduced the levels of implantations, but the results were not statistically significant (U.S. EPA, 2003b). Aldrin did not induce heritable sex-linked recessive lethal mutations in *Drosophila melanogaster* (U.S. EPA, 2003b). Aldrin induced chromosomal aberrations in human lymphocytes *in vitro* and rat and bone marrow cells of orally-exposed mice, but only at cytotoxic concentrations (U.S. EPA, 2003a,b; ATSDR, 2002). Results of assays for unscheduled DNA synthesis were negative in primary rat hepatocytes and human lymphocytes, but were positive in transformed human fibroblasts *in vitro* (U.S. EPA, 2003b). Aldrin did not induce breakage of plasmid DNA in *E. coli* in the absence of metabolic activation but did induce DNA breakage in rat hepatocytes (U.S. EPA, 2003b).

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC ORAL RfD VALUES FOR ALDRIN

A **chronic RfD of 3E-5 mg/kg-day** is listed for aldrin on IRIS (U.S. EPA, 2003a), based on a LOAEL of 0.025 mg/kg-day for hepatotoxicity (centrilobular lesions and increased liver weight) in rats exposed to 0.5 ppm of aldrin in the diet for two years (Fitzhugh et al., 1964). The presence of a chronic RfD on IRIS precludes derivation of a provisional chronic RfD for this chemical.

The data were reviewed in order to determine the most suitable basis for derivation of a subchronic RfD. Neurotoxicity is the primary effect of aldrin exposure in humans. Aldrin intoxication has been observed following exposures that resulted in a dieldrin concentration in blood higher than 200 ng/mL (de Jong, 1991). The body-burden of dieldrin, which persists in fatty tissue, determines whether a particular exposure would increase the blood concentration of dieldrin above the critical level. Induction of liver enzymes in humans has a lower threshold for dieldrin in blood: 105 ng/mL (de Jong, 1991). Hepatic and neurological changes have been described in rodents orally exposed to aldrin. Hepatic effects (increased organ weight, enlarged centrilobular hepatocytes) were observed in Osborne-Mendel rats exposed to aldrin at a dietary concentration of 0.5 ppm for two years, and nephritis was observed at higher exposure levels (Fitzhugh et al., 1964); the study did not establish a NOAEL. This LOAEL, estimated at 0.025 mg/kg-day, was the lowest effect-level identified among the numerous oral toxicity studies

reviewed by U.S. EPA (1987a, 2003a) and, as mentioned above, was the basis for the chronic RfD of $3\text{E-}5$ mg/kg-day on IRIS, as well as the subchronic RfD in the HEAST (U.S. EPA, 1997).

Most of the available subchronic toxicity information for aldrin is in studies that were conducted prior to the establishment of current standard protocols. A LOAEL of 0.043 mg/kg-day was identified for renal toxicity (renal tubular degeneration) in beagles exposed to aldrin at a dietary concentration of 1 ppm for nearly 16 months (Treon and Cleveland, 1955). Renal and hepatic effects (degenerative lesions) were observed at the 3 ppm dietary level (0.12-0.25 mg/kg-day) and degenerative lesions in brain, liver and kidneys were observed in dogs fed at or above the 10 ppm level (≥ 0.9 mg/kg-day). The only newly-located study reported changes in neurobehavioral parameters (impairments in motor coordination, learning and conditioned response times) in rats given 1 mg/kg-day of aldrin by gavage for 90 days (Paul et al., 1992). The NCI (1978) 6-week range-finding studies in rats and mice did not evaluate any endpoints aside from mortality and body weight effects; therefore, the apparent NOAELs in these assays (8 mg/kg-day in rats and 4 mg/kg-day in mice) are not supported by histopathology data. Aldrin had adverse effects on reproduction in dogs exposed in the diet at doses of 0.15 mg/kg-day or higher (Deichmann et al., 1971): delayed estrus and reduced mammary development and milk production in females, reduced sex drive in males, and reduced pup survival at weaning. Another reproductive study reported no effects on fertility or pregnancy rates in dogs at ≥ 0.2 mg/kg-day, but increases in degenerative hepatic lesions in bitches and increased postnatal mortality of pups, concomitant with degenerative hepatic and renal lesions (Kitselman, 1953). In a 3-generation feed study in rats, reduced pup survival at weaning was observed at ≥ 0.125 mg/kg-day (Treon and Cleveland, 1955). In a 6-generation study in mice, 0.45 mg/kg-day was a NOAEL and 0.75 mg/kg-day was a LOAEL for reduced pup survival during lactation (Keplinger et al., 1970). A single gavage dose of 25 mg/kg during gestation increased the incidence of fetal abnormalities but did not affect fetal survival or body weight in mice (Ottolenghi et al., 1974). In hamsters, a single gavage dose of 50 mg/kg during gestation reduced fetal survival and body weight and increased the incidence of fetal abnormalities (Ottolenghi et al., 1974). Reproductive and developmental effects of aldrin appear to occur at maternally-toxic doses.

The LOAEL of 0.043 mg/kg-day for renal lesions in the 16-month toxicity study in beagles (Treon and Cleveland, 1955) can serve as the basis for the subchronic RfD for aldrin. Since developmental studies indicate that fetal effects of aldrin occur at maternally-toxic doses, an RfD based on this LOAEL should be protective against fetal effects (U.S. EPA, 2003b). The provisional **subchronic RfD of $4\text{E-}5$ mg/kg-day for aldrin** is derived by applying an uncertainty factor of 1000 (10 to extrapolate from dogs to humans, 10 to protect sensitive individuals and 10 for the use of a LOAEL) to the dog LOAEL of 0.043 mg/kg-day, as follows:

$$\begin{aligned}
 \text{subchronic p-RfD} &= \text{subchronic LOAEL} / \text{UF} \\
 &= 0.043 \text{ mg/kg-day} / 1000 \\
 &= 0.00004 \text{ or } 4\text{E-}5 \text{ mg/kg-day}
 \end{aligned}$$

Confidence in the critical subchronic study is low because, although it evaluated a range of doses, and included examinations for histopathology and hemocytology, it had relatively small group sizes, omitted some toxicological endpoints (clinical chemistry, and urinalysis), did not identify a NOAEL, and was poorly documented. Confidence in the database is medium since reproductive studies were available, but NOAELs were lacking for some supporting studies. Low-to-medium confidence in the provisional subchronic RfD for aldrin results.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfC VALUES FOR ALDRIN

No human or animal inhalation data were located, precluding derivation of a subchronic or chronic p-RfC for aldrin.

DERIVATION OF A PROVISIONAL CARCINOGENICITY ASSESSMENT FOR ALDRIN

A cancer assessment, including derivation of an oral slope factor of 17 per mg/kg-day and an inhalation unit risk $4.9\text{E-}3$ per $\mu\text{g}/\text{m}^3$, is available for aldrin on IRIS (U.S. EPA, 2003a), precluding derivation of a provisional carcinogenicity assessment for this chemical.

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10-23-2006

Provisional Peer Reviewed Toxicity Values for
Aluminum
(CASRN 7429-90-5)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
i.v.	intravenous
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR ALUMINUM (CASRN 7429-90-5)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

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INTRODUCTION

Verified toxicity values for aluminum (Al) and its compounds are unavailable on IRIS or HEAST (U.S. EPA, 2006, 1997), except for a chronic oral RfD of 4E-4 mg/kg-day for aluminum phosphide. However, occupational guidelines and standards have been established for a number of chemical and physical forms of Al, including, from ACGIH, 8-hour TWA-TLVs of 10 mg/m³ for the compound as a metal dust or oxide, 5 mg/m³ as "pyro" powders or welding fumes, and 2 mg/m³ for soluble salts or organic forms of the metal (ACGIH, 1998). From NIOSH, 10-hour TWA-RELs of 10 mg/m³ are specified for "total" Al dust versus 5 mg/m³ for the respirable portion (NIOSH, 1994). NIOSH covers all other forms of the metal by identical values to those specified by ACGIH (ACGIH, 1998). OSHA PELs for Al include an 8-hour TWA value of 15 mg/m³ for "total" metal dust, versus 5 mg/m³ for the respirable portion (NIOSH, 1994). The U.S. EPA's CARA list (U.S. EPA, 1994) cites a HEA for Al (U.S. EPA, 1987), and ATSDR has updated its toxicological profile of the element (ATSDR, 1998).

The U.S. FDA (2000) has specified a maximum aluminum concentration of 25 mcg/L in large-volume parenterals (LVP) used in total parenteral nutrition (TPN). The FDA regulation applies to all LVPs used in TPN, including but not limited to parenteral amino acid solutions, highly concentrated dextrose solutions, parenteral lipid emulsions, sodium chloride and electrolyte solutions, and sterile water for injection.

Research papers pertinent to the potential toxicological and carcinogenic effects of Al were sought through computer searches of the HSDB, RTECS, MEDLINE and TOXLINE (and its subfiles) databases, covering the time period 1995-1999. The literature searches were conducted in June, 1999.

REVIEW OF PERTINENT DATA

The review by Stokinger (1981) gives an account of Al as an all-pervasive component of products that are central to the daily lives of most Americans. For example, the metal is a crucial part of manufactured products for the building, automobile and container industries, while Al as powder or flake is a component in a number of consumer products, such as paints, fireworks, etc. Al complexes and minerals are used in the brewing and paper industries, and as coagulants for water purification. Aluminum oxide finds application in abrasives, as a catalyst or absorbent, and as a component in fillers. Aluminum chloride is included in cosmetic formulations such as deodorants.

Human exposure to Al arises principally from food and water, through its widespread use in food additives, packaging and cooking utensils and Al-containing medications, particularly antacid, buffered aspirin, anti-ulcer and anti-diarrheal formulations (Marquis, 1989; Lione, 1985). Pennington and Schoen (1995) estimated daily Al intakes of 0.1-0.3 mg/kg-day for infants and children 6 months-6 years of age and 0.1-0.18 mg/kg-day for older children and adults, based on the FDA Total Diet Study (1993) and the U.S. Department of Agriculture Nationwide Food Consumption Survey (1987-1988). These data are in broad agreement with those of Wilhelm et al. (1995) who reported the dietary intake of Al in German children (living in the Duisberg area) as ranging from 0.008 to 0.11 mg Al/kg-day. In addition, these values are consistent with a range of 1-20 mg/day (0.014-0.3 mg/kg -day) for normal oral daily Al intake from food and water reported by other investigators (Ganrot, 1986; Iyengar et al., 1987; Wilhelm et al., 1990). However, users of Al-containing medications can ingest much larger amounts of the element, possibly as high as 840-5000 mg/day (12-71 mg/kg-day) from antacids, 126-728 mg/day (1.8-10.4 mg/kg-day) from buffered aspirins and 828 mg/day (11.8 mg/kg-day) from anti-ulcer compounds when taken at recommended dosages (Lione, 1985).

Toxicokinetics of Aluminum

There is a large amount of information available on the absorption, transfer from tissue to tissue and elimination of Al from the body, including data that have been amassed from studies on either human volunteers or laboratory animals. In general, the chemical appears to be poorly absorbed from the gastrointestinal tract, though the portion of the load that is retained will vary depending on the concentration, the chemical species administered, the fasting or fed state of the

host, gastrointestinal pH, animal model, etc. For example, Yokel and McNamara (1988) administered single oral doses of a number of Al compounds (both water soluble and insoluble) to New Zealand white rabbits and obtained absorbed proportions of the load ranging from 0.27% to 27%. Fractional uptake of Al in humans under normal conditions (i.e., with no intake of large quantities of Al from medicine) was estimated to be 0.1-0.3% assuming an intake of 20 mg Al/day (0.3 mg Al/kg-day) and urinary excretion of 20-50 μg Al/day (0.3-0.7 μg Al/kg-day) (Ganrot, 1986). However, little information is available on the actual mechanism by which the element and its compounds are transported across the brush border. (Wilhelm et al., 1990; Lione, 1985).

Although the overall extent of Al absorption is poor following oral exposure, there may be significant intake of the compound by those taking large amounts of Al compounds in patented remedies. As stated, absorption of Al is influenced by gastrointestinal conditions and content because Al can form various complexes with different solubilities and oxidation states depending on pH and interactions with dietary constituents. At low pH (3-5) in aqueous solutions, the soluble (ionic) forms of the Al prevail (Al^{3+}); at high pH (>8), Al in the form of soluble aluminum oxide is present; and at pH 5-8, the element is predominantly in the form of aluminum hydroxide, which is insoluble (van der Voet and de Wolff, 1986; Wilhelm et al., 1990). Ingested constituents that can influence absorption by forming complexes with Al include phosphate, fluoride, calcium, citrate and lactate. For example, Al is used to bind dietary phosphorus and decrease its absorption as a control for hyperphosphatemia, and citrate and lactate are complexing agents that can significantly increase Al absorption (Slanina et al., 1984, 1985, 1986; Partridge et al., 1989; Domingo et al., 1991; Ittel et al., 1991; Lione, 1985; Wilhelm et al., 1990).

A number of recent reports of studies on the gastrointestinal absorption of Al have examined the influence of organic anions such as citrate. In general, the presence of such components appears to enhance the absorption of Al, within narrow limits. For example, Deng et al. (1998) administered a single oral dose of either distilled water, 2 mmoles/L aluminum chloride or 2 mmoles/L aluminum chloride plus 2 mmoles/L sodium citrate to six male Wistar rats/group. Animals were bled at 1, 2 and 4 hours after dosing, then terminated after 6 hours. Inductively coupled plasma (ICP) was used to measure Al concentrations in blood, bone (tibia), kidney, liver and the intestinal wall. Irrespective of treatment, the appearance of Al in the blood of dosed groups peaked after 1 hour, with the concentration of the element at higher levels in those animals receiving citrate in addition to aluminum chloride. In those animals receiving aluminum chloride alone, significant tissue concentrations of the element were restricted to the gastrointestinal wall. Those receiving citrate displayed measurable quantities of the element in several of the other monitored tissues, including bone.

Sutherland and Greger (1998a) used a similar dosing regimen to examine the kinetics of absorption and elimination of Al in male Sprague-Dawley rats that had received a single oral dose of 0, 0.25, 0.5 or 1 mmoles/L/kg body weight aluminum lactate in 1 mL of 16% citrate. Concentrations of Al in serum, liver, kidney or bone (tibia) were measured at various post-dosing time intervals up to 6 hours. Depending on the dose, absorption factors for Al of up to 4.2% of the administered dose were observed, with the greater proportion retained in bone. The authors reported a slower rate of absorption in those animals receiving Al at the higher doses, an

observation potentially indicating reduced gut motility and/or saturation of the transcellular absorption processes at the higher concentrations. Aluminum deposited in kidney and bone appeared to turn-over at a slower rate than in the liver.

The influence of citrate on the gastrointestinal absorption of Al in man was examined directly by Taylor et al. (1998) who administered a drink containing Al and citrate to three volunteers. Aluminum and citrate concentrations were monitored in serial blood and urine samples for up to 24 hours. The kinetics of citrate and Al differed markedly, the former peaking in plasma after 32 minutes, versus 87 minutes for Al. This suggests that Al probably does not cross the gastrointestinal barrier as the citrate. Furthermore, the authors reported that the overall extent of Al absorption had probably not exceeded 1% in their experiment, a finding that contrasts with the higher values reported by Sutherland and Greger (1998a) in Sprague-Dawley rats and by Deng et al. (1998) in Wistar rats.

As discussed in a report by Glynn et al. (1999), gastrointestinal absorption of Al from aqueous media will be almost impossible to predict, because of the likelihood that the element will become absorbed to food particles in the intestinal lumen. Accordingly, depending on the dose, mode of delivery and caloric state of the experimental animal (fed/fasted), significant amounts of aqueous forms of Al will be absorbed only when available binding sites on food have become saturated. This presents an inherently complex overall picture of the element's absorption since, additionally, the normal dietary content of Al will be substantial. Thus, it may be assumed that some sequestered Al will be absorbed along with non-sequestered water soluble forms of the element, while the rest will be retained within the gastrointestinal tract.

Sutherland and Greger (1998b) used their aluminum lactate in 16% citrate dosing regimen to examine the comparative importance of biliary versus urinary excretion of Al. Five to seven male Sprague-Dawley rats/group who had previously received an implanted bile cannula were treated by gavage. Another similarly-treated cohort of five animals/group were housed in metabolic cages immediately after dosing to provide 0- to 3-hour and 3- to 6-hour urine specimens. At termination, all animals were sacrificed and exsanguinated, and tissue, bile and urine samples were measured by graphite furnace atomic absorption spectroscopy. Among the key findings to emerge from this study was the incremental appearance of Al in bile as early as 15 minutes after dosing. However, overall amounts of Al were greater in the 3-hour urine samples than those that had accumulated in bile samples collected within a similar time frame. The fact that control rats excreted 3 times more Al in bile than in urine during the first 3 hours after dosing led the authors to conclude that, at low exposure to Al (in controls receiving Al solely from food), the liver is capable of excreting the element to the bile, a mechanism that becomes saturated as the level of Al administration becomes increased. Thereafter, urinary excretion becomes the primary route of elimination in circumstances of Al overload.

Aluminum can also be absorbed by inhalation as indicated by age-related deposition in the lungs of the general population and exposure-related increased blood and urine concentrations in workers exposed to Al (Bast-Pettersen et al., 1994; Sjogren et al., 1996; Hosovski et al., 1990; Wilhelm et al., 1990; U.S. EPA, 1987). Aluminum occurs primarily in particulate form in the ambient atmosphere and as various dusts and fumes during its production and use. Common forms of inhaled Al include aluminum oxide (alumina; Al_2O_3), pyro powders

(powder and flake Al-treated to reduce surface oxidation), Al welding fume and soluble salts (e.g., aluminum chloride and sulfate) (ACGIH, 1998).

Neurotoxicity as a Primary Toxicological Effect of Aluminum

One of the greatest health concerns regarding Al is its neurological effects. The first evidence for Al-induced neurotoxicity in humans was seen in patients who, as a result of receiving long-term hemodialysis for chronic renal failure, developed a degenerative neurological syndrome (dialysis dementia) characterized by the gradual loss of motor, speech and cognitive functions (Alfrey, 1993). This dementia, attributable to Al in the dialysate, is usually fatal within 6-9 months after the first clinical signs appear. In addition, many patients received high oral doses of Al to act as phosphate binders. Autopsies of these patients revealed increased concentrations of Al in the gray matter and cerebral spinal fluid (CSF) but no evidence of neurofibrillary degeneration (NFD) despite the elevated Al levels. Once the connection between Al and dialysis dementia was established, Al was removed from dialysis fluid and the incidence of dementia rapidly declined, thereby strengthening the argument that Al was a causal agent in dialysis dementia (Ganrot, 1986).

Amyotrophic Lateral Sclerosis (ALS) and Parkinson's Disease (PD) are other neurological diseases which have been associated with Al exposure. ALS is a progressive disease of the Central Nervous System (CNS) that is characterized by an accumulation of neurofibrillary tangles. In Guam, southern West New Guinea and parts of Japan, there is an unusually high prevalence of ALS and PD. This may be related to the natural abundance of Al coupled with the virtual lack of magnesium and calcium in the drinking water supplies and soil of these areas. In a study designed to evaluate effects of high Al and low calcium levels in the diet, much like the conditions associated with Guam and other similar areas, cynomolgus monkeys were placed on a low calcium diet either with or without supplemental Al and manganese (Garruto et al., 1989). Chronic calcium deficiency alone produced neurodegenerative effects, although neurofibrillary changes were most frequently seen in the monkeys on a low calcium diet supplemented with Al and manganese.

Though a cause and effect relationship between Al and three forms of chronic encephalopathy in humans: senile dementia of the Alzheimer type (SDAT, Alzheimer's Disease), endemic Amyotrophic Lateral Sclerosis (ALS) and endemic Parkinsonism-dementia (PD, a mixture of Parkinsonism and senile dementia) has been suggested, there is no firm evidence that it plays a causal role in the development of these diseases (Ganrot, 1986; Lione, 1985). The condition is degenerative and characterized by the progressive loss of speech, motor and cognitive functions, with death typically occurring within 1-6 months. Autopsies of patients revealed increased concentrations of Al in the gray matter and cerebral spinal fluid (CSF), though with no conclusive evidence of NFD or other neuropathological changes despite the elevated Al levels.

The neurotoxicity of Al is well documented in certain animal species. Aluminum induces a spectrum of behavioral abnormalities and brain neurofibrillary degenerative changes in rabbits and cats when injected intracranially or parenterally in high doses, though hamsters and monkeys are less sensitive (Ganrot, 1986; Lione, 1985). Such studies have been designed as models for

the possible neurotoxicological effects of Al in humans. However, it should be noted that the neurofibrillary changes in affected animals differ in morphological detail from those associated with SDAT. As discussed further in the Oral Toxicity section, oral doses of Al can also induce neurobehavioral effects in adult mice and rats and in their developing offspring. In general, such neurotoxic effects of Al appear to be more subtle than those induced through routes of administration that by-pass the gastrointestinal tract, perhaps reflecting the lower doses of Al reaching the brain.

Recent reports of studies on the effects of Al on neurotoxicity in animals have sought to define the biochemical mechanisms that are impaired when Al crosses the blood-brain barrier. However, a unifying concept has yet to emerge, though the passage of the element into various regions of the brain has been clearly demonstrated (Deloncle et al., 1995). Among the many biochemical functions and processes that appear to be perturbed by the presence of Al in the brain are the peroxidation status of biological membranes (Katyal et al., 1997; Deloncle et al., 1999), inhibition of the neuronal glutamate-nitric oxide-cyclic GMP pathway (Cucarella et al., 1998), and the marked reduction of protein- and non-protein-bound thiols and the specific activity of Na^+/K^+ and Mg^{++} ATPases (Katyal et al., 1997). The relative importance of each of these mechanisms and how (or whether) they interact to bring about the observed physiological changes remains unclear.

Other Effects of Aluminum

Osteomalacia was frequently observed among long-term dialysis patients with neurological signs and is commonly attributed to Al overload (Ganrot, 1986; Lione, 1985). This bone condition is characterized by widened osteoid (unmineralized bone matrix) with no fibrosis, reduced mineralization rate, skeletal pain and a strong tendency for fractures, lack of response to vitamin D therapy and increased Al concentration in bone. Effects on bone histology and elevated bone Al levels have also been observed in patients with normal renal function who received total parenteral nutrition with Al-contaminated casein as a protein source, and in parenteral Al loading induced osteomalacia in rats and dogs (Lione, 1985).

There are a number of published reports of studies in which the carcinogenicity of aluminum compounds has been evaluated. These include oral exposure studies in which the compounds were made available to experimental animals in the drinking water or diet (Schroeder and Mitchener, 1975a,b; Oneda et al., 1994), and inhalation epidemiological studies, in which the incidence of tumor formation in persons exposed to aluminum-containing dusts and fumes in an occupational setting was compared to unexposed individuals (Spinelli et al., 1991; Thériault et al., 1984, 1990; Armstrong et al., 1986; Tremblay et al., 1995; Selden et al., 1997; Cullen et al., 1996; Dufresne et al., 1996; Ronneberg and Langmark, 1992). However, it has been generally concluded that the inferential association between exposure to Al and marginally increased incidences of tumors of the bladder and/or lung are confounded because of the co-exposure of subjects in such settings to other harmful and potentially carcinogenic substances, such as polycyclic aromatic hydrocarbons (PAHs and coal tar pitch volatiles (CTPV) (Ronneberg and Langmark, 1992). Therefore, the issue of the potential carcinogenicity of Al compounds remains uncertain.

Human Studies

Oral Exposure

Few reports have been identified that address the toxicological effects of Al in humans exposed orally. Furthermore, in a review, Reiber et al. (1995) pointed to the conflicting findings that have been reported when the incidence of neurological symptoms has been assessed in relation to Al exposure in either cross-sectional, ecological or case-control epidemiological studies. Among the more recent studies that have used this approach, Martyn et al. (1997) discussed the findings of a case-control study involving 441 men in England and Wales who were afflicted with either Alzheimer's disease, brain cancer, dementia or other neurological conditions. Assessing the historical exposure of these subjects failed to establish a link between Al in drinking water at the prevailing concentrations (below 0.2 mg/L) and the incidence of one or more of the conditions under investigation. No data were located regarding the oral carcinogenicity of aluminum compounds in humans.

Inhalation Exposure

Neurobehavioral effects were evaluated in a group of 87 Al foundry workers who were occupationally exposed to 4.6-11.5 mg/m³ Al fumes and dust for a mean of 12.0 years [standard deviation (SD) 4.5 years, shortest exposure 6 years] compared to an unexposed control group (n=60) who were matched for age, job seniority and social status to exposed subjects (Hosovski et al., 1990). It is reported that environmental Al concentrations were measured for each worker separately during the winter and summer, implying that personal sampling may have been used and that the contributing concentrations are time-weighted averages. In certain places, the number of particles ranged as high as 329-1020/cm² air, and dust particle sizes were ≤1, 1-5 and ≤5 microns in 65.6, 26.6 and 7.6% of the samples, respectively. Tests of psychomotor ability (simple and complex reaction time, oculomotor coordination), intellectual ability (Wechsler intelligence, performance intelligence and verbal intelligence quotients and Wechsler subtests on information processing, memory, understanding, calculation, coding, picture completion, picture grouping, object assembling, assembling of cubes and common concepts) and cerebral damage (Bender visual motor test) were conducted. Performance of the exposed workers was found to be significantly (p<0.02) impaired on the complex reaction time, oculomotor coordination, memory, coding, picture completion and object assembling tests. However, the investigators noted that the performance deficits had no clinical manifestations, and that additional studies were probably needed to confirm the possibility of cerebral damage. The study yielded a lowest available non-duration adjusted LOAEL of 4.6 mg Al/m³ for psychomotor and cognitive impairment during repeated 8-hour occupational exposures (Hosovski et al., 1990), that could be corrected for discontinuous exposure (10 m³/20 m³ and 5 days/7 days) to yield a LOAEL_{HEC} of 1.64 mg/m³ Al.

Aluminum oxide powders were administered to Canadian miners (mainly underground gold and uranium miners) in known exposures as a means of prophylaxis against silicosis (Stokinger, 1981; Rifat et al., 1990). Data in which more than 42 million Al treatments (≈150,000 man-years) had been given over a period of 27 years ending in 1971 were reviewed

by Stokinger (1981). The effectiveness of this treatment is uncertain but no lung damage or other ill effects (not specified) were observed. The powders (McIntyre powder) were prepared by grinding Al pellets so that 96% of the particles were $\leq 1.2 \mu\text{m}$ in diameter. During this process most of the particles became oxidized to aluminum oxide; the powder contained 85% aluminum oxide and 15% elemental Al. According to Stokinger (1981), recommended exposure concentrations were 30,000 particles of respirable size per cubic centimeter (ppcc) for 10 minutes/day or 10,000-20,000 ppcc for 20 minutes/day (total treatment days not indicated). Rifat et al. (1990) stated that the recommended exposure was to an Al dust concentration of 20,000-34,000 parts per ml air in the miners' changing rooms before each shift for 10 minutes. Stokinger (1981) reported that the 30,000 ppcc concentration corresponds to $\approx 350 \text{ mg/m}^3$, which is equivalent to an 8-hour average concentration of 2 mg/m^3 . Based on the Stokinger (1981) data and the fact that one unspecified study used levels 30 times higher than advised, the TLV of 10 mg/m^3 is recommended for Al dust (ACGIH, 1998).

The increasing awareness of the potential neurotoxicity of Al has resulted in a number of investigations of the incidence of neurotoxicological symptoms in Al workers. Although treatment with McIntyre powder had not produced apparent adverse effects, a neurobehavioral evaluation of male miners (261 exposed to McIntyre powder, 346 unexposed) who started working between 1940 and 1979 (additional duration data not reported) was performed in 1988-1989 (Rifat et al., 1990). There were no significant differences between exposed and unexposed miners in reported diagnoses of neurological disorder. Results of cognitive testing (Mini-Mental State Examination for general cognitive function, Ravens colored progressive matrices test for reasoning and Symbol Digit Modalities Test for spatial perceptual accuracy and information processing), however, showed that the exposed group had significantly ($p \leq 0.001$) impaired performance on at least one test, and when all test scores were summed. Also, the likelihood of scores in the impaired range increased with duration of exposure.

A neurologic syndrome was described in Al smelting plant potroom workers (White et al., 1992). Twenty-five men were evaluated for suspected work-related neurologic illness based on findings in three patients studied previously. The average duration of employment was 18.7 years (SD, 3.6; range, 12-23 years), 15 of the patients were working at the time of evaluation, and 10 had taken early retirement or medical leave due to workplace-related symptoms (mean length of time since exposure was 1.3 years ranging from 0.2-5 years). Quantitative exposure level data were not reported, but 21 of the workers had been employed in the potroom prior to installation of fume hoods for a mean duration of 5.3 years (range 3-7 years). Symptoms most often reported by the patients were frequent loss of balance (88%), memory loss (84%) and joint pain (84%); other symptoms included dizziness (80%), numbness (80%), parasthesias (72%) and tremor (68%). Neurologic examinations showed mild to moderate signs of lack of coordination (tremor, dyssynergy of upper extremity limb movement or ataxia) in 84% of the patients. Neuropsychologic effects were evaluated in 21 of the patients using the Wechsler Adult Intelligence Scale-Revised (intellectual functioning), Wide Range Achievement Test-Revised (academic functioning), Halstead-Reitan Neuropsychological Test Battery (neuropsychological assessment) and Minnesota Multiphasic Personality Inventory (personality functioning). Memory function was assessed with the Wechsler Memory Scale (14 patients) and Wechsler Memory Scale-Revised (8 patients). The memory function evaluation showed mild to moderate impairment on subtests of immediate recall for verbal or visual information (70-75% of the

tested patients) and delayed verbal or visual recall (50-70%). Other effects included mild or moderate impairment on Halstead-Reitan tests of abstract reasoning and flexible thinking (42% of the tested patients), memory for tactile information (53%) and sustained attention and discrimination of tonal and speech patterns (44 and 64%, respectively). On the Wechsler memory and Halstead-Reitan tests, mild and moderate impairment was defined as scores 1.5-2 and ≥ 2 standard deviations below the mean of the normal population, respectively. Most (89%) of the patients tested with the Minnesota Multiphasic Personality Inventory had abnormally elevated scores (≥ 2 SDs above the population mean) indicative of clinical depression. Significant positive correlations were found between severity of incoordination (signs and symptoms) and degree of exposure (qualitative) before the introduction of the ventilation hoods.

White et al. (1992) noted two other studies that described neurologic problems among Al smelter workers. Thus, an evaluation of 444 electrolysis workers found neuropsychiatric changes in 123 (28%), “neurotic syndromes” in 89 (20%) and “slight pyramidal and cerebellar changes” in 39 (9%) (Langauer-Lewowicka and Braszczyńska, 1983). In the second study, symptoms including mental confusion, concentration and memory problems were described in six potroom workers (Cawthon, 1988).

In another study of Al production workers, neuropsychological effects were assessed in 38 elderly men who had been exposed for at least 10 years exclusively in the potroom (n=14), foundry (n=8) or other manual labor departments of the same plant (n=16, control group) (Bast-Pettersen et al., 1994). The mean ages and employment durations of the groups were in the ranges of 62.5-63.5 and 19.2-19.6 years, respectively. The men were examined soon after or just before retirement in 1991. Limited environmental monitoring data indicates that the degree of Al exposure varied between the subgroups and over the years. Average annual total dust concentrations in the potroom were reduced significantly from 9.5 mg/m³ in 1977 to 3.0 mg/m³ in 1990. Aluminum levels were not specifically reported, but the average Al content in the total potroom dust was approximately 20% by weight; other constituents of the dust included fluoride and coal tar pitch components. Data from an Al uptake/excretion study of workers from the same plant indicated that the level of Al exposure was approximately 8 times higher in the potroom than in the foundry (0.48 and 0.06 mg/m³, respectively) (Drablos et al., 1992). Medical examinations (including lung function, standard laboratory tests and serum and urine Al concentrations) and a neuropsychological test battery were performed. The battery assessed six mental functions (neuropsychiatric symptoms, motoric/sensoric, reaction time, psychomotor speed/efficiency, memory/learning and intelligence) using a questionnaire and 15 different objective tests. Some subtle deficits were found in potroom workers that were not considered to be indicative of a significant neurological syndrome. The findings in potroom workers included a subclinical tremor as indicated by results of a static steadiness test [time scores on one of two test indices were significantly worse in comparison with the control group (84% slower, $p=0.03$)], and possible tendencies (i.e., test results that were about 1 SD below normal mean values but not statistically significant) for increased risk of impaired visuospatial organization (Block Design subtest of the Wechsler Adult Intelligence Scale) and psychomotor tempo (one Halstead Reitan Trail Making test). Although these findings were not considered to be indicative of a neurologic syndrome, it was suggested that they may be early signs of CNS impairment. Additionally, the finding of a subclinical tremor seems to be consistent with the tremor and other

signs of incoordination observed in 84% of the patients in the White et al. (1992) study summarized above.

Studies of Al welders are consistent with those of Al smelter workers in indicating that occupational exposure to Al can be neurotoxic. CNS function was evaluated in 17 welders who had an average of 15 years (range 5-27 years) experience, with the last 4 years exclusively with Al (Hanninen et al., 1994). Most of the welders had equipment that ventilated the welding masks but the respiratory protection was not always used. The assessment included measurements of serum and urinary Al, neuropsychological tests (simple reaction time, three tests for psychomotor speed, two tests for visual and spatial ability, four memory tests and two verbal ability tests), a symptom questionnaire and neurological interview, quantitative electroencephalography (QEEG) and P-300 event-related auditory-evoked responses. Serum and urine Al levels were 3.5 and 8.5 times higher, respectively, than an unexposed reference population. The welders performed normally on the neuropsychological tests, although correlation analysis of test scores and exposure parameters showed weak negative associations between the four memory tests and urinary Al level and a positive association between the variability (standard deviation) of visual reaction times and serum Al levels. Analysis of the QEEG data showed that serum Al levels were positively correlated with the amount of delta and theta activity in the brain frontal region and negatively correlated with the amount of alpha activity in the frontal region. Results of this study (disturbances of memory and attention, QEEG changes similar to those in patients with Al encephalopathy) were interpreted as consistent with known CNS effects of Al, but insufficient for establishing a definite relationship between Al exposure and effects.

In another study of Al welders, CNS evaluations were performed on 38 men who had at least 5 years exposure (mean 17.1 years) and a control group of 44 railway track welders exposed to metal fumes other than Al (mean 13.8 years) (Sjogren et al., 1996). Limited monitoring data indicated that the median exposure to welding fumes was 10 mg/m³ and that the Al content was 40% of the total fumes. Symptom questionnaires, psychological tests (simple reaction time, finger tapping speed and endurance, digit span, vocabulary, tracking, symbol digit coding, cylinders, olfactory threshold and Luria-Nebraska motor scale), neurophysiological indices [electroencephalography, P-300 auditory-evoked responses, brain-stem auditory evoked responses and diadochokinesis (ability to perform rapidly alternating movements with one limb)] and blood and urine Al levels were assessed. The blood and urine Al concentrations were approximately 3 and 7 times higher in the Al welders than in the controls, but there were no clear correlations between duration of exposure to Al and concentration of Al in blood or urine. The Al welders reported more acute CNS symptoms (e.g., concentration difficulties) and had decreased motor function in five tests (finger tapping in non-dominant hand, two tasks from the Luria-Nebraska motor scale, pegboard peg movement with dominant hand, amplitude of diadochokinesis in dominant hand) when compared to the control group. Urinary Al concentration was significantly correlated with acute CNS symptoms, but not with any of the performance measures. To further study possible dose-effect relationships of Al exposure, the Al welders were combined with the control group and divided into three exposure categories according to urinary Al levels, using the 50th and 75th percentiles as category dividers. The group with the highest mean urinary Al level had significantly more acute CNS symptoms and significantly reduced performance on one of the motor function tests (a Luria-Nebraska motor

scale task) when compared to the group with the lowest Al level. In an earlier study of 65 welders with ≥ 10 years of exposure to Al fumes, the highest exposure category (based on exposure duration) was 2.8 times more likely than unexposed workers to have three or more neuropsychiatric symptoms (Sjogren et al., 1990).

A body of epidemiological evidence has pointed to an increased incidence of cancers of various kinds in workers employed in the aluminum production industry. However, as discussed in a review by Ronneberg and Langmark (1992), the concern about potential cancer hazards in the aluminum industry has primarily arisen because of exposures to polycyclic aromatic hydrocarbons (PAHs) and coal tar pitch volatiles (CTPVs) rather than to Al *per se*. Thus, while a number of studies have provided inferential data linking occupationally exposed aluminum workers with an increased risk of developing tumors of the bladder or lung (Gibbs, 1985; Thériault et al., 1984, 1990; Armstrong et al., 1986; Spinelli et al., 1991; Pearson et al., 1993; Tremblay et al., 1995), it would be unwise to ascribe any excess tumor formation to the effects of Al in view of the concurrent exposure to well-documented carcinogenic PAHs such as benzo(a)pyrene. The issue is further complicated by the likely exposure of production workers to other substances such as fluorides, sulfur dioxide, aromatic amines and asbestos (Ronneberg and Langmark, 1992; Tremblay et al., 1995; Dufresne et al., 1996), and to the possible effects of cigarette smoking in affected individuals. Consequently, these studies have failed to provide direct evidence for the carcinogenicity of Al fumes and dusts.

Animal Studies

Oral Exposure

Numerous subchronic animal studies were located in the biomedical/toxicological literature but only those that define the threshold region of the oral dose-response relationship are summarized in this paper. A major limitation of many of the studies of Al toxicity is the lack of complete information on total dietary (e.g., food and drinking water) intake of Al and of other elements that are known to effect Al biokinetics and toxicity (e.g., calcium and magnesium). Estimated or reported dosages used in studies in which Al content of the basal diets are not reported must be assumed to underestimate the actual experimental dosages. The magnitude of the underestimate may be considerable. For example, a range of Al contents of 200-1200 mg Al/kg for commercial grain-based diets (Golub et al., 1992b) would provide 30-200 mg Al/kg bw-day in a subchronic or chronic mouse bioassay [based on U.S. EPA (1988) default values for body weight and food intake]. On this basis, studies in which complete dietary Al intakes were not reported or could not be estimated may provide some information about the hazards of oral exposure to Al but are inappropriate for establishing NOAELs or LOAELs for the critical effect of Al. NOAELs and LOAELs from studies that provide estimates of total Al dosages, or otherwise provide information relevant to determining the NOAEL/LOAEL boundary for the critical effect of Al are presented in Table 1 and are summarized below.

Systemic toxicity

Groups of 10 female Sprague-Dawley rats were administered aluminum nitrate nonahydrate in sugar-containing drinking water at doses of 360, 720 and 3600 mg/kg-day (26, 52

and 259 mg Al/kg bw-day, respectively) for 100 days (Domingo et al., 1987). A control group received sugar-containing distilled water only. Sugar had been added to the drinking water of all groups to reduce the taste-aversive effects of Al. The level of Al in the diet was not reported. Animals were housed in metabolic cages to facilitate the collection of fecal and urine samples. Food and water consumption were measured daily, body weights were noted weekly and blood samples were taken at monthly intervals and at termination to monitor clinical chemistry and hematological parameters. At termination, all animals were necropsied, and the weights of major organs (brain, heart, lungs, kidneys, liver and spleen) were monitored. Aluminum concentrations were measured in various tissues, pieces of which were processed for histopathological examination. A significant decrease ($p < 0.05$) in body weight gain was observed in the 259 mg Al/kg-day group, attributed by the authors to decreased food intake. Overall, no consistent variations in hematological (hemoglobin, hematocrit) or clinical chemistry (SGOT, SGPT, alkaline phosphatase, urea, creatinine, total protein, cholesterol, glucose) parameters were observed. No histopathological alterations in the heart, liver, kidney, spleen, brain and cerebellum were observed. Interpretation of these data was complicated by the concurrent exposure of the rats to high doses of nitrate of up to 475 times the RfD for nitrate (1.6 mg nitrate-nitrogen/kg-day) which is based on methemoglobinemia in humans (U.S. EPA, 1999). Therefore, because of nitrate co-exposure, the absence from the study design of a food-restricted control group and uncertainty surrounding the contribution of Al in food, the apparent effect of Al on body weight gain cannot be conclusively attributed to Al alone.

Some recent studies have identified a number of potential toxicological responses in laboratory animals exposed orally to Al compounds in a subchronic or chronic dosing regimen. In most cases, however, only one dose level was employed in the study compared to controls, and since the amount of Al in the diet was not given, the resulting dose level represents an incremental dose of Al compared to that of controls as baseline. However, while these studies may offer inadequate quantitative dosimetric information for NOAEL/LOAEL identification and consequent RfD development, they provide a qualitative indication of a range of potential toxicological responses that might be induced in humans exposed to the element. For example, Garbossa et al. (1998) studied the potential for water-soluble Al to affect the erythropoietic integrity of late erythroid progenitor cells in the bone marrow. Three groups of five male Wistar rats/group were either (1) gavaged with citrate at a dose of 1.0 μmol Al/g-day (27 mg/kg-day), 5 days/week, for 15 weeks, (2) had drinking water containing 100 mmol Al/L made available to them as the citrate for the same length of time or (3) maintained as controls. As calculated by the authors, the dose associated with the applied concentration of Al in drinking water approximated to 14-17 μmol /g-day (420 mg/kg-day). Rats had access to a standard chow diet, though with no indication of the baseline concentration of Al provided therein. At the end of the in-life phase of the study, all rats were sacrificed, and samples of blood were obtained for hematological investigation. Femoral bone marrow cells were flushed with physiological medium, stimulated with recombinant human erythropoietin, then monitored for the comparative incidence of colony-forming units-erythroid (CFU-E). Further tests were carried out to monitor the osmotic fragility and average life-span of erythrocytes from each test group. The animals in the group receiving Al at the higher dose showed decreased hematocrit, hemoglobin concentration, median osmotic fragility and erythrocyte life-span values compared to controls. The content of Al increased in the serum and bone of both exposed groups, the distribution of concentrations in bone correlating inversely with the extent of an animal's CFU-E development.

That Al in drinking water may have the ability to cause histopathological changes and altered hepatic enzyme activities was suggested by Basu et al. (1997) who made available aluminum chloride in drinking water to groups of eight male Sprague-Dawley rats at a dose of 50 mg/kg-day (10.1 mg Al/kg-day) for 40 days. Additionally, other groups of similarly-treated rats received drinking water containing either 0, 50, 100, 200 or 400 ppm (mg/L) added calcium (Ca), as the chloride. The authors reported increased specific activities of acid and alkaline phosphatases in liver 10,000 x g supernatants from Al-receiving animals versus controls, and in alkaline phosphatase activity in equivalent kidney preparations. The presence of Ca in the drinking water appeared to reverse these changes, plus the accompanying histopathological features associated with them.

Konishi et al. (1996) examined the ability of Al and Ca to cause opposite and potentially harmful effects in laboratory animals, in relation to the well-documented association between Al and the onset of osteomalacia. Male STD Wistar rats were divided into four groups (n=4), receiving either (1) a normal diet (Group I), (2) a normal diet supplemented with Al (Group II), (3) a Ca-deficient diet (Group III) or (4) a Ca-deficient diet with supplemental Al (Group IV), for 10 weeks. Blood samples were taken at termination, and then animals were perfused with paraformaldehyde/glutaraldehyde fixative. Levels of Ca, iron (Fe) and Al in serum and bone were measured by atomic absorption spectrophotometry, and sections of the resected right tibia were prepared for histopathological examination after decalcification in 5% formic acid in 10% formalin.

There were statistically-significant changes in body weight gain when those of groups 3 and 4 were compared to animals from groups 1 and 2, the values for the latter groups remaining constant from about 4 weeks of dosing. In discussing their histopathological findings, the authors described no decrease in the thickness of cortical bone in Group II compared to control, while bone specimen from Groups III and IV showed “an increase in osteoid as well as osteoblasts and osteoclasts”, in addition to other disturbances of ossification. Such effects were considered to suggest bone fragility, with changes being more marked in Group IV compared to III. The amount of Al in the tibia of exposed rats was significantly greater in Group II than in Group I, whereas the average levels in Groups III and IV showed a further increase in Al deposition, most notably in group IV. There were also differences among the groups in the concentration of Fe in bone (tibia), and in the concentrations of Al, Ca, Fe and the levels of parathyroid hormone in blood. The authors concluded that Ca deficiency appeared to potentiate the deposition of orally administered Al in bone, and the attendant inhibition of ossification. Iron deposition was also thought to play a role in the osteogenic disturbance, where Ca is deficient.

A histopathological investigation indicated profound changes in the cerebrovascular and neuronal integrity when male Long-Evans rats (n=9) were exposed for 52 weeks to 0.5 ppm aluminum fluoride in drinking water (Varner et al., 1998). This corresponded to an Al dose of 0.019 mg/kg-day, based on a default drinking water consumption of 0.057 L/day, and a default body weight of 0.472 kg for male Long-Evans rats (U.S. EPA, 1988). Dual control groups received either NaF (fluoride controls) or double distilled deionized water. Tissue levels of Al were measured in brain, liver and kidney by the use of a direct current plasma technique.

Animals receiving aluminum fluoride showed poor survival compared to the other groups, with 6/9 having died by week 48. The tissue concentrations of Al were increased in the brain and kidney compared to both the control groups, with Al-fluorescence being used to demonstrate that Al deposition was mostly in the vasculature. Morphological and histopathological changes due to treatment were apparent in the liver, kidney and spleen. Some changes in neuronal integrity were also evident in the hippocampus and neocortex. Other cytological changes in the brain were associated with chromatid clumping, pyknosis and vacuolation.

A report by Somova et al. (1997) describes a study in which 10 male Wistar rats/group received either 0, 5 or 20 mg/kg-day aluminum chloride by gavage in water for 6 months. At termination, all animals were exsanguinated, then subjected to a necropsy in which excised pieces of liver, kidney and cardiac and skeletal muscle were taken for histopathological examination. Pieces of brain were examined by electron as well as light microscopy, and all tissues were monitored for Al concentration by atomic absorption spectrophotometry. As tabulated by the authors, Al in plasma and all of the listed tissues was dose-dependently increased to levels that were statistically significantly greater than controls. However, though described in qualitative terms and illustrated photographically, the Al-induced lesions did not receive a quantitative treatment in the report. Thus, while at least some of the low dose rats displayed NFD (neuro fibrillar degeneration) of the hippocampal region of the brain, insufficient data are provided in the report to apply this observation to the identification of a NOAEL or LOAEL.

Dietary experiments

Six Beagle dogs/sex/group were fed a diet providing either, in males, 0, 118, 317 or 1034 mg/kg-day sodium aluminum phosphate (0, 3.4, 9.0 or 29.4 mg Al/kg-day, respectively) or, in females, 0, 112, 361 or 1087 mg/kg-day sodium aluminum phosphate (0, 3.2, 10.3 or 30.9 mg Al/kg bw-day, respectively), for 6 months (Katz et al., 1984). No information was available on the level of Al in the diet, and no compound-related effects on body weight gain, hematological and clinical chemistry parameters (parameters not specified) or histopathological endpoints (major organs and tissues examined) were observed. A highest NOEL of 30.9 mg Al/kg-day could be tentatively identified in this study, but this would not include the contribution of Al from the basal diet, nor reflect the identification of any toxicological effects, since the NOEL occurred at the upper limit of the dose-response curve.

Neurotoxicity

A number of studies have been reported in which neurotoxicological/neurobehavioral effects have been explicitly evaluated. In others, the effects of Al on neurological developmental have been addressed. For example, Golub et al. (1989) fed diets containing Al as the lactate at 25 (controls), 500 or 1000 mg Al/kg diet (3.3, 65 or 130 mg Al/kg-day) to groups of 15 female Swiss-Webster mice for 6 weeks (Golub et al., 1989). No mice were exposed to lactate alone. While no statistically significant differences in food intake or body weight gain were observed, mice fed the highest Al concentration gained less weight than the controls or low-dose group. As reported by the authors, a significant decrease (20%) in spontaneous motor activity (i.e., total, vertical and horizontal movement) was observed in the 130 mg Al/kg-day group. Activity in the

65 mg Al/kg-day group was not significantly different than the controls. Thus, the highest NOAEL is 65 mg Al/kg-day and the LOAEL is 130 mg Al/kg-day.

Neurobehavioral effects of aluminum lactate were evaluated in groups of 12 female N:NIH Swiss-Webster mice (4.5-5.5 weeks old) that were fed 25 (controls) or 1000 mg Al/g diet for 90 days (Golub et al., 1992a). Based on a food factor of 0.19 kg diet/kg body weight/day calculated using an algorithm relating food consumption to body weight (U.S. EPA, 1988) and reported body weight data (the time-weighted average weight is 25.4 g), the dosage in the treated mice is estimated to be 190 mg Al/kg bw-day. No mice were exposed to lactate alone. A neurobehavioral test battery used by Donald et al. (1989) was administered at the beginning of the experiment (day 0) and after 45 and 90 (± 3) days, with motor activity evaluated at the latter two time points. Aluminum levels were measured in brain, femur and liver at the end of the exposure period.

Body weight was significantly increased in the treated mice but no exposure-related changes in food intake or overt signs of neurotoxicity were observed. Results of the neurobehavioral tests showed significantly decreased hindlimb grip strength at 90 days, decreased air puff startle response at 90 days and decreased auditory startle response at 45 days in the treated mice. Spontaneous motor activity was reduced at 90 days as indicated by decreased total activity counts, horizontal activity counts and percentage of intervals with high activity counts. Aluminum concentrations in the brain and liver were increased approximately 3-fold in the treated mice, but brain and liver lipid peroxidation indices were not altered.

Male Wistar rats (6-8 per group) were exposed continuously for 6 months to food containing 1.52 mg Al/kg (normal diet) or 1000 mg Al/kg as aluminum chloride with citrate (Florence et al., 1994). The average daily Al intake was estimated to be 0.13 or 84 mg Al/kg bw-day, assuming a body weight of 0.305 kg (arithmetic mean of default mature weight of male Wistar rats and the starting weight in this study of 0.11 kg) and a food intake of 0.026 kg food/kg bw-day, calculated using an algorithm relating food intake to body weight (U.S. EPA, 1988). The citrate content of the diet was in a 1:1 stoichiometric proportion to Al, therefore, the estimated daily intake was 598 mg/kg-day. Rats exposed to Al developed histopathological abnormalities in brain tissue, not specific to any brain region, characterized by extensive cytoplasmic vacuolization in astrocytes, swelling of astrocytic processes, particularly of astrocyte end-feet abutting blood vessels. Neurons also exhibited vacuolization and nuclear inclusions. Although no specific behavioral assays were reported, the investigators noted that "no significant behavioral changes were observed". Accordingly, the functional significance of the histopathological lesions is uncertain. The lesions appear to differ from the NFD observed with parenteral Al exposures (Kowall et al., 1989; Wakayama et al., 1993); or from exposures to Al in combination with calcium deprivation (Garruto et al., 1989; Kihira et al., 1995; Mitani, 1992). The LOAEL for histopathological changes in the brain was 84 mg Al/kg-day.

Male Sprague-Dawley rats (40 per group) were exposed in drinking water to 0, 50 or 100 mg Al/kg bw-day as aluminum nitrate with citric acid for 6.5 months beginning at 21 days of age, 8 months of age or 16 months of age (Domingo et al., 1996). The citric acid dosage was 355 or 710 mg/kg-day in the 50 or 100 mg Al/kg bw-day groups, respectively. Controls did not receive citric acid. Dietary Al intake was not reported; the rats were maintained on Panlab rat

chow. Animals from control and exposed groups were subjected to a number of neurobehavioral tests, and at termination, Al levels were measured in various excised regions of the brain. The authors observed the highest Al levels in the olfactory bulb and rhachidical bulb, while the cortex and thalamus were the regions showing the lowest Al content. However, compared to controls, there were no significant effects ($p > 0.05$) of Al (with citric acid) on spontaneous motor activity (open-field) or passive avoidance operant training or performance (grid floor shock, light/dark shuttle box). Thus, the NOAEL was 100 mg Al/kg-day with citric acid; although this does not include the Al contribution from food. This study is listed on Table 1 because the NOAEL, although probably underestimated because of unreported Al intake from food, is still lower than the LOAELs from other studies.

Groups of six male albino rats were administered 0 or 25 mg Al/kg bw-day as aluminum nitrate in normal saline by gavage, 10% ethanol in drinking water, or 25 mg Al/kg bw-day by gavage combined with 10% ethanol in drinking water, 6 days/week for 6 weeks (Flora et al., 1991). The level of Al in the diet was not reported. Urinary Δ -aminolevulinic acid (ALA), blood ALA-dehydratase (ALAD), blood zinc protoporphyrin (ZPP), glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) in serum and liver and brain biogenic amines and their metabolites [dopamine (DA), norepinephrine (NE), 5-hydroxytryptamine (5-HT), homovanillic acid (HVA) and 5-hydroxyindolacetic acid (5-HIAA)] were evaluated at the end of the treatment period. Treatment with Al alone caused significantly increased blood ALAD ($p < 0.01$), decreased liver GPT ($p < 0.05$), decreased brain DA ($p < 0.01$), increased brain NE ($p < 0.05$) and decreased brain 5-HT ($p < 0.05$). Compared to treatment with Al alone, concurrent exposure to ethanol and Al produced significantly decreased ALAD, increased ALA, increased ZPP, increased liver GPT, increased serum GOT and increased brain HVA. Significant changes found only in the combined Al and ethanol group included increased serum GPT, increased brain NE and decreased brain 5-HT. Treatment with ethanol alone only inhibited blood ALAD. The rats were co-exposed to relatively high levels of nitrate [comparable to those in the Domingo et al. (1987) subchronic study], but it seems likely that some of the changes (i.e., effects on brain chemicals) are related to aluminum which is known to be neurotoxic. Because the toxicological significance of the changes is unclear due to lack of evaluation of neurobehavioral performance and other endpoints, there is uncertainty whether the 25 mg Al/kg-day dose is a NOAEL or a LOAEL, an uncertainty compounded by the absence of information about the level of Al in the basal diet.

Reproductive/developmental toxicity

A number of studies have been carried out to examine the effects of Al compounds on developmental toxicity, particularly their effects on postnatal neurobehavioral development. For example, Bernuzzi et al. (1989) exposed groups of 6-12 pregnant Wistar rats to aluminum chloride or aluminum lactate in the diet on gestational days 1 through 21. The rats received nominal daily doses of 0, 100, 300, 400 mg Al/kg as aluminum chloride or 0, 100, 200 or 400 mg Al/kg as aluminum lactate. No rats were exposed to lactate alone, and information regarding level of Al in the basal diet was not reported. On the average, there was a less than 10% decrease in maternal body weight gain and no effect on food or water intake. No significant difference in litter size was observed. However, postnatal mortality increased 55% and 26% in offspring of the rats exposed to 300 or 400 mg Al/kg-day, respectively. The offspring of dams

fed ≥ 300 mg Al/kg-day weighed significantly less than controls on postnatal day 1. Decreased body weight was also observed on postnatal days (PD) 4 and 14 in the offspring of rats fed 400 mg Al/kg-day as aluminum lactate. The following tests were used to assess neuromotor development (maturation): righting reflex, grasping reflex, negative geotaxis, suspension test and locomotor coordination. The tests were performed on PDs 4, 6, 9, 12 and 20, respectively. Impairment of neuromotor development (righting and grasping reflexes) was observed in the pups exposed to ≥ 200 mg Al/kg-day. Impaired grasping reflex was also observed in the 100 mg/kg-day aluminum lactate group. Offspring of rats fed 400 mg/kg-day also exhibited altered performance on the locomotor coordination test.

A follow-up study by the same research group found that ingestion of 400 mg Al/kg bw-day as aluminum lactate had no effect on postnatal mortality, body weight and righting and grasping reflex tests (Muller et al., 1990), although significant differences between control and exposure groups were noted in locomotor coordination and operant conditioning tests. Significant differences between controls and exposed groups in the negative geotaxis test were limited to those pups of dams treated during the second and third weeks of gestation, a finding interpreted by the authors to indicate the possibility of long-term effects on the central nervous system of trans-placenta exposure to Al during a later organogenic phase. According to Muller et al. (1990), the contradictions between this and their earlier study (Bernuzzi et al., 1989) could be related to environmental modifications. In particular, the mothers and pups were much more protected in the Muller et al. (1990) study than in the previous one because they were housed in plastic cages instead of wire mesh cages and received cotton to build nests. Body temperature of the pups, therefore, may have been more adequately maintained in the Muller et al. (1990) study. As discussed in this study, toxicity in pups can be confounded by insufficient body temperature, and delayed pup weight gain could explain the differences in neuromotor performance.

Muller et al. (1990) administered diets supplemented with 0 or 400 mg Al/kg bw-day as aluminum lactate to groups of 6-9 pregnant Wistar rats on days 1-7, 1-14 or 1-21 of gestation. No rats were exposed to lactate alone, and information regarding level of Al in the basal diet was not reported. Neuromotor development was assessed on postnatal days 4, 6, 9, 12 and 20 using tests of righting reflex, grasping reflex, negative geotaxis, suspension and locomotor coordination, respectively. Learning ability was also tested on PD 65 using operant conditioning. No effects on maternal body weight or food intake were observed in dams exposed on gestational days 1-7 or 1-14. In the dams exposed on gestational days (GD) 1-21, a significant decrease in maternal body weight (26 and 35%, respectively) was observed on days 16 and 19 of gestation. Decreased food intake was also observed on day 19 of gestation. No effects on litter size, postnatal mortality or postnatal body weight were observed. Impairment of neuromotor development ($p < 0.05$) was observed in two of the five tests (negative geotaxis and locomotor coordination); no differences between the three treated groups were observed. For the operant conditioning test, there were significant differences ($p < 0.05$) between the treated and control young rats. No differences between the three treated groups were observed. The LOAEL for developmental toxicity is 400 mg Al/kg-day, but this does not include the contribution of Al from the basal diet.

Groups of 10 pregnant Sprague Dawley rats were administered 180, 360 or 720 mg/kg-day aluminum nitrate nonahydrate by gavage (13, 26, 52 mg Al/kg bw-day) on GDs 6-14

(Paternain et al., 1988). A vehicle (water) only control group was used. The level of Al in the diet was not reported. Aluminum exposed dams gained significantly less weight than the controls. No significant effects on the numbers of litters, corpora lutea, total implants, live fetuses, resorptions or runt fetuses were observed. Significant decreases in fetal body weight and tail length were observed at all three Al doses; decreased fetal body length was also observed at the 52 mg Al/kg-day dose level. No dose-related external or visceral malformations were observed in the offspring. However, a significant increase in the incidence of skeletal malformations (delayed ossification, hypoplastic deformed ribs) was observed at all three treatment levels. In addition, the incidence of hematomas was significantly increased at the high dose. Because the rats were co-exposed to relatively high levels of nitrate [comparable to those in the Domingo et al. (1987) subchronic study], the effects of treatment cannot be conclusively attributed to Al alone, in the absence of a nitrate-exposed control group.

By contrast to the striking findings of potentially teratogenic effects of aluminum nitrate in Sprague-Dawley rats, as described above (Paternain et al., 1988), equivalent experiments by Domingo et al. (1989) in Swiss mice did not reveal any reproductive, developmental or teratogenic effects of Al, when administered to dams as the hydroxide. Domingo et al. (1989) administered by gavage 0, 66.5, 133 or 266 mg/kg-day aluminum hydroxide (0, 23.9, 47.8 or 95.5 mg Al/kg bw-day) to groups of 20 pregnant Swiss mice on GD 6-15. The level of Al in the diet was not reported. The dams were killed on GD 18. No compound-related effects were observed on maternal mortality, clinical signs, body weight, food intake or absolute or relative heart, lung, spleen, liver, kidney and brain weights. In addition, no compound-related effects were observed on numbers of implantations, resorptions, live and dead fetuses, sex ratio and the incidences of external malformations, internal soft-tissue defects or skeletal abnormalities. Therefore, this study identifies a NOEL of 95.5 mg Al/kg-day by default for reproductive, developmental and teratogenic toxicity in mice. However, neuromotor development was not assessed and the contribution of Al from the basal diet was not stated in the report.

A number of studies have been designed to evaluate the influence of citrate or lactate on the potential developmental toxicity of Al. For example, Gomez et al. (1991) exposed groups of 15-19 pregnant Sprague-Dawley rats to either distilled water (controls) or 133 mg Al/kg bw-day in the form of either aluminum hydroxide (384 mg/kg-day), aluminum citrate (1064 mg/kg-day) or aluminum hydroxide (384 mg/kg-day) concurrent with citric acid (62 mg/kg-day) by gavage on GD 6-15. The level of Al in the diet was not reported and no rats were exposed to citric acid alone. Terminations were performed on GD 20. Maternal and fetal evaluations showed exposure-related effects only in the group exposed to aluminum hydroxide and citric acid concurrently. Significant changes included reduced maternal body weight gain on GDs 6-20 (but not at sacrifice on day 20), reduced fetal body weight and some skeletal variations (increased delayed occipital and sternbrae ossification and increased absence of xiphoides). No effects were seen on maternal food consumption or clinical signs, maternal absolute or relative liver, kidney or brain weights, gravid uterine weight, corpora lutea/dam, implantations/litter, pre- or postimplantation loss/litter, viable or nonviable implants/litter, fetal sex ratio or fetal malformations (external, visceral or skeletal). This study identified a stand alone minimum LOAEL of 133 mg Al/kg-day for non-neurobehavioral developmental toxicity of aluminum hydroxide and aluminum citrate in rats. Although confidence in this LOAEL is low (because aluminum hydroxide administered concurrently with citric acid induced did developmental

effects and because the dose does not include a contribution of Al from the basal diet) the value is consistent with the developmental NOAEL of 95.5 mg Al/kg-day for aluminum hydroxide in mice (Domingo et al., 1989).

In a similar experimental protocol, groups of 11-13 pregnant female Swiss albino (CD-1) mice were administered 57.5 mg Al/kg bw-day as either aluminum hydroxide (166 mg/kg-day), aluminum lactate (627 mg/kg-day) or aluminum hydroxide (166 mg/kg-day) concurrent with lactic acid (570 mg/kg-day) by gavage on gestation days 6-15 (Colomina et al., 1992). Other groups were treated with lactic acid alone (570 mg/kg-day, equivalent to the amount in 627 mg/kg of aluminum lactate) or distilled water (controls). The level of Al in the diet was not reported. Fetal evaluations were performed on GD 18, including examinations for skeletal and visceral abnormalities in approximately two-thirds and one-third of the pups, respectively. The investigators noted that the dose of Al (57.5 mg/kg-day) is equivalent to ingestion of 3.5 g Al/day by a 60 kg person, which is higher than the usual quantities of Al ingested therapeutically for peptic disorders. Maternal body weight gain was significantly lower than control values in the aluminum lactate-treated mice when evaluated over GDs 6-9 (92%), 6-12 (55.6%) and 0-18 (38.5%) and in the mice treated with combined aluminum hydroxide and lactic acid evaluated over GDs 6-12 (37.8%), 6-15 (42.7%) and 0-18 (15.7%). The decreased maternal weight gain in the aluminum lactate group was accompanied by significantly reduced food consumption during gestation days 6-18. Significant developmental and/or teratological effects in the aluminum lactate group included 16% reduced fetal body weight ($p < 0.01$) and increased incidences of cleft palate (13.2%, $p < 0.05$), dorsal hyperkyphosis (i.e., excessive flexion of spine) (13.5%, $p < 0.05$) and delayed parietal ossification (15.4%, $p < 0.01$). These developmental effects were not observed in any of the control or aluminum hydroxide exposed pups, and the only other significant changes in the other groups were decreased maternal relative liver weight and delayed fetal parietal ossification in the lactic acid only exposure group. Other types of internal or skeletal malformations or variations were not found in any of the fetuses. Additionally, no effects were seen on maternal absolute or relative kidney weight, gravid uterine weight, numbers of implantation sites/litter, live or dead fetuses, resorptions, postimplantation loss/litter, litters with dead fetuses or fetal sex ratio in any of the groups. By analogy to the findings of the Domingo et al. (1989) and Gomez et al. (1991) studies, the lack of developmental effects of aluminum hydroxide at the tested dose could be related to low solubility and absorption.

In a more recent study, pregnant Swiss mice were administered gavage doses of 0 or 104 mg Al/kg bw-day as aluminum hydroxide on days 6-15 of gestation (Colomina et al., 1994). Dietary Al intake was not reported; the mice were maintained on Panlab rodent chow. Compared to controls, there were no effects ($p > 0.05$) of Al on maternal body or organ weight, number of implantations per litter, number of resorptions per litter, number of dead fetuses per litter, percentage of positive post-implantation loss, sex ratio or fetal body weight per litter. Gross external, visceral or skeletal examination of fetuses revealed no abnormalities or developmental variations. Thus, the NOAEL for development effects from this study is 104 mg Al/kg-day, however, this does not include the Al contribution from food. Thus, based on this study and the previous study (Colomina et al., 1992), aluminum lactate appears to be more potent as a developmental toxicant in mice than the less water soluble aluminum hydroxide.

Groups of 16 pregnant Swiss-Webster mice were fed 25 (control group), 500 or 1000 mg Al/kg diet as aluminum lactate throughout gestation and lactation (Donald et al., 1989). The control diet was fed to pups that were selected for post-weaning neurobehavioral assessment. Reported maternal doses were 5, 100 and 200 mg Al/kg bw-day at the beginning of pregnancy and 10.5, 210 and 420 mg Al/kg bw-day near the end of lactation. No mice were exposed to lactate alone. There were no treatment-related changes in maternal survival, body weight (measured on GD 0 and 16 and PDs 0, 5, 10, 15 and 20), food intake, toxic signs or neurobehavior (evaluated after pups were weaned at PD 21 using the same test battery used for the pups and described below), or on litter size or postnatal growth and development in pups as assessed by body weight, toxic signs on PDs 0-55, and by crown-rump length on PDs 0 and 20. Neurobehavioral maturation was tested in two pups per litter on PDs 8-18 with a 12-item test battery (fore- and hindlimb grasp, fore- and hindpaw placement on sticks of 2 widths, vibrissa placing, visual placing, auditory and air puff startle, eye opening and screen grasp, cling and climb). A neurobehavioral test battery was administered to six pups per litter at age 25 days (4 days postweaning) or 39 days (fore- and hindlimb grip strengths, temperature sensitivity of tail, negative geotaxis, startle reflex to air puff and auditory stimuli) or age 21 and 35 days (foot splay). The pre-weaning neurobehavioral testing showed that a significant ($p=0.007$) number of pups in the high dose group had impaired vertical screen climb performance. The postweaning neurobehavioral assessment showed significantly ($p<0.05$) altered performance on several tests. These included decreased forelimb grip strength at age 39 days in the low dose group, increased hindlimb grip strength at age 25 days in both low and high dose groups, increased foot splay distance at age 21 days in both low and high dose groups and at age 35 days in the low dose group, and increased forelimb grip strength at age 25 days and decreased thermal sensitivity at age 25 and 39 days in the high dose group. There were no treatment-related changes in concentrations of Al in pup liver or bone (brain tissue was not analyzed).

In a more recent study of similar design by the same group of investigators, groups of 14 and 9 female Swiss Webster mice (6-8 weeks old) were fed 25 (control) or 1000 mg Al/g diet as aluminum lactate, respectively, during gestation and lactation (Golub et al., 1992b). The 1000 mg/g concentration was selected based on the demonstration of neurobehavioral effects in weanlings at this level (Donald et al., 1989). No mice were exposed to lactate alone. Using food intake and body weight values estimated from reported data, maternal doses are estimated to be approximately 4.3 and 174 mg Al/kg bw-day at the beginning of gestation and 4.8 and 607 at the end of the lactation period. At birth, litters were fostered either within or between groups to provide four groups of offspring that were exposed to excess Al via maternal diet during gestation, lactation, both or neither (i.e., 25 ppm during gestation and lactation, 1000 ppm during gestation and 25 ppm during lactation, 25 ppm during gestation and 1000 ppm during lactation, and 1000 ppm during gestation and lactation). Maternal effects included significantly ($p\leq 0.015$) reduced (10-12%) body weight gain and food intake in the treated group during late pregnancy and lactation, and signs of neurotoxicity (hindlimb splaying and dragging) in one treated dam at postnatal day 21 (weaning); this dam had seizures and died 4 days later. No treatment-related effects on litter size, birth weight, crown-rump length, righting ability at birth, sex ratio or postnatal survival were observed. Both gestation-only and lactation-only exposure caused significantly ($p<0.05$) decreased body weight gain in the treated pups beginning on postnatal day 10; combined gestation and lactation exposure produced the greatest decrease (approximately 24% at weaning). Neurobehavioral testing using the same battery as Donald et al. (1989) was

performed at weaning on the dams and on a total of 12, 16, 12 and 6 pups (1 male and 1 female pup per litter) from the control, gestation-only, lactation-only and combined gestation and lactation groups, respectively. Results of this testing showed effects only in pups, including significantly decreased forelimb grip strength after gestation-only exposure, increased hindlimb grip strength after both gestation and lactation exposure, decreased temperature sensitivity after lactation-only exposure, and longer negative geotaxis latency after lactation-only exposure. In general, the findings of this study are consistent with those of Donald et al. (1989) in showing neurodevelopmental effects at the 1000 mg/kg dietary concentration, although intake dosages are dissimilar at the end of lactation. Using the dosage at the beginning of gestation, this study defines a LOAEL of 174 mg/kg-day for developmental effects.

The Donald et al. (1989) study differs from that of Golub et al. (1992b) in that offspring were not fostered, were tested at a later age (25 vs. 21 days), were allowed 4 days of recovery from the treated diet prior to testing, participated in other behavioral tests currently, and experienced no growth retardation. The effects found only in the cross-fostered groups in the Golub et al. (1992b) study (lower forelimb strength after gestation exposure and altered negative geotaxis latencies after lactation only exposure) were not observed by Donald et al. (1989). Increased footsplay was observed by Donald et al. (1989) but not by Golub et al. (1992b), perhaps due to an opposing effect of smaller pup body size in this study. Neither gestation or lactation exposure affected pup brain or liver Al concentrations, but lactation exposure caused significantly lower manganese and iron concentrations in liver and manganese concentrations in brain.

In a further extension of the two previous studies (Donald et al., 1989; Golub et al., 1992b), pregnant female Swiss-Webster mice were exposed continuously to a semi-purified diet containing 7 (control), 500 or 1000 mg Al/kg from the time of conception, through pregnancy and lactation (Golub et al., 1995). At weaning, pups were exposed to the same Al diet as their mothers (500 or 1000 mg Al/kg) until they were 150-170 days of age or were switched to the control diet (7 mg Al/kg) for the same time period. Based on reported dosages in previous studies by the same investigators, estimated daily dosages for mice exposed to 1000 mg Al/kg diet were as follows: 200 mg/kg bw-day in pregnant mice, 420 mg/kg-day in lactating mice and 130 mg/kg-day in offspring (Golub et al., 1994); doses for the mice exposed to 500 mg Al/kg diet were assumed to be approximately half of that of mice fed 1000 mg Al/kg, or 100 mg/kg-day in pregnant mice, 210 mg/kg-day in lactating mice and 65 mg/kg-day in offspring. Compared to the control diet, the Al diet had no effect on dam weight, gestation length, litter size, pup weight, offspring growth or organ weights. Operant conditioning (nose poke) of offspring for delayed spatial alternation or discrimination reversal tasks was initiated at 50 days of age and continued 5 days/week for a total of 35 sessions. A neurobehavioral test battery was conducted when the offspring were 150-170 days of age (forelimb and hindlimb grip strength, temperature sensitivity, negative geotaxis, air puff and auditory startle response). Maternal and pre-weaning exposure to 500 mg Al/kg significantly affected ($p < 0.05$) operant training in the offspring, but not performance after training in delayed spatial alternation or discrimination reversal tasks (i.e., decreased number of training sessions to achieve the training criteria). This exposure also significantly decreased forelimb and hindlimb grip strength and puff startle response ($p < 0.05$). Pre-weaning and combined pre- and post-weaning exposure to 1000 mg Al/kg significantly increased ($p < 0.05$) incidence of cagemate aggression at the time behavioral

testing. No effects were observed on auditory startle response, temperature sensitivity or negative geotaxis in offspring. Histopathological examination of the brain and spinal cord revealed no treatment-related changes. Thus, the LOAEL for combined maternal and pre-weaning exposure on neurobehavioral effects in mice would approximate to 100 mg Al/kg-day (estimated daily maternal dosage).

Pregnant Charles River CD rats were administered gavage doses of 0, 250, 500 or 1000 mg Al/kg bw-day ("experiment A") or 0, 5, 25, 50, 250 or 500 mg Al/kg bw-day ("experiment B") as aluminum lactate in distilled water on GDs 5-15 (Agarwal et al., 1996). Dietary Al intake was not reported. Offspring were examined for body weight, anogenital distance, oestrus cycle regularity (after puberty), duration of pseudopregnancy induced by mechanical stimulation of the cervix, oocyte production induced by an injection of human chorionic gonadotropin, and male and female gonad weights. Aluminum had no effect on litter size and no consistent effects on birth weight were observed. For example, birth weights were decreased in male offspring from dams that received 250 mg Al/kg-day, but not at higher dosages, and the effect was observed only in experiment A. Female offspring birth weights decreased at certain dosage levels in experiment A and increased at these same dosage levels in experiment B. Similar inconsistencies between experiment A and B were observed for gonadal weights, anogenital distance, time to puberty (vaginal opening), duration of pseudopregnancy or numbers of superovulated oocytes. A significantly increased ($p < 0.05$) number of abnormal oestrus cycle lengths (defined as less than 4 days or greater than 5 days) occurred in offspring from dams that received 250 mg Al/kg-day (in experiment A, the endpoint was not measured in experiment B). However, the effect was most pronounced in the first three oestrus cycles (of five observed) and not detected by the 5th cycle. Thus, the NOAEL for temporary disturbance of the oestrus cycle in offspring of dams administered Al is 250 mg Al/kg-day. NOAELs for all other reproductive endpoints in this study were 1000 mg Al/kg-day. These NOAELs do not include the contribution of Al in food.

In a three-generation study, Ondreicka et al. (1966) exposed initial groups of seven female and three male Dobra Voda mice to either 0 or 19.3 mg Al/kg bw-day as aluminum chloride in drinking water. The diet also contained 160 to 180 ppm Al, giving an estimated intake of 27-31 mg/kg-day based on default values for food consumption and body weight for chronic exposure of mice (U.S. EPA, 1988). Using this estimate, the total Al intakes (drinking water and food) were 27 mg/kg-day (controls) and 46.3 mg/kg-day (exposed group). The P_0 group produced three litters (designated F_{1a} , F_{1b} and F_{1c}) and the F_{1a} group produced two litters (designated F_{2a} and F_{2b}) from which the weanlings were exposed to Al in the drinking water starting at 4 weeks of age. There was no difference in body weight gain among the groups in the P_0 generation, a result that contrasted with the striking decrease in this parameter in the treated F_{1b} , F_{1c} , F_{2a} and F_{2b} groups. Though no effects on erythrocyte count, hemoglobin levels or histopathology of the liver, spleen and kidneys were observed in the P_0 , F_1 or F_2 generations at the end of the study and no significant differences were seen in the number of litters or offspring between the exposed and control groups, the study identified a LOAEL of 46.3 mg Al/kg-day, based on the observed changes in body weight gain.

Other toxicological effects of aluminum

In a study designed to determine the effects of oral Al exposure on susceptibility to bacterial infection, female Swiss-Webster mice (13-14 per group) were exposed to a diet containing 25 (control), 500 or 1000 mg Al/kg as aluminum lactate during pregnancy, through lactation and for 10 days following weaning of the pups (Yoshida et al., 1989). Based on reported dosages in previous studies by the same investigators, estimated daily dosages for mice exposed to 1000 mg Al/kg diet are as follows: 200 mg/kg-day during pregnancy and 420 mg/kg-day during lactation; doses for the mice exposed to 500 mg Al/kg diet are assumed to be approximately half of that of mice fed 1000 mg Al/kg, or 100 mg/kg-day in pregnant mice and 210 mg/kg-day in lactating mice (Golub et al., 1994). At weaning, dams and pups were inoculated with a tail vein injection of *Listeria monocytogenes* and monitored for mortality for 10 days. In a separate experiment, female mice, 6 weeks of age, were exposed to the same dietary Al levels for 6 weeks and then inoculated with *L. monocytogenes*. Estimated Al dosages were 5, 98 or 195 mg Al/kg bw-day for the 25, 500 or 1000 mg Al/kg dietary levels, respectively, based on a default food factor of 0.195 kg diet/kg bw-day assuming a reference "subchronic" food intake and body weight for female B6C3F1 mice over the period from weaning to 90 days (U.S. EPA, 1988). Inoculation resulted in significantly greater ($p < 0.025$) mortality in dams exposed to 500 or 1000 mg Al/kg diet compared to controls. There were no differences in mortality between the groups of inoculated pups or between groups of inoculated adult mice exposed to Al for 6 weeks. The LOAEL for pregnant mice was 100 mg Al/kg bw-day and the NOAEL for adult, non-pregnant mice was 195 mg Al/kg bw-day. Although the exposure duration in this study was only 7 weeks, it is included in Table 1 because it provides the only dose-response data on the effects of Al on resistance to pathogens.

Carcinogenicity studies

Schroeder and Mitchener (1975a) exposed 52 Long-Evans rats/sex/group to 0 or 5 ppm Al as potassium aluminum sulfate in drinking water for life. Based on default values for drinking water consumption and body weight for this strain of rat in a chronic study (U.S. EPA, 1988), these values are equivalent to Al doses of 0.472 and 0.67 mg/kg-day, for males and females, respectively. Study endpoints included body and heart weight; serum glucose, cholesterol and uric acid; and urinary protein, glucose and pH. All animals were necropsied at the time of natural death, and histological examinations were carried out on heart, lung, kidney, liver, spleen and gross tumors, for approximately 50% of the animals in the group. The only remarkable finding was a significant increase ($p < 0.005$) in gross tumor incidence in exposed male rats [13/25 (52%) compared to 4/26 (15%) in controls], although the tumor sites were not reported. Six of the tumors in the exposed males (46% of total) were considered malignant compared to two malignant tumors (50% of total) in the male controls. There were no significant differences in tumor incidences between exposed and control females.

In another study by the same investigators, 54 Swiss mice/sex/group were exposed to drinking water containing 0 or 5 ppm Al as aluminum potassium sulfate for life (Schroeder and Mitchener, 1975b). Based on default values for drinking water consumption and body weight for B6C3F1 mice in a chronic study (U.S. EPA, 1988), these values approximate to Al doses of 1.2 mg/kg-day in both males and females. Study endpoints included body weight, gross pathology,

and some limited histology of the heart, lung, liver, kidney and spleen. The incidences of gross tumors were 15/41 (36.6%) and 11/38 (28.9%) in exposed and control males, respectively, and 19/41 (46.3%) and 14/47 (29.8%) in exposed and control females, respectively, differences that did not achieve statistical significance by Fisher's exact test, although incidences of multiple tumors and lymphoma leukemia were considered by the authors to be significantly increased in females ($p < 0.025$ and $p < 0.05$, respectively). However, a definitive assessment of aluminum carcinogenicity in both this and the rat study (Schroeder and Mitchener, 1975a) is precluded by the limitations of the pathology examinations and reporting.

In a more recent study, the tumorigenic potential of aluminum potassium sulfate was assessed in B6C3F1 mice chronically exposed in the diet (Oneda et al., 1994). Sixty animals/sex/group were fed a diet containing 0, 1.0, 2.5, 5.0 or 10.0% (w/w) for 20 months. These concentrations of aluminum potassium sulfate (as the dodecahydrate) are equivalent to 0, 569, 1422, 2844 and 5687 ppm Al. Using food factors calculated with an algorithm relating food consumption to body weight (U.S. EPA, 1988) and body weight data estimated from growth curves reported by the investigators, the dosages of aluminum are estimated to be 0, 95, 237, 483 or 1024 mg Al/kg-day in males and 0, 97, 242, 512 or 1110 mg Al/kg-day in females. Clinical signs, food consumption, and body weight were evaluated weekly. Hematology, clinical chemistry or urine endpoints were not assessed. Necropsies that included organ weight measurements and comprehensive histological examinations (including brain) were performed on all animals, including those that died during the course of the study. Survival rates were higher than control values in all treated male and female groups, ranging from 86.7-95.0% compared to 73.3% in males and 86.7-91.7% compared to 78.3% in females. No changes in food consumption were observed, but body weight gain was increased in both sexes at 95-97 and 237-242 mg Al/kg-day (weights were 10-23% higher than controls at end of study), was similar to controls in both sexes at 483-512 mg Al/kg-day, and decreased in both sexes at 1024-1110 mg Al/kg-day (11-16% lower than controls at end of study). There were no exposure-related increased incidences of tumors, other proliferative lesions or non-neoplastic lesions. In fact, the incidence of spontaneous hepatocellular carcinomas was significantly decreased in males at 1024 mg Al/kg-day (5.5% compared to 20.5% in controls, $p < 0.01$).

Inhalation Exposure

Groups of 20 weanling Fischer 344 rats/sex and 20 weanling Hartley guinea pigs/sex were exposed to 0, 0.25, 2.5 or 25 mg/m³ aluminum chlorhydrate [$\text{Al}_2(\text{OH})_5\text{Cl} \cdot x(\text{H}_2\text{O})$] for 6 hours/day, 5 days/week for 6 months (Steinhagen et al., 1978). Analysis of the aluminum chlorhydrate by the investigators showed it to contain 24.5% Al, indicating that the animals were exposed to 0, 0.061, 0.61 and 6.1 mg Al/m³. Body weights were measured weekly for the first 8 weeks and biweekly thereafter. At the end of the exposure period, 10 animals (5/sex) of each species were sacrificed for organ weight measurements (heart, lung, liver, kidney, spleen and brain) and histological examination of the lungs, liver and kidney. In addition, comprehensive histological examinations were performed on animals in the control and 6.1 mg AL/m³ groups. The remainder of the animals was used for hematology evaluation (RBC, WBC, hematocrit and hemoglobin) and Al measurements in blood and tissues. Apparent effects of Al included multifocal granulomatous pneumonia in both species at ≥ 0.61 mg Al/m³, significantly increased absolute and relative lung weights in both species, and decreased body weight gain in rats and

minimal lung edema in guinea pigs at 6.1 mg Al/m³. The granulomatous reaction was characterized by foci of giant vacuolated particle-containing macrophages in the lungs and macrophages that did not appear to contain vacuoles or other evidence of phagocytized material in the peribronchial lymph nodes. There was a significant dose-related accumulation of Al in the lungs of both species at ≥ 0.061 mg Al/m³. However, a NOAEL of 0.061 mg/m³ could be identified for the onset of compound-induced histopathological effects.

In other studies, groups of 14-30 guinea pigs, rats and hamsters were exposed to fine metallic Al powders (pyro, atomized and flaked) at concentrations of 15, 30, 50 or 100 mg powder/m³ air for 6 hours/day, 5 days/week for 6 months (Gross et al., 1973). Alveolar proteinosis occurred in exposed animals of all three species after 2 months of exposure, but fibrosis or other pulmonary changes did not develop. Similarly, groups of 23 or 46 rats and 48 hamsters were exposed to undetermined concentrations of Al fumes or Al powder (20% Al, 80% Al(OH)₃) for morning hours only or morning and afternoon for up to 20 months (Christie et al., 1963). Effects were similar for both forms of Al in both species, including initial increased alveolar macrophage proliferation followed by nodular hyalinized areas, with development of pneumonia but no fibrosis.

Exposure to 2.18 mg Al fibers/m³ for 6 hours/day, 5 days/week for up to 86 weeks produced slightly increased alveolar macrophages and some irritation of the nasal passages in a group of 50 Alderly Park rats (Pigott et al., 1981). Finally, a study by Drew et al. (1974) observed the development of granulomatous nodules also developed in male hamsters that were exposed to 8 mg Al/m³ of *Alchlor* (a propylene glycol complex of aluminum-chloride-hydroxide) for 6 hours/day, 5 days/week for 20 or 30 exposures. The alterations persisted at the longest post treatment observation (6 weeks) and consistently developed at the bifurcation of the bronchioloalveolar ducts, which is a likely site of particulate deposition.

DERIVATION OF A PROVISIONAL CHRONIC RfD FOR ALUMINUM

This survey of the toxicological effects of Al in rodents suggests that neurotoxicological and developmental (including neurodevelopmental) endpoints are among the most sensitive indicators of Al toxicity. However, as vehicles for the development of toxicity values such as a provisional chronic RfD, the latter group of studies are considered to be more appropriate, since the level of exposure to Al appears to be better characterized. In fact, neurobehavioral deficits have been observed in mice and rats exposed during various stages of development and in subchronic studies (Bernuzzi et al., 1989; Donald et al., 1989; Golub et al., 1989, 1992a, b, 1995; Muller et al., 1990), as described above. These deficits include impaired operant learning, changes in grip strength, altered startle response and impaired motor coordination. In addition, several studies have shown that oral Al can produce histopathological changes in the CNS, although the histopathological lesions have yet to be causally related to the neurobehavioral deficits. Thus, Florence et al. (1994) reported histopathological changes in the brain of rats exposed to dietary Al for 6 months, the changes including the appearance of vacuolation of the cell body and cell processes of astrocytes in the brain and swelling of astrocytic processes. In addition, more localized vacuolization of neurons in the brain also was observed. These changes

were observed in rats exposed to elevated Al in the diet and are distinct from the NFD that has been observed in rats, rabbits and monkeys maintained on elevated dietary Al in combination with reduced dietary calcium (Garruto et al., 1989; Kihira et al., 1994; Mitani, 1992; Yano et al., 1989; Yoshida et al., 1990) or in rabbits administered intracisternal or intraventricular injections of Al (Kowall et al., 1989; Wakayama et al., 1993). Interpretation of the low-calcium studies is complicated by the observation that NFD was observed in animals maintained on low-calcium diets without excess Al and was enhanced by the addition of excess Al to these diets (Garruto et al., 1989; Kihira et al., 1994). Furthermore, Al has been shown to inhibit the gastrointestinal absorption of calcium (Orihuela et al., 1996), an effect that may exacerbate the calcium deprivation induced by low calcium diets. Thus, it is not clear whether calcium deprivation enhances the neurotoxicity of Al or Al exacerbates the adverse effects of calcium deprivation.

Donald et al. (1989) and Golub et al. (1995) are co-principal studies that identify a LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice exposed to dietary aluminum lactate (soluble aluminum) during gestation and lactation. The neurotoxicity associated with this LOAEL is consistent with LOAELs from other developmental and subchronic neurobehavioral studies in mice and rats which used higher dietary dosages of aluminum lactate or aluminum chloride (Golub et al., 1989, 1992a,b; Bernuzzi et al., 1989; Muller et al., 1990). Of the above, Golub et al., (1995) is the only study in which a histopathological examination of the brain and spinal cord was conducted and no abnormalities were reported. The Florence et al. (1994) study indicates that histopathological abnormalities of the CNS can occur in rats exposed subchronically to 84 mg/kg-day; although this is lower than the LOAEL for neurobehavioral effects, it was not chosen as the principal study because the functional significance of the histopathological lesions are uncertain.

A number of studies were identified that, at face value, appeared to indicate LOAELs at lower doses than the 100 mg Al/kg-day value selected herein, for example, Paternain et al. (1988) and Colomina et al. (1992). However, in these as in many of the studies under consideration, insufficient information on dietary Al (Al content and/or feed type) was reported to permit a reliable estimation of the overall dose level to which the animals were subjected.

Other developmental studies with aluminum hydroxide and/or citrate in mice and rats identified a NOAEL which are equivalent (95.5 mg Al/kg-day), or a minimum LOAEL that was greater (133 mg Al/kg-day) than the 100 mg Al/kg-day critical LOAEL (Domingo et al., 1989; Gomez et al., 1991), an overlap potentially related to differences in effective doses due to variations in unreported Al dietary content and factors affecting absorption such as chemical form (e.g., the use of less absorbable aluminum hydroxide). In addition, the LOAEL of 43.3 mg Al/kg-day for decreased body weight gain in mice exposed to aluminum chloride for 180-390 days (Ondreicka et al., 1966) was thought be inappropriate for risk assessment due to the small sample size and to the poor reporting of study details. Aluminum nitrate caused alterations in levels of brain biogenic amines and hepatic and hematological indices in rats exposed to 21.4 mg Al/kg-day for 6 weeks (Flora et al., 1991). This dose is not a LOAEL because insufficient information is available to determine if the effects are adverse.

Therefore, the LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice (Donald et al., 1989, Golub et al., 1995) is selected as the basis for the provisional chronic

RfD. The LOAEL is considered minimal because the results of the postweaning neurobehavioral test battery indicate that performance deficits may be marginal. In particular, of the three observed effects (decreased forelimb and increased hindlimb grip strengths, increased hindlimb foot splay distance), one effect (increased grip strength) has unclear toxicological significance and two effects (increased grip strength and foot splay distance) did not persist after 2 weeks of no further exposure.

Application of an uncertainty factor (UF) of 100 (3 for use of a minimal LOAEL, 10 for interspecies extrapolation and 3 for intrahuman variability where the critical effects have been observed in a sensitive sub-group) results in a provisional RfD of

$$\text{p-RfD} = 1\text{E-0 mg Al/kg-day.}$$

The provisional RfD of **1E-0 mg Al/kg-day** is approximately 3-fold higher than estimated normal daily Al intake of approximately 0.2-0.3 mg/kg-day (Iyengar et al., 1987; Ganrot, 1986; Wilhelm et al., 1990). Chronic users of medications such as antacids, buffered aspirins and antiulceratives would be expected to ingest much larger amounts of Al, possibly as high as 10-70 mg/kg-day. However, these subjects would not represent the most sensitive population (developing infants), as indicated by the animal data.

Low confidence is placed in the co-critical studies, because they only identify a LOAEL for a sensitive effect and evaluated comparatively small numbers of animals. Confidence in the data base is low because the most reliable supporting data for neurotoxicity of Al in humans are of limited general relevance (e.g., dialysis encephalopathy is manifested in patients with impaired renal function and excessive Al uptake from intravenous exposure). In fact, neurotoxicity remains to be assessed in animals chronically exposed to Al, and developmental morphology has not been adequately investigated in two animal species. These limitations in the Al data base do not increase uncertainty in the RfD; therefore, a data base uncertainty factor was not used. However, reflecting the low confidence in the co-critical studies, there is low overall confidence in the RfD.

DERIVATION OF A PROVISIONAL CHRONIC RfC FOR ALUMINUM

Al seems to be the most likely cause for the generally and consistently reported psychomotor and cognitive effects (particularly signs of impaired coordination) in Al production workers and welders (Bast-Pettersen et al., 1994; Rifat et al., 1990; Hosovski et al., 1990; White et al., 1992; Hanninen et al., 1994; Sjogren et al., 1990, 1996). In addition, there is strong evidence that Al is neurotoxic by other routes of exposure. Thus, a degenerative neurological syndrome (dialysis dementia) has been documented in humans with chronic renal failure, apparently due to an increased exposure to Al from dialysis treatment and/or ingestion of phosphate binding agents which contain Al (Alfrey, 1993). This syndrome is characterized by gradual loss of motor, speech and cognitive functions. Neurotoxicity, particularly neuromuscular effects such as decreased motor activity, startle responsiveness and grip strength, has also been observed in mice following subchronic oral exposure and in the offspring of mice and rats exposed orally during gestation and/or lactation. Based on this information, as well as evidence

that Al is absorbed by Al production workers and welders, the hypothesis that the occupational studies are indicative of a neurotoxic effect of Al appears to be justified. However, the only occupational study that has yielded suitable monitoring data is that of Hosovski et al. (1990), in which workers were exposed to presumed time-weighted average (TWA) concentrations of 4.6-11.5 mg Al/m³ magnitude for an average of 12 years. Using 4.6 mg Al/m³ as the LOAEL for psychomotor and cognitive impairment for an 8-hour occupational exposure (Hosovski et al., 1990) and corrections for discontinuous exposure (10 m³/20 m³ and 5 days/7 days), the LOAEL_{HEC} is 1.64 mg/m³. Applying an uncertainty factor of 300 for intrahuman variability (10), use of a LOAEL (10) and an incomplete database (3) yields a provisional RfC of

$$\text{p-RfC} = 1.64 \text{ mg/m}^3 / 300 = 5\text{E-}3 \text{ mg/m}^3.$$

The lack of inhalation developmental studies may increase uncertainty in the database because oral data in animals indicate that neurotoxic and morphological developmental effects may occur at lower doses than neurotoxicity in adults. Additionally, there is uncertainty related to the lack of corroborating data on air concentrations associated with neurotoxicity. Confidence in the critical study is low to medium because only a LOAEL was identified. Confidence in the database is medium because (1) there are no corroborating data on effect levels (NOAELs and additional LOAELs), (2) no data are available for developmental neurotoxicity by the inhalation route and (3) a well-designed two-generation reproduction study is lacking. Reflecting the low to medium confidence in the critical study and database, there is low to medium confidence in the provisional RfC.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR ALUMINUM

Weight-Of-Evidence Classification

A considerable number of epidemiological studies have examined the incidence of excess tumor formation in persons occupationally exposed to Al in the form of dusts or fumes. In general, a body of inferential evidence exists for an increase in cancer of the bladder and lung through such occupational exposure to Al, although conclusions linking these responses to the effects of Al are confounded by attendant co-exposure to other harmful emissions such as PAHs and by cigarette smoking. A 20-month exposure of B6C3F1 mice to Al potassium sulfate dodecahydrate in the diet at concentrations up to 10% w/w displayed no indication of compound-related carcinogenicity and, in general, no indication of adverse toxicological effects of any kind (Oneda et al., 1994). Similarly, the life-time exposure of Swiss mice and Long-Evans rats to 5 ppm Al as aluminum potassium sulfate in drinking water provided no convincing evidence for the carcinogenicity of Al compounds (Schroeder and Mitchener, 1975a,b). Gene reversion experiments on Al compounds resulted in negative results in *S. typhimurium* (Ahn and Jeffrey, 1994). Taking all of the evidence of Al carcinogenicity together, and in accordance with the U.S. EPA (2005) cancer guidelines, aluminum is classified as *inadequate information to assess carcinogenic potential*. The basis for this classification is insufficient evidence in epidemiological/occupational studies, lack of demonstrated carcinogenicity or mutagenicity in

available animal studies, lack of positive evidence of non-carcinogenicity and lack of mode of action data for aluminum.

Quantitative Estimates of Carcinogenic Risk

Due to insufficient data, a provisional oral slope factor and inhalation unit risk could not be developed.

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Table 1. Summary of oral toxicity data for aluminum^a

Study	Type	Species	Al	Exposure Concentration (ppm)	Exposure Dosage (mg Al/kg-day)	Exposure Frequency and Duration	Critical Effect	NOAEL (mg Al/kg-day)	LOAEL (mg Al/kg-day)	FEL (mg Al/kg-day)
Ondreicka et al., 1966	Subchronic 3-gen dietary	Dobra Voda mice	chloride	--	27 (control), 46	Continuous, 180-390 days	Decreased body weight gain in F1 and F2.	--	46	--
Golub et al., 1989	Subchronic dietary	S-W mice	lactate	25 (control), 500,1000	3.3 (control), 65,130	Continuous, 6 weeks	Decreased spontaneous motor activity; decreased weight gain.	65	130	--
Golub et al., 1992a	Subchronic dietary	S-W mice	lactate	25 (control), 1000	190	Continuous, 90 days	Decreased hindlimb grip, decreased spontaneous motor activity, decreased startle response.	--	190	--
Florence et al., 1994	Subchronic dietary	Wistar rat	chloride (with citric acid)	1.52 (control), 1000	0.13 (control), 84	Continuous, 6 months	Histopathological changes in brain astrocytes and neurons.	--	84	--
Domingo et al., 1996	Subchronic drinking water	Sprague Dawley rats	nitrate (with citric acid)	--	0, 50, 100 (plus unreported dietary Al)	Continuous, 6.5 months	Operant conditioning and performance	100	--	--
Yoshida et al., 1989	Subchronic dietary	S-W mice	lactate	25 (control), 500, 1000	5 (control), 98, 195	Continuous, 7 weeks	Increased mortality from <i>L. monocytogenes</i> inoculation	195	--	--
Donald et al., 1989	Developmental dietary	S-W mice	lactate	25 (control), 500, 1000	5 (control), 100, 200	Continuous, gestation and lactation	Neurobehavioral effects.	--	100	--
Golub et al., 1992b	Developmental dietary	S-W mice	lactate	25 (control), 1000	4 (control), 174	Continuous, gestation and lactation	Neurobehavioral effects.	--	174	--
Golub et al., 1995	Developmental dietary	S-W mice	lactate	7, 500, 1000	1 (control), 100, 200	Continuous, gestation, lactation to maturity	Neurobehavioral effects.	--	100	--

Table 1. Summary of oral toxicity data for aluminum^a

Study	Type	Species	Al	Exposure Concentration (ppm)	Exposure Dosage (mg Al/kg-day)	Exposure Frequency and Duration	Critical Effect	NOAEL (mg Al/kg-day)	LOAEL (mg Al/kg-day)	FEL (mg Al/kg-day)
Yoshida et al., 1989	Developmental dietary	S-W mice	lactate	25 (control), 500, 1000	4 (control), 100, 200	Continuous, gestation and lactation	Increased mortality of dams from <i>L. monocytogenes</i> inoculation	--	100	--

^aStudies for which total dosages were reported or could be estimated (unless otherwise noted).

2-2-2005

Provisional Peer Reviewed Toxicity Values for

Ammonia
(CASRN 7664-41-7)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR AMMONIA (CASRN 7664-41-7)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

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Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

The HEAST (U.S. EPA, 1997) lists subchronic and chronic oral reference doses (RfDs) of 34 mg/L for ammonia. A comment in the HEAST indicates that 34 mg/L is a concentration in drinking water that is specifically related to the organoleptic (taste) threshold and that a safe concentration for ammonia may be higher than 34 mg/L, but the data are inadequate to assess the safe level. The source document for derivation of the HEAST subchronic and chronic oral RfD values is the Health Effects Assessment (HEA) for Ammonia (U.S. EPA, 1987). The HEAST subchronic and chronic RfD values are based on a determination of the organoleptic (taste) threshold of ammonia in redistilled water by Campbell et al. (1958). The value selected for the HEAST subchronic and chronic RfDs was supported by the closely similar value of 35 mg/L identified as the taste threshold for ammonia in a World Health Organization Environmental Health Criteria (EHC) document (WHO, 1986) and as the ambient water quality criterion to

protect human health derived by U.S. EPA (1981). No oral assessment is included on IRIS (U.S. EPA, 2003) or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2002). No relevant documents other than the HEA and the AWQC document were included in the CARA list (U.S. EPA, 1991, 1994a).

The HEAST includes a value of $1\text{E-}1\text{ mg/m}^3$ for the subchronic inhalation RfC. The HEAST subchronic RfC used the same data and is the same as the RfC ($1\text{E-}1\text{ mg/m}^3$) reported in IRIS (U.S. EPA, 2003). The IRIS chronic RfC value was derived from a free-standing NOAEL of 6.4 mg/m^3 (9.2 ppm) identified for lack of evidence of decreased pulmonary function or changes in subjective symptomatology in an occupational study of workers exposed to ammonia in a soda ash (sodium carbonate) facility (Holness et al., 1989). A LOAEL was not identified in the study. The NOAEL was adjusted for intermittent exposure to a value of 2.3 mg/m^3 and divided by a composite uncertainty factor (UF) of 30. The composite UF included a factor of 10 for protection of sensitive individuals and a factor of 3 for database deficiencies, including lack of chronic data, proximity of the occupational $\text{NOAEL}_{\text{HEC}}$ to a $\text{LOAEL}_{\text{HEC}}$ observed in a subchronic inhalation study in rats (Broderson et al., 1976), and lack of data on reproductive or developmental toxicity. The RfD/RfC Workgroup verified the RfC on February 21, 1991. The HEA had previously derived subchronic and chronic inhalation RfDs of 0.36 mg/m^3 by dividing the ammonia air odor threshold of 3.6 mg/m^3 (Carson et al., 1981) by an uncertainty factor of 10 to obtain an estimate of the lower bound limit for odor detection.

The public review draft of the ATSDR Toxicological Profile on ammonia (ATSDR, 2002) derived an intermediate oral minimal risk level (MRL) value of 0.3 mg/kg-day based on a duration-adjusted NOAEL of 39.5 mg/kg-day for weight loss in rats exposed to ammonium sulfamate in drinking water for 90 days (Gupta et al., 1979) and an uncertainty factor of 100 (10 for extrapolation from rats to humans and 10 to protect sensitive individuals). ATSDR (2002) also derived a chronic inhalation MRL of 0.3 ppm ($200\text{ }\mu\text{g/m}^3$) based on a duration-adjusted NOAEL of 3.1 ppm in the Holness et al. (1989) study and an uncertainty factor of 10 for human variability. The State of California (OEHHA, 2002) has derived a chronic inhalation reference exposure level of $200\text{ }\mu\text{g/m}^3$ (0.3 ppm) for ammonia. This value ($200\text{ }\mu\text{g/m}^3$) is based on the occupational study of Holness et al. (1989) with a duration adjusted NOAEL of 2 mg/m^3 and an uncertainty factor of 10 for intraspecies variability. The OEHHA (2002) used the same methodology as ATSDR. ACGIH (2001) lists a TLV-TWA of 25 ppm (17 mg/m^3) and a STEL of 35 ppm (24 mg/m^3) for ammonia. These values are intended to minimize the potential for acute ocular and respiratory tract irritation. NIOSH (2002) lists values of 25 ppm (18 mg/m^3) and 35 ppm (27 mg/m^3) for the REL-TWA and REL-ST, respectively. OSHA (2002) lists a value of 50 ppm (35 mg/m^3) for the PEL-TWA.

Ammonia is not included in the HEAST (U.S. EPA, 1997) cancer table. IRIS (U.S. EPA, 2003) and the Drinking Water Standards and Health Advisories list (U.S. EPA, 2002) do not provide a carcinogenicity assessment for ammonia. IARC (2002) has not evaluated the

carcinogenicity of ammonia. NTP (2002) does not list ammonia among the chemicals it considers to be known human carcinogens or reasonably anticipated to be human carcinogens.

Literature searches to identify studies relevant to the derivation of provisional toxicity values for ammonia were conducted for the period 1988 through September 18, 2002. Databases searched included: TOXLINE, MEDLINE, TSCATS, RTECS, CCRIS, DART, EMIC/EMICBACK, HSDB, GENETOX and CANCERLIT. Additional literature searches were conducted through May 2004 by NCEA-Cincinnati using TOXLINE, MEDLINE, Chemical and Biological Abstract databases and no relevant information was found.

REVIEW OF PERTINENT DATA

Human Studies

Holness et al. (1989) studied workers exposed to ammonia in a sodium carbonate production plant. Fifty-two of the 64 available workers agreed to participate in the study. The control group consisted of 31 office and stores workers employed at the plant who were without previous exposure to ammonia. Information was collected on age, height, work history, smoking history, respiratory symptoms, and skin and eye complaints. Respiratory questions were based on an American Thoracic Society questionnaire. Sense of smell was evaluated at the beginning and end of the work week. Pulmonary function tests were performed at the beginning and end of each work shift on two test days. The parameters measured were forced vital capacity (FVC), forced expiratory volume in one second (FEV_1); and forced expiratory flow rate at 50% and 75% of the vital capacity (FEF_{50} and FEF_{75}). Mean time-weighted average (TWA) exposures to ammonia were determined by personal air sampling over one shift following NIOSH recommendations. The average sampling time was 8.4 hours. The mean age of the exposed workers was 38.9 ± 11.7 years and the average duration of exposure was 12.2 ± 8.9 years. Only weight differed significantly when demographics for the exposed and control workers were compared. Time-weighted average airborne concentrations of ammonia were 9.2 ± 1.4 ppm (6.4 mg/m^3) and 0.3 ± 0.1 ppm (0.2 mg/m^3) for the exposed and control groups, respectively. Although no significant difference was evident between exposed and control groups in reporting of respiratory symptoms, workers reported that exposure at the plant aggravated specific symptoms including coughing, wheezing, nasal complaints, eye irritation, throat discomfort, and skin problems. No significant differences were evident between the exposed and control groups in reporting of respiratory symptoms, sense of smell, baseline lung function, or change in lung function at the beginning and end of a work week. No significant relationships between level or length of ammonia exposure and lung function results were demonstrated. The NOAEL in this study was 9.2 ppm (6.4 mg/m^3), based on lack of evidence for decreased pulmonary function or changes in subjective assessments of respiratory symptoms. A LOAEL was not identified in this study.

Ferguson et al. (1977) exposed healthy human volunteers (2/concentration) employed in an alkali plant to ammonia concentration of 25 ppm, 50 ppm, or 100 ppm, 5 days/week for six weeks. Conclusions of the study were actually based on 5 weeks of exposure as a result of technical difficulties during the first week of the study. Toxicity was assessed by subjective and objective indications of eye and respiratory tract irritation, pulse rate, respiration rate, pulmonary function (FVC, FEV), physical examination, and the ability to perform routine tasks. Exposure to ammonia did not result in abnormalities of the chest, heart, vital organs, neurological response, task performance or significant weight changes as assessed during weekly medical examinations. Transient irritation of the throat was observed at exposures of 50 ppm (4 hours/day).

More recently published occupational studies were examined to identify data potentially suitable for calculation of a subchronic RfC. Ballal et al. (1998) reported the results of a cross-sectional study of male workers employed in two fertilizer plants in Saudi Arabia. Exposure to ammonia concentrations of 25 ppm and above were significantly associated with respiratory symptoms including wheezing, cough, phlegm, dyspnea, and asthma. Ali et al. (2001) examined the pulmonary function of workers (gender not specified) in an ammonia-producing factory in Saudi Arabia. Cumulative exposure of greater than 50 mg/m³-years was associated with significantly reduced FEV₁ and FVC. Symptomatic workers (i.e., those reporting cough, phlegm, wheeze, and/or dyspnea) showed significantly reduced FEV₁ and FEV₁/FVC ratio when compared to asymptomatic workers. Neither study adequately reported details of worker exposure, such as the number of hours worked per week, and were not further considered for derivation of reference doses.

A number of studies have examined the relationship between inhalation exposure to pollutants (including ammonia) in livestock confinement buildings and occurrence of respiratory symptoms and/or changes in pulmonary function in workers (Heerderik et al., 1990; Choudat et al., 1994; Donham et al., 1995, 2000; Reynolds et al., 1996; Vogelzang et al. 1997, 2000; Cormier et al., 2000). Exposure to ammonia concentrations of 2.3 to 20.7 ppm was associated with symptoms of bronchial reactivity, inflammation, cough, wheezing, or shortness of breath and decrements in pulmonary function as measured by FEV₁, maximum expiratory flow rate, and maximal mid-expiratory flow rate. These data are of limited use for derivation of a subchronic toxicity reference value for ammonia because workers were concurrently exposed to other potential respiratory toxicants such as dusts, endotoxins, and nitrogen dioxide.

The carcinogenic potential of ammonia via the inhalation route has not been assessed in humans.

The experimental database for human oral exposure to ammonia consists of acute and short-term studies of exposure to ammonium chloride. No subchronic or chronic duration oral exposure studies were located in the literature examined. The availability of studies on ammonium chloride is a result of its use for experimental induction of hyperchloremic metabolic

acidosis. Few of the available studies have been designed or conducted to specifically assess the toxicity of ammonia or ammonium ion in response to oral dosing.

U.S. EPA (1981) reviewed fifteen existing short-term studies of ammonium chloride in humans. Administration of ammonium chloride to all age groups produced metabolic acidosis, with increased susceptibility observed in infants. The results of these studies indicate that metabolic acidosis, impaired glucose tolerance, and reduced tissue sensitivity to insulin may result from doses of ammonium chloride greater than or equal to 100 mg/kg-day (31.8 mg ammonia/kg-day, as estimated by U.S. EPA, 1981). Although frank toxicity was not reported, U.S. EPA (1981) expressed concern for potential bone demineralization as a result of impaired acid-base balance.

In the longest duration human study found, Lemann et al. (1966) investigated the electrolyte balance of five men who were given doses of ammonium chloride to induce metabolic acidosis. Each individual served as his own control. Following baseline observations, each subject was given a small initial dose which was progressively increased over a period of six to nine days, after which the dose remained constant until administration of ammonium chloride was discontinued after day 18. U.S. EPA (1987) reported total doses of 733 mEq (approximately 93 mg/kg-day) for the initial loading period and 2771 mEq (approximately 177 mg/kg-day) for the remainder of the experiment. During ammonium chloride loading, net fixed acid production was increased by an average of 3425 mEq. Progressive acid retention was initially accompanied by a progressive decrease in serum bicarbonate concentration. Serum bicarbonate levels dropped as acid was retained during the first nine days of ammonium chloride dosing, stabilized at a reduced level by about day 12, and rose slightly between days 13 to 18, but did not return to baseline levels until after treatment with ammonium chloride was discontinued. Calcium and phosphorus balances became negative as a result of urinary losses, suggesting to the study authors that slow dissolution of bone mineral was occurring to provide additional buffering capacity. The LOAEL in this study was the initial dose of 93 mg/kg-day.

The carcinogenic potential of ammonia via the oral route has not been assessed in adequately designed epidemiological studies.

Animal Studies

Broderson et al. (1976) continuously exposed F344 rats (6 rats/sex/dose) to ammonia concentrations of 25, 50, 150, or 250 ppm for seven days prior to inoculation with *Mycoplasma pulmonaris* and for 28 to 42 days following inoculation. These exposures were conducted using purified ammonia from a commercial source. In addition, one treatment group was exposed to ammonia produced from a natural source (soiled bedding) for 30 days following inoculation. Each treatment group had a corresponding control group that was inoculated with *M. pulmonaris* and exposed only to background levels of ammonia. Additional groups were exposed to

background or high levels of ammonia (trace and 250 ppm, respectively) without *M. pulmonaris* inoculation. Toxicity was assessed by observation of clinical signs and histopathological examination of nasal passages, middle ear, trachea, lungs, liver, kidney, adrenal, pancreas, testicle, spleen, mediastinal nodes, and thymus. Clinical signs were similar in control and exposed groups during the pre-inoculation exposure period. Signs of murine respiratory mycoplasmosis (MRM) were observed in all groups approximately 10 days after inoculation. All levels of ammonia from bedding or the commercial source increased the severity of the rhinitis, otitis media, tracheitis, and pneumonia characteristic of MRM. The prevalence and extent of gross atelectasis and consolidation were greater in rats exposed to high ammonia concentrations (i.e., ammonia concentrations greater than background) and the prevalence of microscopic respiratory lesions was also greater. The prevalence of gross and microscopic lung lesions differed significantly from controls when data from all high exposure groups were summed and compared with pooled control data. Regression analysis indicated a positive relationship between ammonia concentration and prevalence of gross or microscopic lesions. Exposure of uninoculated rats to ammonia resulted in lesions that were unlike those of MRM and which were restricted to the nasal passages. A LOAEL of 25 ppm (17.4 mg/m³) was identified in this study.

Schoeb et al. (1982) inoculated pathogen-free F344 rats with *M. pulmonis* and exposed groups to trace or 100 ppm (70 mg/m³) concentrations of ammonia for up to 28 days. Growth of *M. pulmonis* was greater in ammonia-exposed rats than in controls and serum immunoglobulin response to the inoculum was also greater in the exposed population. Results of an experiment conducted in rats with cannulated tracheas demonstrated that the nasal passages absorbed virtually all ammonia at administered concentrations of 500 ppm (348 mg/m³) or below.

Coon et al. (1970) continuously exposed male and female Sprague-Dawley and Long Evans rats for a minimum of 90 days to ammonia concentrations of 0, 40, 127, 262, 455, or 470 mg/m³. A LOAEL of 262 mg/m³ was identified on the basis of nasal discharge in 25% of the rats and nonspecific degenerative and circulatory changes in the lungs and kidneys. The upper respiratory tract was not examined for microscopic lesions. In another series of experiments, Coon et al. (1970) exposed rats, guinea pigs, rabbits, dogs and monkeys to ammonia concentrations of 0, 155, or 770 mg/m³ for 8 hours/day, 5 days/week for a total of 30 exposures. This study identified a LOAEL of 770 mg/m³ for lung inflammation in rats and guinea pigs and ocular and nasal irritation in dogs and rabbits. The upper respiratory tract was not examined for presence of lesions.

Anderson et al. (1964) conducted a series of experiments that included continuous exposure of guinea pigs and Swiss albino mice to 20 ppm (13.9 mg/m³) ammonia for up to six weeks and exposure of Leghorn chickens for up to 12 weeks. A separate group of guinea pigs was exposed to 50 ppm (35 mg/m³) ammonia for six weeks. Although no effects were observed after exposure to 20 ppm for four weeks, gross lesions including edema, congestion, and hemorrhage were observed in the lungs of all three species after six weeks. Grossly enlarged and

congested spleens, congested livers and lungs, and pulmonary edema were observed in guinea pigs exposed to 50 ppm ammonia for six weeks.

Weatherby (1952) exposed guinea pigs to 0 or 170 ppm (118 mg/m³) 6 hours/day, 5 days/week for up to 18 weeks. No adverse effects were observed in animals exposed for 6 to 12 weeks. Mild changes were observed in the spleen, kidney suprarenal glands, and liver at 18 weeks. No effects on the lungs were observed. The upper respiratory tract was not examined for lesions.

No effects on ovarian or uterine weights were observed in pigs exposed by inhalation to approximately 5 or 35 ppm ammonia for 6 weeks (Diekman et al., 1993). Continuous exposure of female pigs to approximately 35 ppm ammonia from 6 weeks prior to breeding through gestation day 30 did not significantly affect age to puberty, number of live fetuses, fetus-to-corpus luteum ratio, or fetal length when compared to females exposed to 7 ppm ammonia for the same duration (Diekman et al., 1993).

Limited subchronic and chronic toxicity data are available for ammonia. In an early study, Seegal (1927) administered ammonium chloride doses of 0 or 372 mg/kg-day to rabbits by gavage for 36 days and observed episodes of severe metabolic acidosis and epithelial degeneration in the renal tubules. Similar effects plus softening of the teeth, skull, and ribs were observed at a dose of 234 mg/kg-day given by gavage for 11 months.

Freedman and Beeson (1961) exposed 12 adult male Sprague-Dawley rats to 1.6% ammonium chloride in the drinking water for periods of up to three weeks to evaluate effects on the kidney. Six control animals were provided with tap water. An additional group of 10 rats was given drinking water containing 1% ammonium chloride for an additional 2.5 months to assess subchronic effects. No abnormalities were detected by urinalysis, gross pathology or histologic examination. Physiological adaptation to metabolic acidosis was indicated by increased glutaminase activity per gram of kidney with duration of treatment. No data on water consumption or body weight of the test animals were provided. Assuming that the rats weighed 250 grams and consumed 25 mL of drinking water per day, U.S. EPA (1987) estimated a time-weighted average dose of approximately 360 mg/kg-day.

Gupta et al. (1979) conducted a subchronic exposure study in adult female and weanling male and female ITRC rats (20/sex/age/dose). The test animals were treated with ammonium sulfamate (NH₄SO₃NH₂) at doses of 0, 100, 250, or 500 mg/kg-day, 6 days per week for 30, 60, or 90 days. The ammonium sulfamate was given as a 10% solution, but the study report did not clearly indicate whether the dose was administered by gavage. Food and water consumption, appearance, behavior, and body weight were monitored during the study. Hematological parameters and organ weights were measured at interim and terminal sacrifices and tissue samples were collected for histopathological examination. Food and water consumption were

decreased in male and female weanlings at the 500 mg/kg-day dose relative to the controls. No compound-related clinical signs of toxicity were observed in dosed rats. Body weight of adult females receiving 500 mg/kg-day was significantly reduced at 60 day (9%) and 90 days (16%) when compared to the control group. No significant differences were noted in hematological parameters, organ weights, or histopathology. Although the study authors indicated that ammonium sulfamate would be expected (on the basis of its structure) to cause metabolic acidosis, this prediction does not appear to have been confirmed experimentally. These data identify NOAEL and LOAEL values of 250 and 500 mg/kg-day as ammonium sulfamate, respectively. The effective dose of ammonia at each of these dose levels is uncertain, because under certain conditions the sulfamate ion is hydrolyzed to bisulfate ion and ammonia (U.S. EPA, 1981, 1987). Assuming no hydrolysis of the sulfamate ion, these doses correspond to 37.3 and 74.8 mg/kg-day of ammonia, respectively.

Bodega et al. (1993) fed diets containing 0 or 20% ammonium acetate to pathogen-free female Wistar rats (5 rats/group) for 3, 7, 15, 45, or 90 days to assess effects on glial fibrillary acidic protein (GFAP) in the spinal cord. The ammonium acetate in the diet was supplemented by addition of 5 mM ammonium acetate to the drinking water. The total exposure from the combined food and water was not provided by the author. Exposure to ammonium acetate had no effect on behavior, water consumption, or spinal GFAP levels of the test animals. Body weight gain was significantly reduced in dosed animals at all time points. Body weight gain in animals exposed to ammonia for 90 days was 69% of the control value.

Fazekas (1939, 1954a,b) conducted studies in rabbits that ranged from 3 to 17 months in duration. The administration of various ammonium salts (carbonate, chloride, sulfate, hydrophosphate, acetate, or lactate) or ammonium hydroxide resulted in enlargement of the parathyroids. Similar results were obtained with a variety of other chemicals (sodium dihydrophosphate, sodium ammonium phosphate, calcium chloride, hydrochloric acid, acetic acid, lactic acid) (Fazekas, 1954a). The chemicals were given for three week periods separated by one week intervals. The administered dose of ammonium salts in this study is unclear, but based on descriptions in secondary sources is likely to be less than or equal to 0.4 mg/kg-day. In a related study, similar treatment of rabbits with ammonium chloride or ammonium sulfate resulted in fluctuations in serum calcium and phosphorus levels (Fazekas, 1954b). Rabbits given gavage doses of 100 mg/kg by gavage on alternate days and then daily for 17 months developed enlarged adrenal glands. An initial fall in blood pressure of 20 to 30 mm Hg was followed by a gradual rise to levels 10 to 30 mm Hg after several months of treatment.

In a chronic study, Barzel and Jowsey (1969) exposed male Sprague-Dawley rats to 1.5% ammonium chloride in the drinking water for 330 days. The effects of ammonium on animals receiving a nutritionally complete diet included decreased bone content of fat-free solid and calcium; decreased body weight and body fat; and decreased blood pH and plasma carbon dioxide. Barzel (1975) reported effects on bone (decreased density, ash weight, and calcium

content), but no effects on growth, in intact and ovariectomized female rats exposed to 1.5% ammonium chloride in the drinking water for 300 days. U.S. EPA (1981) estimated an average daily dose of 1500 mg/kg-day for both studies.

The carcinogenic potential of ammonia has been investigated in an oral bioassay conducted in mice. Toth et al. (1972) exposed male and female Swiss mice (49-50/sex/dose) to 0.1, 0.2, or 0.3% ammonium hydroxide in the drinking water for their lifetime. U.S. EPA (1987) estimated an average daily dose of 565 mg/kg-day at the highest concentration. Male and female C3H mice (40/sex) were exposed to 0.1% ammonium hydroxide in the drinking water for their lifetime. This concentration corresponded to average daily doses of approximately 270 mg/kg-day, respectively, as calculated by U.S. EPA (1987). While data for a control group are reported in the publication, it is not clear whether this group was run concurrently with the ammonia treatment groups. The mice were examined and weighed at weekly intervals. Moribund animals were humanely sacrificed. Complete necropsies were performed on all animals and the liver, kidney, spleen, lung, and organs with gross lesions were processed for histopathological examination. No evidence for carcinogenicity was observed in males or females of either strain.

Two studies have examined the interaction of ammonia with other compounds in the induction of tumors. Uzvölgyi and Bojan (1980) investigated the interaction of ammonia with diethyl pyrocarbonate (DEPC) in induction of lung tumors in CFPL mice (a urethane-sensitive strain). Mice given gavage doses of either ammonia or DEPC alone did not develop lung tumors, whereas development of lung tumors was observed in mice dosed with both ammonia and DEPC. Induction of tumors in the sensitive CFPL strain may have resulted from formation of urethane *in vivo* from ammonia and DEPC (Uzvölgyi and Bojan, 1985). Tsujii et al. (1995) studied the effect of ammonia on tumor development in male Sprague-Dawley rats pretreated with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) in the drinking water for 24 weeks and subsequently exposed to drinking water containing 0 or 0.01% ammonia for an additional 24 weeks. Exposure to ammonia significantly increased the incidence, multiplicity, size, and depth of tumors in the glandular stomach and stimulated cell proliferation in the gastric mucosa.

Reproductive and developmental toxicity data on ammonia from animal studies are limited. Treatment of virgin female rabbits with oral doses of various ammonium salts (carbonate, chloride, hydrophosphate, or sulfate) or ammonium hydroxide was associated with enlargement of the ovaries, follicle maturation, and formation of corpora lutea (Fazekas, 1949). Enlargement of the uterus, hypertrophy of the teats, and secretion of milk were also reported in treated rabbits. However, several aspects of this study are poorly documented, including the use of controls and exact method of dose administration.

Minaña et al. (1995) examined the effect of prenatal exposure to 20% ammonium acetate in the diet on NMDA receptor function in Wistar rats. As judged from graphically presented data, offspring of dams treated from day 1 of pregnancy through lactation had body weights at

birth that were comparable to the control group. The body weight of weanlings maintained on a diet containing 20% ammonium acetate was reduced by approximately 27% and 26% in males and females, respectively, at 120 days of age when compared to animals maintained on the control diet. Rats exposed to ammonia during pregnancy and lactation and fed an unsupplemented diet at weaning had a lower growth rate than the controls until day 60, indicating persistent effects of prenatal and lactational exposure to ammonia. Prenatal exposure to ammonia reduced binding of [³H]MK-801 to NMDA receptors in primary cultures of cerebellar neurons by approximately 60%. No data were provided for feed intake in this study; therefore, an average daily dose can not be reliably estimated.

Other Studies

Information on the toxicokinetic properties of ammonia have been reviewed and summarized in ATSDR (2002). Inhalation exposure studies in humans show that ammonia dissolves in the mucous of the upper respiratory tract. At low levels of exposure, most inhaled ammonia is retained in the upper respiratory system. As the ammonia concentration increases, the capacity of the upper respiratory system is saturated and a larger percentage is absorbed. Development of nasal and pharyngeal irritation, but not tracheal irritation, following exposure is consistent with retention of inhaled ammonia in the upper respiratory tract. Animal data provide supporting evidence for high nasal retention. Quantitative differences in the amount of ammonia in inhaled and exhaled air suggest that small amounts are absorbed across the nasopharyngeal membranes into the systemic circulation. Limited systemic absorption is also inferred from lack of change in blood nitrogen and urinary-ammonia compounds following exposure. The available evidence suggests that ammonium absorbed via inhalation would be distributed to all body compartments by the blood. Ammonium reaching the tissues would be used in protein synthesis or as a buffer, with excess levels reduced by urinary excretion or conversion in the liver to glutamine and urea. Absorbed ammonia is excreted by the kidneys as urea and urinary ammonium compounds. Bioaccumulation to toxic levels is not expected to occur from chronic inhalation exposure based on the low levels of absorption and existence of multiple effective mechanisms for detoxification and excretion.

Human data indicate that ingested ammonium compounds are readily absorbed. The absorbed ammonium ion is transported via the hepatic portal vein to the liver, where most is metabolized to urea in healthy individuals. Data from animals and humans suggest that little of the ingested compound reaches the systemic circulation as ammonia or ammonium ion. Ingested ammonium compounds are excreted primarily in the urine as urea. Small amounts may be excreted in the sweat or in exhaled air.

Genotoxicity data are available from studies in humans, mice, *Escherichia coli*, *Drosophila melanogaster*, and cultured chick fibroblast cells. Yadav and Kaushik (1997) conducted cytogenetic assays on blood samples collected from 22 workers exposed to ammonia

gas (ambient level = 0.09 mg/m³) during production of nitrogen fertilizers and 42 unexposed staff employed at the same facility. The exposed workers did not show clinical symptoms of ammonia toxicity. The mitotic index, total number of chromosome aberrations (CA), and frequency of sister chromatid exchange (SCE) were significantly increased in the exposed workers as compared to their matched controls. The frequency of CA and SCE increased with the duration of exposure. Concurrent exposure to other compounds such as nitrogen dioxide was not addressed in the study report. The frequency of micronuclei was significantly increased in Swiss albino mice treated with intraperitoneal doses of ammonia ranging from 12.5 to 50 mg/kg as compared to controls (Yadav and Kaushik, 1997). Positive results were obtained for reverse mutation in *E. coli*, but only at levels of ammonia that were cytotoxic (Demerec et al., 1951). Negative results were reported for ammonium sulfate in *Salmonella typhimurium* and *Saccharomyces* (Litton Bionetics, 1975). Positive results were observed for chromosomal aberrations in chick fibroblasts treated with buffered ammonium chloride (Rosenfeld, 1932). Reduced cell division and inhibition of DNA repair were observed in mouse fibroblasts treated with ammonia and/or ammonium chloride (Visek et al., 1972; Capuco, 1977). Lobasov and Smirnov (1934) reported slightly mutagenic activity in *D. melanogaster*. Auerbach and Robson (1947) obtained doubtful, probably negative, results for sex-linked recessive mutations in *D. melanogaster* and reported negative results for dominant lethality.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC ORAL RfD VALUES FOR AMMONIA

Adequate toxicity data for derivation of provisional subchronic or chronic RfD values are not available. The human experimental database consists primarily of older acute and short-term studies in which ammonium chloride was used to induce metabolic acidosis. The longest duration of exposure among these studies was 18 days. The animal database includes one study (Gupta et al., 1979) that was reasonably well-documented, evaluated appropriate endpoints, and included a histopathological evaluation of potential target tissues. This study was not used to derive p-RfD values for two reasons. First, the test article was ammonium sulfamate, which is hydrolyzed under certain conditions to bisulfate ion and ammonia. It is not known whether hydrolysis occurred when the compound was administered to rats; thus, the actual dose of ammonia/ammonium ion administered to the test animals is uncertain. Second, comparison of the data from this study to results from human studies suggests that health effects may occur in humans at lower concentrations of ammonium salts. Gupta et al. (1979) identified a NOAEL equivalent to 37.3 mg ammonia/kg-day and a LOAEL equivalent to 74.8 mg ammonia/kg-day (assuming no hydrolysis of the sulfamate ion; the actual dose may differ). This LOAEL is higher than the 31.8 mg/kg-day level of concern identified for humans by U.S. EPA (1981) for potential bone demineralization. Route-to-route extrapolation is not feasible for derivation of oral reference values because the toxicokinetic properties of ammonia differ significantly for the oral and inhalation pathways. This evaluation of data adequacy is consistent with previous

assessments conducted by U.S. EPA (1981, 1987), which did not use the existing toxicity data for derivation of reference doses.

Because adequate data are lacking for oral exposure to ammonia, previous determinations of toxicity reference values (U.S. EPA, 1981, 1987, 1997) have used organoleptic (taste) data to estimate acceptable ammonium levels in drinking water at 34-35 mg/L. However, organoleptic (taste) data are not reliable predictors of either toxicity or intake. Furthermore, WHO (1986) has identified several limitations of the "triangle test" methodology used to derive the organoleptic (taste) threshold for ammonia: 1) the definition of the threshold is somewhat arbitrary; 2) McBride & Laing (1979) have reported significant positional bias in using the triangle test to determine taste threshold; and 3) the triangle test is not intended to mimic environmental exposures in which the taste thresholds could be substantially higher. Due to the high uncertainty associated with use of the organoleptic (taste) data for ammonia, no oral subchronic or chronic p-RfD is derived.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfC VALUES FOR AMMONIA

A **chronic RfC of 1E-1 mg/m³** is listed for ammonia on IRIS (U.S. EPA, 2003) based on lack of evidence of decreased pulmonary function in human workers exposed to an estimated concentration of 6.4 mg/m³ for an average of 12.2 years (Holness et al., 1989). The presence of a chronic RfC on IRIS precludes derivation of a provisional chronic RfC for this chemical.

The occupational study of Holness et al. (1989) and the subchronic study conducted in rats by Broderick et al. (1976) were also considered to be an appropriate basis for derivation of a provisional subchronic RfC. Holness et al. (1989) identified a NOAEL of 9.2 ppm (6.4 mg/m³) for apparent lack of effect on pulmonary function or changes in subjective assessments of symptoms in workers exposed to ammonia for a mean duration of 12.2 years while employed at a sodium carbonate production plant. A LOAEL was not identified in this study. The NOAEL_{HEC} was calculated using the default dosimetric adjustment for human data (U.S. EPA, 1994b), as follows:

$$\text{NOAEL}_{\text{ADJ}} = 6.4 \text{ mg/m}^3 \times 5\text{days}/7\text{days} = 4.6 \text{ mg/m}^3$$

$$\begin{aligned}\text{NOAEL}_{\text{HEC}} &= \text{NOAEL}_{\text{ADJ}} \times (\text{VE}_{\text{ho}}/\text{VE}_{\text{h}}) \\ &= 4.6 \text{ mg/m}^3 \times (10 \text{ m}^3/20 \text{ m}^3) \\ &= 2.3 \text{ mg/m}^3\end{aligned}$$

where,

VE_{ho} = human occupational default minute volume (10 m³/8 hours; U.S. EPA, 1994b)

VE_h = human ambient default minute volume (20 m³/24 hours; U.S. EPA, 1994b)

A $LOAEL_{HEC}$ was calculated from the rat data of Broderson et al. (1976) for comparison with the human $NOAEL_{HEC}$. These researchers identified a $LOAEL$ of 17.4 mg/m³, the lowest concentration tested, for increased severity of rhinitis and pneumonia (with respiratory lesions) in F344 rats inoculated with *M. pulmonis* and continuously exposed to ammonia. The $LOAEL_{HEC}$ is calculated using the procedure for a respiratory effect of a category 1 gas in the extrathoracic region (U.S. EPA, 1994b) as follows:

$$LOAEL_{ADJ} = LOAEL_{OBSERVED} = 17.4 \text{ mg/m}^3 \text{ (continuous exposure)}$$

$$LOAEL_{HEC} = LOAEL_{ADJ} \times RGDR_{ET}$$

$$\begin{aligned} RDGR_{ET} &= (V_E / SA_{ET})_A / (V_E / SA_{ET})_H \\ &= (0.14 \text{ m}^3/\text{day} / 15 \text{ cm}^2) / (20 \text{ m}^3/\text{day} / 200 \text{ cm}^2) = 0.093 \end{aligned}$$

$$\begin{aligned} LOAEL_{HEC} &= 17.4 \text{ mg/m}^3 \times 0.093 \\ &= 1.62 \text{ mg/m}^3 \approx 1.6 \text{ mg/m}^3 \end{aligned}$$

where:

$RDGR_{ET}$ = regional gas deposition ratio in the extrathoracic region

V_E = ventilation rate (m³/day)

SA_{ET} = surface area of extrathoracic region (cm²)

A, H = subscripts denoting laboratory animal and human, respectively

$(V_E)_A$ = 0.14 m³/day (subchronic, female F344 rats; U.S. EPA, 1988)

$(V_E)_H$ = 20 m³/day (U.S. EPA, 1988)

$(SA_{ET})_A$ = 15 cm² (U.S. EPA, 1994b)

$(SA_{ET})_H$ = 200 cm² (U.S. EPA, 1994b)

A **subchronic p-RfC of 0.1 mg/m³ (1E-1 mg/m³)** is derived by applying a composite uncertainty factor of 30 to the human $NOAEL$ of 2.3 mg/m³ (Holness et al., 1989). The composite UF includes a factor of 10 to protect sensitive individuals and a factor of 3 for proximity of the animal $LOAEL$ to the human $NOAEL$ and database limitations, including lack of adequate reproductive and developmental toxicity studies. The UF is applied to the human $NOAEL_{HEC}$ of 2.3 mg/m³, as follows:

$$\begin{aligned} \text{subchronic p-RfC} &= NOAEL_{HEC} / UF \\ &= 2.3 \text{ mg/m}^3 / 30 \\ &= 0.1 \text{ mg/m}^3 \text{ or } 1\text{E-1 mg/m}^3 \end{aligned}$$

The animal LOAEL of 1.62 mg/m³ is in close proximity to the human NOAEL of 2.3 mg/m³; however, the extrathoracic effects observed in the animal study were mild and reversible. Furthermore, the animal LOAEL_{HEC} of 1.6 mg/m³ gives a sixteen-fold comparative ceiling to the p-sRfC of 0.1 mg/m³. This adds confidence to the human NOAEL. Thus, the human NOAEL is considered for the derivation of this subchronic p-RfC. Thus the subchronic p-RfC, based on pharmacokinetics, remained the same as the RfC.

Confidence in the principal study is medium because the study was conducted in humans (but the sample size was relatively small), data were collected on males only, and a LOAEL was not identified. Although complaints of exacerbated upper respiratory symptoms were recorded in the principal study and support the extrathoracic region as the critical region for effects, an objective assessment of the workers' nasal epithelium was not performed. However, the observation of mild extrathoracic effects in animals at a HEC similar to the NOAEL support the human findings.

Confidence in the database is medium. The developmental, reproductive, and chronic toxicity of ammonia have not been tested, but toxicokinetic data suggest that ammonia is absorbed by the nasal passages at concentrations comparable to the NOAEL_{HEC} and systemic distribution is unlikely (U.S. EPA, 2003). Medium confidence in the subchronic p-RfC follows.

DERIVATION OF A PROVISIONAL CARCINOGENICITY ASSESSMENT FOR AMMONIA

Human data on the carcinogenic effects of ammonia or ammonia compounds are not available. Among animals, no evidence for carcinogenicity was observed in two strains of mice administered ammonium hydroxide in drinking water for two years or in a urethane-sensitive strain of mice administered ammonia in water by gavage for 4 weeks. There is some indication that ammonia contributes to the development of cancer when coadministered with DEPC (via formation of urethane) or MNNG (via stimulation of cell proliferation in the gastric mucosa). Limited genotoxicity testing of ammonia has produced mixed results. Under the proposed guidelines (U.S. EPA, 1999), the data for carcinogenicity of ammonia *are inadequate for an assessment of human carcinogenic potential*.

Derivation of quantitative estimates of cancer risk for ammonia is precluded by the absence of data indicating a carcinogenic effect for this chemical.

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Provisional Peer Reviewed Toxicity Values

Benz[a]anthracene
(CASRN 56-55-3)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards

NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES
FOR BENZ[a]ANTHRACENE (CASRN 56-55-3)**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

This document has passed the STSC quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

INTRODUCTION

IRIS (U.S. EPA 1990b) reports that an RfD for benz[a]anthracene is not available at this time. Neither the HEAST (U.S. EPA, 1997) nor the Drinking Water Regulations and Health Advisory list (U.S. EPA, 2000) report an RfD for benz[a]anthracene. ATSDR (2000) has not published a Toxicological Profile for benz[a]anthracene, though a discussion of benz[a]anthracene is included in the profile for polycyclic aromatic hydrocarbons (PAH) (ATSDR, 1995). No oral MRLs were derived for benz[a]anthracene. IARC (1973, 1983) monographs on benz[a]anthracene and the NTP status report (NTP, 2000) were consulted for relevant information. The World Health Organization (WHO, 2000) has not published an Environmental Health Criteria document for benz[a]anthracene.

The CARA lists (U.S. EPA, 1991, 1994b) report no relevant documents specific for benz[a]anthracene. A Drinking Water Criteria Document (U.S. EPA, 1990a) for PAH exists, but an RfD for benz[a]anthracene was not derived.

Literature searches were conducted from 1989 to June, 2000 for studies relevant to the derivation of an RfD. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, RTECS, GENETOX, HSDB, CCRIS, TSCATS, EMIC/EMICBACK, and DART/ETICBACK.

A carcinogenicity assessment for benz[a]anthracene is available on IRIS (U.S. EPA, 1990b). This assessment, verified 02/07/1990, was based on a Carcinogen Assessment of Coke Oven Emissions (U.S. EPA, 1984a) and a Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs) (U.S. EPA, 1990). Benz[a]anthracene was assigned to weight-of-evidence Group B2, probable human carcinogen, based on increased incidences of pulmonary and hepatic tumors in mice exposed by gavage (Klein, 1963) or intraperitoneal injection (Wislocki et al., 1986), positive results in tests for complete carcinogenicity and initiating activity in skin painting assays in mice (multiple studies reviewed by IARC, 1973), and injection site sarcomas in mice injected subcutaneously (Steiner and Edgecomb, 1952; Steiner and Falk, 1951). Supporting data from genotoxicity tests included positive results for mutations in bacteria and mammalian cells, and transformed mammalian cells in culture. It was noted that benz[a]anthracene is a component of mixtures that are known to produce cancer in humans, although there are no human data that specifically link benz[a]anthracene with human cancers. However, due to the lack of adequate oral data for benz[a]anthracene, an oral slope factor was not included on IRIS (U.S. EPA, 1990b).

The HEAST (U.S. EPA, 1997) reports the availability of the weight-of-evidence assessment on IRIS, but contains no additional information. The Drinking Water Standards and Health Advisories list (U.S. EPA, 2000) includes the cancer group B2 designation for benz[a]anthracene, but does not include additional cancer risk information. A Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs) (U.S. EPA, 1984b) was located, but no relevant documents specific to benz[a]anthracene were found in the CARA database (U.S. EPA, 1991, 1994b).

The International Agency for Research on Cancer (IARC, 1973, 1983, 1987) evaluated benz[a]anthracene for carcinogenicity and placed the chemical in Group 2A (probable human carcinogen), finding that there is sufficient evidence that benz[a]anthracene is carcinogenic to experimental animals and that the chemical is active in short-term genotoxicity tests. CalEPA derived an oral slope factor for benz[a]anthracene which is based on a relative potency factor approach (CalEPA, 1999). The ATSDR (1995) Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs) and the NTP (2000) management status report were searched for relevant information. Updated literature searches for cancer data were conducted from 1989 to 2000. The databases searched were TOXLINE, MEDLINE, CANCERLIT, CCRIS, TSCATS, HSDB, RTECS, GENETOX, DART/ETICBACK, and EMIC/EMICBACK.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No studies were located regarding oral exposure of humans to benz[a]anthracene.

No studies were located regarding the carcinogenicity of benz[a]anthracene in humans following oral exposure.

Animal Studies

No oral studies in animals suitable for derivation of an RfD were located. The majority of available studies examined mixtures of PAHs containing benz[a]anthracene, rather than the pure compound. A study by Klein (1963) examined the carcinogenic effects of benz[a]anthracene in mice following gavage exposure (3 exposures/week, ~1.5 mg/exposure in 0.05 mL volume, for durations between 344 and 600 days). However, the study examined only one exposure level and only reported tumor incidence; noncancer endpoints were not evaluated.

Klein (1963) observed increased incidence of pulmonary adenoma and hepatoma in male mice treated with 3% benz[a]anthracene solution by gavage for 5 weeks. This study is not suitable for quantitative cancer risk assessment due to the short exposure duration and use of a single dose level (U.S. EPA, 1990a). No other studies were located that could be used as the basis for derivation of an oral slope factor for benz[a]anthracene.

Other Studies

A number of genotoxicity studies (reviewed by ATSDR, 1995; IARC, 1973; U.S. EPA, 1984b, 1990a) indicate that benz[a]anthracene is genotoxic to bacteria and mammalian cells.

DERIVATION OF A PROVISIONAL RfD FOR BENZ[a]ANTHRACENE

A provisional RfD for benz[a]anthracene cannot be derived due to the lack of suitable human and animal data.

DERIVATION OF A PROVISIONAL RfC FOR BENZ[a]ANTHRACENE

A provisional RfC for benz[a]anthracene cannot be derived due to the lack of suitable human and animal data.

DERIVATION OF A PROVISIONAL ORAL SLOPE FACTOR FOR BENZ[a]ANTHRACENE

A provisional oral slope factor for benz[a]anthracene cannot be derived because human data are lacking and the oral cancer data in animals are inadequate. However, the Appendix to this document contains a screening value that may be useful in certain instances. Please see the attached Appendix for details. A provisional unit risk is not developed because of lack of available data.

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APPENDIX

DERIVATION OF A SCREENINGVALUE FOR BENZ[a]ANTHRACENE

For reasons noted in the main PPRTV document, it is inappropriate to derive provisional toxicity values for benz[a]anthracene, oral slope factor. However, information is available for this chemical which, although insufficient to support derivation of a provisional toxicity value, under current guidelines, may be of limited use to risk assessors. In such cases, the Superfund Health Risk Technical Support Center summarizes available information in an Appendix and develops a "Screening Value." Appendices receive the same level of internal and external scientific peer review as the PPRTV documents to ensure their appropriateness within the limitations detailed in the document. In the OSRTI hierarchy, Screening Values are considered to be below Tier 3, "Other (Peer-Reviewed) Toxicity Values."

Screening Values are intended for use in limited circumstances when no Tier 1, 2, or 3 values are available. Screening Values may be used, for example, to rank relative risks of individual chemicals present at a site to determine if the risk developed from the associated exposure at the specific site is likely to be a significant concern in the overall cleanup decision. Screening Values are not defensible as the primary drivers in making cleanup decisions because they are based on limited information. Questions or concerns about the appropriate use of Screening Values should be directed to the Superfund Health Risk Technical Support Center.

In this appendix, we briefly examine the Agency's development of a quantitative cancer dose-response analysis for benz(a)anthracene (B[a]A), a polycyclic aromatic hydrocarbon (PAH). In 1993, the U.S. EPA developed an estimated order of potency value of 0.1 for B[a]A, relative to the carcinogenicity of a second PAH, benzo(a)pyrene (B[a]P). We then examine the development of this estimated order of potency value considering the relative potency factor method developed in the U.S. EPA's Supplementary Chemical Mixtures Guidance (U.S. EPA, 2000). We conclude that there is uncertainty in applying an RPF value, which was developed in a rodent bioassay, to an oral slope factor. We identify uncertainties regarding the application of this value in risk assessments. The discussion focuses on animal bioassay data rather than *in vitro* methods, because we do not know whether such studies provide relevant measures of relative potency for humans. We also do not discuss studies that have compared potencies for non-cancer effects because the IRIS database does not quantify an oral RfD for B[a]P; other sources of variability associated with the development of RfD estimates complicate the application of relative potency approaches.

In 1992, the U.S. EPA published the 'Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (U.S. EPA, 1992), which details the development of an oral cancer slope factor (OSF) for a PAH, B[a]P. This document also classified seven other PAHs including B[a]A as probable human carcinogens, but, citing limited dose-response information, did not develop cancer slope factor estimates for these seven PAHs. In 1993, the Agency published the 'Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons' (U.S. EPA, 1993), which describes the development of an approach for quantifying the cancer risk associated with these seven PAHs by comparing the relative carcinogenic potency of each

compound to that of B[a]P. The Agency's approach was mathematically equivalent to the toxicity equivalence factor (TEF) approach (U.S. EPA, 1989). The TEF approach as applied to dioxins assumed that a single TEF value could be developed for each dioxin congener and that this same value could be used for different health endpoints, different routes of exposure and different durations of exposure. However, the underlying scientific data for B[a]A and B[a]P did not satisfy all of the criteria recommended for implementing the TEF method (Barnes, et al., 1991). The Agency acknowledged that this approach did not meet all of the criteria for TEF development and coined an alternative term, "estimated order of potency" to distinguish this approach from TEFs citing the following additional reasons:

- approach applied to a small subset of the PAHs, instead of all PAHs
- approach limited to the cancer endpoint, instead of all health endpoints
- slope factor derivation based on B[a]P exposure only from an oral pathway, instead of deriving this value based on multiple exposure routes
- uncertainty about such an application given the current understanding of the toxicodynamics associated with PAH carcinogenicity

The Agency approach assumed that the human carcinogenicity of the seven PAHs could be predicted using an oral cancer slope factor that was developed for B[a]P. To analyze the carcinogenicity of B[a]A relative to B[a]P, the Agency utilized the results of a chronic mouse dermal bioassay reported by Bingham and Falk (1969), which relied on the bioassay methods published by Horton et al. (1965). In the bioassay, groups of mice were treated with either B[a]P or B[a]A. B[a]P or B[a]A was applied to an area of shaved skin on the back of each mouse twice weekly until the animal developed a tumor or died. We note that Bingham and Falk (1969) did not report solvent control tumor incidences.

The ability of B[a]A to elicit rodent skin tumors then was quantitatively compared to that of B[a]P (Equation 1). U.S. EPA (1992) describes a potency analysis by T. Thorslund of ICF-Clement Associates under contract with U.S. EPA. In the application of these models it was assumed that carcinomas can develop from papillomas. The relative potency of each PAH was calculated as the ratio of the estimated times-to-tumor with the potency of BAP indexed as 1. Point estimates (maximum likelihood estimates) were compared rather than upper bound estimates. Based on this approach the U.S. EPA (1993) recommended an "estimated order of potency" value for B[a]A of 0.1, relative to B[a]P. This was described as an interim recommendation. Time-to-tumor analyses rely on measures of response time and dose. Consequently, this integrated measure of response time is an imperfect measure upon which to base an estimate of the relative potency of one chemical to another.

$$RPF = \frac{TT_{B[A]P}}{TT_{B[A]A}} \quad \text{Equation 1}$$

Where:

RPF Relative Potency Factor (unitless)

TT Time-to-tumor (days)

B[A]A Benz[a]Anthracene

B[A]p Benz[a]Pyrene

U.S. EPA (1993) also analyzes the relative potency of B[a]A based on several other bioassays. Based on an intraperitoneal injection study (Wislocki et al. 1986), US EPA (1993) reported a range of "estimated order of potency" values of 0.06-0.52 for B[a]A. Wislocki et al. (1986) administered B[a]P and B[a]A intraperitoneally to newborn CD-1 mice on postnatal days 1, 8, and 15 and the mice were sacrificed after 1 year. The range of potency values was calculated using liver and lung tumor incidence data. The relevance of this exposure route to environmental exposures is questionable and the applicability of relative potency comparisons to such exposures is not known. The US EPA (1993) also discusses a manuscript by Nisbet and Lagoy, which recommends a relative potency value of 0.1 for B[a]A based on comparisons of tumorigenic potencies with B[a]P (essentially the same as those reviewed by Clement Associates, 1988). The RPFs were derived from previously reported review papers (Nisbet and LaGoy, 1992; Rugen et al., 1989; Clement Associates, 1988; Chu and Chen, 1984), as well as the primary literature describing pulmonary implant, skin painting, subcutaneous injection, and mouse skin DNA binding studies. The relative potency values of B[a]A were comparable across multiple testing modalities.

In 2000, the Agency published the 'Supplementary Guidance for Chemical Mixtures,' which describes the relative potency factor method. Similar to the TEFs and estimated order of potency methods, this method is based on the concept of dose addition. The fundamental assumption of dose additive mixture methods is that the components' toxicity is mediated through the same toxic mode of action; then, the toxicity of mixtures consisting of components that act through a common mode of toxic action can be predicted by the component compounds' toxicity. The toxicity of the marginally studied components of the mixture can be estimated by scaling to the toxicity of a well-studied component of the chemical mixture (referred to as the index chemical). To implement this approach, the index chemical must have adequate toxicologic dose-response data for relevant routes of exposure. The toxicity of each of the other components of the mixture is predicted by scaling its exposure level by its toxicity relative to the index chemical. This scaling factor, called the Relative Potency Factor (RPF), is based on a comparison of the results of toxicologic assays with those results for the index chemical. The product of the measured exposure concentration of each mixture component and its RPF is considered to be an equivalent dose in units of the index chemical (i.e., dose of Chemical I \times RPF_I = Index Chemical Equivalent Dose of Chemical I). The index chemical exposure equivalents of all the mixture components are summed to express the total mixture exposure in terms of an equivalent exposure to the index chemical. The risk posed by the mixture is quantified by comparing the mixture's index chemical equivalent dose to the dose-response function of the index chemical. A key advantage of this mixture component method is that, based on the available data, the application of an RPF can be limited to specific toxicologic effects, exposure routes, exposure durations, or dose ranges. The EPA stated that RPF applications that have no such limitations are called toxicity equivalence factors (TEFs).

Based on this application of the RPF method and the OSF listed on IRIS for B[a]P, 7.3 per mg/kg-day, we provide a **screening oral slope factor of 0.7 per mg/kg-day (0.1 x 7.3)**. Screening level values should be used only for screening and after consultation with the Superfund Health Risk Assessment Center.

The following should be considered in the application of the B[a]A RPF value to the OSF for B[a]P.

- The B[a]A RPF value of 0.1 was developed using chronic exposure data, applications to other exposure durations should include analyses of toxicokinetics and toxicodynamics.
- The B[a]A RPF value of 0.1 was developed using cancer data, applications to other toxicity endpoints should include analyses of toxicokinetics and toxicodynamics.
- The B[a]A RPF value of 0.1 was developed based on a small number of exposure routes, applications to other routes of exposure should compare toxicokinetics and toxicodynamics of B[a]A across relevant routes of exposure.
- Use of the maximum likelihood estimates to develop the RPF value (U.S. EPA, 1992) of 0.1 for B[a]A is appropriate

A number of additional uncertainties are identified:

- The Bingham and Falk study does not report a vehicle control. The lack of such a control increases the uncertainty in the quantitative interpretation of the results.
- The limited review of the toxicological literature on B[a]A failed to identify data that characterize whether the carcinogenicity of B[a]A and B[a]P results from a common toxic mode of action. Evidence presented here is based on gross observations of benign and malignant tumor development in rodents. Additional toxicodynamic analyses are needed to characterize whether these compounds share a common toxic mode of action in rodents. Information is also needed to determine if this mode of action is relevant to humans. We emphasize that this assumption of common toxic mode of action is *critical* to application of a dose additive method such as the RPF method and that the molecular evidence supporting this assumption for carcinogenicity of B[a]A and B[a]P is not evaluated here.
- The RPF value for B[a]A is based primarily on a point of contact exposure with shaved dermis. Although other similar RPF values have been derived based on other exposure pathways (U.S. EPA, 1993), the applicability of the exposure routes used in these studies to environmentally relevant RPFs estimates is not known. The OSF for B[a]P is based on the increased incidences of squamous cell papillomas and carcinomas in the forestomachs of mice, rats and hamsters administered BAP via the diet or by gavage (Neal and Rigdon, 1967; Knauf and Rice, 1992). These were likely point of contact tumors. Additional data are needed to characterize the absorption, distribution, metabolism and elimination of B[a]A; such data need to be compared with similar studies in B[a]P as well as toxicodynamic studies to assess the uncertainty in cross-route applications of this RPF. Again, in addition to such studies in rodents, an evaluation of the kinetics of both compounds in humans would be useful in the evaluation of the RPF value to other routes of exposure.
- Applications of this RPF value for B[a]A to other types of risks (e.g., non-cancer) should be considered carefully. In this analysis, we did not survey the literature to evaluate whether

data are available to evaluate the relative potency of B[a]A to B[a]P for other health endpoints.

- Additional empirical data are needed on the additivity of carcinogenic effects of PAHs. Results of testing simple mixtures of PAHs and mixture components must be compared to assessments made from bioassays of complex PAH environmental mixtures. The conduct of such studies, while not critical to the development of this RPF value, could improve the overall confidence in the results of cancer risk estimates derived from dose-additive models of PAH carcinogenicity.

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May 9, 2008

Michael Sivak
U.S. EPA, Region 2

ASSISTANCE REQUESTED: PPRTVs for Bis(2-chloroethoxy)methane, Chloroethane, Cobalt and Chlorobenzene (*Onondaga Lake*)

ENCLOSED INFORMATION:

- Attachment 1: **PROVISIONAL TOXICITY VALUES FOR BIS(2-CHLOROETHOXY)METHANE (CASRN 111-91-1)**
- Attachment 2: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR CHLOROETHANE (CASRN 75-00-3)**
- Attachment 3: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR CHLOROBENZENE (CASRN 108-90-7)**
- Attachment 4: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR COBALT AND COMPOUNDS (CASRN 7440-48-4) Derivation of Subchronic and Chronic Oral RfDs**

Attachment 5: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR COBALT AND COMPOUNDS
(CASRN 7440-48-4) Derivation of Subchronic and
Chronic Inhalation RfCs**

Attachment 6: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR COBALT AND COMPOUNDS (CAS
NO. 7440-48-4) Derivation of a Carcinogenicity
Assessment**

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (6)

cc: STSC Files

9-22-2006

Provisional Peer Reviewed Toxicity Values for
Bis(2-chloroethoxy)methane
(CASRN 111-91-1)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL TOXICITY VALUES FOR
BIS(2-CHLOROETHOXY)METHANE (CASRN 111-91-1)**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Neither subchronic nor chronic RfDs or RfCs for bis(2-chloroethoxy)methane (BCM) are available on IRIS (U.S. EPA, 2006a), the HEAST (U.S. EPA, 1997) or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). A carcinogenicity assessment for bis(2-chloroethoxy)methane is available on IRIS (U.S. EPA, 2006a) that includes a classification of Group D, not classifiable as to human carcinogenicity, based on no human or animal data. The CARA list (U.S. EPA, 1991a, 1994) includes no documents for this chemical. The toxicity of bis(2-chloroethoxy)methane has not been reviewed by ATSDR (2006), IARC (2006), or WHO (2006). ACGIH (2006), NIOSH (2006) and OSHA (2006) have not established occupational exposure limits for this compound. The NTP (2006) Management Status Report provided no relevant information. A technical report on haloethers prepared for EPA in 1975 (Durkin et al., 1975) and the Ambient Water Quality Criteria Document for Chloroalkyl Ethers (U.S. EPA, 1980) were reviewed for pertinent information. Literature searches were conducted from 1965 to September 2002 in TOXLINE, CANCERLIT, MEDLINE, GENETOX, HSDB, EMIC/EMICBACK, DART/ETICBACK, RTECS and TSCATS for relevant studies. During April 2004, these databases were again searched for relevant studies; none were identified that would change the conclusions of the risk estimate.

REVIEW OF PERTINENT DATA

Bis (2-chloroethoxy)methane is a synthetic organic chemical used as a solvent and as a reactant in the manufacture of polysulfide elastomers. More than 95% of polysulfide elastomers are made from bis (2-chloroethoxy) methane starting material and sodium polysulfide. The resulting products are used as heat- and solvent resistant sealants. The U.S. Food and Drug Administration has approved bis(2-chloroethoxy)methane for use in the manufacture of resins approved for direct contact with food packaging materials. Bis (2-chloroethoxy)methane is on the U.S. EPA's 1990 High Production Volume chemical list (U.S. EPA, 2006b); in 1977, 10 to 50 million pounds of bis(2-chloroethoxy)methane were produced in the U.S. (HSDB, 2005).

Human Studies

Oral Exposure. No reports were located regarding the subchronic or chronic toxicity or carcinogenicity of bis(2-chloroethoxy)methane in humans by oral exposure.

Inhalation Exposure. No reports were located regarding the subchronic or chronic toxicity or carcinogenicity of bis(2-chloroethoxy)methane in humans by inhalation exposure.

Animal Studies

Oral Exposure. Non-fasted Sprague-Dawley rats (10/sex/dose group) were treated with oral doses of 0, 10, 20, 40, 80, or 120 mg/kg-day of bis(2-chloroethoxy)methane by daily gavage in corn oil for 90 days (Bio/Dynamics, 1990a). Physical observations, body weight and food consumption measurements were recorded weekly. Hematology and clinical chemistry evaluations were performed after one month of treatment and at termination. Ophthalmoscopic examinations took place at termination. Complete gross post-mortem examination was conducted on all animals. The control and high-dose groups received comprehensive histopathological examinations, while only the kidneys, liver, lungs and gross lesions were examined in the intermediate dose groups.

Of the 20 rats that received the highest dose (120 mg/kg-day), all ten males and seven of ten females died or were killed in moribund condition prior to completion of the study (Bio/Dynamics, 1990a). One death occurred after a single dose and seven more occurred during the first week. Subsequent deaths occurred as late as day 76 of the study. These deaths were considered by the researchers to be chemical-related; myocardial degeneration seen by microscopic examination in all 120 mg/kg-day rats that died after day 14 of the study was considered by the researchers to be a possible cause of death. One female in the 80 mg/kg-day dose group died on day 78; a death that was also considered to be chemical-related by the investigators in part because microscopic examination revealed myocardial degeneration similar to that seen in the 120 mg/kg-day animals that died. One female in the 40 mg/kg-day group died due to gavage error, but no chemical-related deaths were observed in the 10, 20 or 40 mg/kg-day dose groups. Rats killed in moribund condition and some of those that died exhibited emaciation, poor food consumption, hypothermia, lethargy/prostration, dyspnea, gasping, moist

rales, ataxia, abnormal posture, slight tremors, salivation, and brown-yellow stains on the snout, paws, ventral surface and anogenital area. Clinical signs were unremarkable in rats that survived the experiment. In male rats treated with the highest dose of bis(2-chloroethoxy)methane, body weight was significantly reduced by 17-18 % after weeks 1 (n=6 survivors) and 2 (n=4 survivors), and 7 to 21% thereafter (n=1 or 2 survivors). Mean body weights of males receiving 80 mg/kg-day were slightly lower than control during the second two months of the study: 6% deficit at week 5 and 10% deficit at week 12 (differences from control not statistically significant). Mean body weights for males in the lower dose groups were similar to controls, and no effect on body weight was evident in females at any dose level. Food consumption was reduced in the high-dose male group during the first 2 weeks of the study, but was similar to controls subsequently in the 2 survivors of this group. Food consumption was similar to or higher than controls in all other test groups. Ophthalmological examinations were unremarkable.

No statistically significant changes in hematological parameters were observed (Bio/Dynamics, 1990a). Exposure to 120 mg/kg-day induced statistically significant alterations in several clinical chemistry parameters in both males and females. The alterations that were considered to be indications of an effect of exposure to 120 mg/kg-day of bis(2-chloroethoxy)methane were: 1) slight elevations in serum aspartate aminotransferase (AST) at one month in both males and females, with a marked, statistically significant elevation in AST among high-dose females at study termination (no high-dose males survived to study termination), 2) a statistically significant elevation in serum alkaline phosphatase in males at one month and nonsignificant elevations in females at both one and three months, and 3) increased blood urea nitrogen (BUN) in females at 1 month (nonsignificant) and 3 months (statistically significant). In the 80 mg/kg-day group, there was a slight, statistically significant increase in serum alanine aminotransferase (ALT) among male rats at 3 months. No changes in clinical chemistry parameters were observed among male or female rats receiving 10, 20, or 40 mg/kg-day of bis(2-chloroethoxy)methane compared to controls.

Absolute and relative liver weights were statistically significantly increased in a dose-related fashion in female rats treated with 80 or 120 mg/kg-day (Bio/Dynamics, 1990a). Liver weight measurements were not available for males in the 120 mg/kg-day dose group due to early mortality; the only statistically significant change in males was a small increase in relative liver weight at 40 mg/kg-day. Histopathologic examination of the liver revealed a dose-related increased incidence of minimal-to-slight hypertrophy of the centrilobular hepatocytes in males treated with 20, 40, or 80 mg/kg-day (0/10, 0/10, 3/10, 4/10, 6/10, and 0/10 in the 0, 10, 20, 40, 80, and 120 mg/kg-day groups, respectively). The difference from controls was statistically significant in the 40 and 80 mg/kg-day groups (Fisher exact test conducted for this assessment). The lesion was not observed in males of the 120 mg/kg-day group, but rats in this group all died early. Liver lesions were not found in female rats. Mean adrenal weights (absolute and relative) were reduced relative to control among male rats receiving 20, 40, or 80 mg/kg-day. This effect on adrenal weight, however, was not observed among females and adrenal morphology was normal; thus, the toxicological significance of this effect on the adrenal gland is uncertain. Significant increases in relative kidney and testes weights in male rats at 80 mg/kg-day were considered by the researchers to be secondary to reduced body weight in this group. Kidney lesions, seen only in male rats, were increased incidences of minimal to moderate tubular nephrosis, accompanied in some cases by birefringent intracytoplasmic inclusions in the

convoluted tubular epithelium, and hyaline droplets in the epithelial cytoplasm of the proximal convoluted tubules. The incidence and severity of the renal lesions increased with dose, with the 10 mg/kg-day group being similar to controls and the 80 mg/kg-day group showing the most pronounced effects.

Other organs affected by the 120 mg/kg-day dose were the heart (myocardial degeneration), brain and spinal cord (vacuolization, gliosis), spleen, bone marrow, and thymus (atrophy, hypocellularity), and epididymides (oligospermia, degenerated seminal product); however, these organs were not systematically examined in rats receiving lower doses (Bio/Dynamics, 1990a). Of particular interest is the heart. Postmortem examination revealed slight-to-moderate degeneration of the myocardium in all high-dose animals that died after 2 weeks of exposure to bis(2-chloroethoxy)methane. The overall incidence of myocardial degeneration was 6/10 males and 6/10 females at 120 mg/kg-day (versus 0/10 for controls of each sex). The authors speculated that myocardial degeneration was a possible cause of death. Despite the prevalence of this effect among high-dose rats of both sexes, and absence among controls, the authors did not conduct histopathological examinations of the hearts of rats receiving lower doses, aside from one female from the 80 mg/kg-day group (the female that was found dead on day 78) and one female from the 40 mg/kg-day group that died accidentally in week 5. Histological examination revealed myocardial degeneration in the 80 mg/kg-day female, but not the 40 mg/kg-day female.

The renal effects seen in male rats are consistent with the pattern of early stages of alpha_{2u} globulin-associated rat nephrotoxicity, as established by the Risk Assessment Forum (U.S. EPA, 1991b), wherein the Agency concluded these renal effects are not appropriate as a critical effect for human health risk assessment. This study identified a LOAEL of 20 mg/kg-day based on liver lesions (hypertrophy of the centrilobular hepatocytes) in male rats and a NOAEL of 10 mg/kg-day following subchronic oral administration of bis(2-chloroethoxy)methane.

More recently, the general toxicity of BCM was evaluated in mice (Battelle, 2002a) and rats (Battelle, 2002b) exposed to BCM (in 95% ethanol) dermally for 5 days per week for 90 days. Applied doses for rats and mice were 0, 50, 100, 200, 400 and 600 mg/kg. Duration adjusted doses were 0, 36, 71, 143, 286 and 429 mg/kg. Available reports do not indicate whether the dose site was occluded. For all rats, the 600 mg/kg dose was lethal, and observations consistent with heart failure were noted in some rats in the 400 and 600 mg/kg dose groups. BCM was lethal in two of 10 female rats receiving 400 mg/kg. Selected organs were histologically examined at sacrifice. Hematology and clinical chemistries were not altered. Histopathic cardiomyopathy was considered the most toxicologically significant finding, and a dose-dependent increase in severity was noted in the 400 and 600 mg/kg dose groups. In male rats, histologic alterations were noted in the glandular stomach, mesenteric lymph nodes, spleen, thymus, Harderian gland and olfactory epithelium, but only in high dose animals. Findings in female rats differed only in that spleen, Harderian gland and olfactory epithelium were affected at 400 mg/kg and renal tubular (cortex) damage was noted in high dose females.

In mice, Battelle (2002b) reported no findings of lethality in males, but BCM was lethal to 3/10 female mice receiving 600 mg/kg. Erythrocyte-related parameters (RBC, hemoglobin, hematocrit) were significantly reduced in male mice at and above 200 mg/kg and both absolute

and relative kidney weights were increased at 400 and 600 mg/kg. In female mice, absolute liver weight was increased and myocardial vacuolization were observed at 400 mg/kg. At 600 mg/kg, additional findings included histopathic alterations in heart and liver, erosion and inflammation of the stomach and duodenum, and reductions in erythrocyte parameters. Dunnick et al (2004a) also reported the results from this study and noted an increased (2/10) incidence of myocyte cytoplasmic vacuolization in female rats exposed to 200 mg/kg, with incidences of 5/10 and 8/10 in the two higher doses, respectively.

From these studies, a dermally applied, duration adjusted LOAEL of 71 mg/kg-day is indicated for decreased hemoglobin content in male mice and increased incidence of myocyte cytoplasmic vacuolization in female rats. Correspondingly, the NOAEL values would be 36 mg/kg-day. Special considerations and information must be available to translate this dermally applied dose to a corresponding internal dose. Some pertinent information describe the distribution and elimination of ¹⁴C from a ¹⁴C-labelled BCM dermal administration study (Mathews and Jeffcoat, 2002). In those studies, BCM was dermally applied. *Ex vivo* studies with excised skin demonstrated a loss of 85% of the applied dose within one hour of application. Absent a capacity of absorption and removal from the site, these results indicate that up to 85% of the administered dose may be lost to volatilization within the first hour of application. Results from dermal studies in rats exposed to 10 and 0.1 mg/kg with and without dose site appliances (covers) demonstrated that dermal absorption resulted in a total absorbed dose of approximately 15% of the administered dose with appliances and approximately 40 to 44% of applied dose without appliance, seemingly indicative of additional ingestion via grooming (Mathews and Jeffcoat, 2002). In mice with the dermal appliance, these samples accounted for approximately 9 and 18% of a dermally applied dose of 0.1 or 10 mg/kg, with dose site accounting for approximately 1% of the administered dose. Mice administered BCM without the site-protective appliance absorbed 13 and 21% of applied doses of 0.1 and 10 mg/kg, respectively. The pattern of tissue distribution, extent of urinary elimination and other pharmacokinetic information, demonstrated for total radiolabel derived from ¹⁴C-labelled BCM, demonstrate appreciable similarity between dermal and oral exposures. While these data indicate dermal absorption, potential and undescribed differences in the metabolism of orally and dermally exposed animals exist and complicate the development of a dermal correction factor, especially so in light of studies that seem to indicate thioglycolic acid as the potentially bioactive (toxic) metabolite (Mathews and Jeffcoat, 2002). This metabolite is common to other cardiotoxic compounds, as well. Without further adjustment, the dermally applied, duration adjusted NOAEL values indicated by Battelle (2002a,b) and quantified by Dunnick et al (2004a) are higher than NOAEL value (10 mg/kg-day) for liver lesions developed from orally administration studies (Bio/Dynamics, 1990a).

In a range-finding study for the oral subchronic study (Bio/Dynamics, 1990a), non-fasted Sprague-Dawley rats (5/sex/dose group) were treated with 0, 20, 40, 50, 60, 80, or 100 mg/kg-day of bis(2-chloroethoxy)methane by daily gavage in corn oil for two weeks (Bio/Dynamics, 1990b). When no signs of toxicity were noted after one week of dosing, the 20 and 40 mg/kg-day doses were increased to 150 and 200 mg/kg-day, respectively, for the second week of treatment and satellite groups of 5 rats/sex/group were started on doses of 120 or 160 mg/kg-day. Animals were observed twice daily for mortality and gross toxicity. Physical examinations and body weight and food consumption measurements were performed weekly. Blood was collected

from all rats surviving to study termination for hematology and clinical chemistry evaluations. Complete gross postmortem examinations were performed on all animals. The brain, heart, liver, kidneys, adrenals, and gonads were weighed for animals killed at terminal sacrifice. Histopathology was not performed.

Doses of 120 mg/kg-day and above clearly produced treatment-related mortality (7/10-10/10 dead after 1-9 doses) (Bio/Dynamics, 1990b). The only deaths in the lower dose groups were single deaths in the 60 and 80 mg/kg-day groups (1/5 females and 0/5 males died in each group after 16 doses) that may also have been due to treatment. Findings in rats that died or were sacrificed moribund included clinical signs (lethargy, tremor, dyspnea, irregular gait, yellow or brown staining of the anogenital area, salivation, moist rales, hypothermia, and general poor condition in some rats just prior to death), antemortem weight loss, hematological changes (increased hemoglobin, hematocrit, and red blood cell count in males, but not females), and serum chemistry changes (increases in serum markers for hepatotoxicity and nephrotoxicity, including ALT, AST, alkaline phosphatase, BUN, and glucose). Due to the high mortality in the ≥ 120 mg/kg-day dose groups, meaningful comparisons based on group means were not possible for these groups. Among the 20-100 mg/kg-day groups, there were no significant differences from controls for food intake or body weight, and no clinical signs were observed. The only significant hematology finding was an increase in red blood cell count in females, but not males, at 100 mg/kg-day. Blood urea nitrogen was significantly increased in the 50, 80, and 100 mg/kg-day female groups, and non-significantly increased in the 60 mg/kg-day female group. The magnitude of the change from controls was small for this parameter ($\approx 20\%$) and did not increase with dose. No other serum chemistry changes were seen in females or males. Absolute and relative liver weights were significantly increased in females in the 80 and 100 mg/kg-day groups (by 21-27%, a moderate change for this parameter). No other significant organ weight changes were found. Gross postmortem examination revealed no abnormalities attributable to bis(2-chloroethoxy)methane. The results of this study support the finding of the subchronic study that the liver is an important target for bis(2-chloroethoxy)methane.

In a short term study to characterize and examine the short-term time course of BCM-induced cardiotoxicity, rats were exposed dermally for up to 12 days to 400 and 600 mg/kg BCM in 95% ethanol (Dunnick et al, 2004b). Within two days of exposure to 600 mg/kg, most but not all cardiac myocytes examined showed toxic effects. Mitochondrial alterations were the most prominent, but other alterations included distention of the sarcoplasmic reticulum, myofibrillary degeneration and occasional Z-banding misalignments. Severe disintegration of mitochondria and the presence of megamitochondria were observed. Swelling of the sarcoplasmic reticulum was presented as a sign of cellular injury due to loss of membrane function in maintaining water balance. The authors noted in animals surviving to day 16 a "resolution of the manifestations of the lesions".

Inhalation Exposure. No reports were located regarding the subchronic or chronic toxicity of bis(2-chloroethoxy)methane in animals by inhalation exposure.

Other Studies

Toxicokinetics. The disposition of BCM was investigated in rats and mice by Research Triangle Institute (RTI) under contract to NIEHS (Mathews and Jeffcoat, 2002). In that study, male and female F-344 rats and male and female B6C3F1 mice received 14-C-labeled BCM via the oral, intravenous (i.v.) and dermal routes. BCM appeared poorly absorbed via dermal application, potentially due to volatility, and so will not be further presented here. Initial 72-hr studies characterized the tissue distribution and elimination of a 10 mg/kg gavage (water vehicle) dose of BCM. Parent BCM and 14C-CO₂ were quantified in expired air, and total 14C was quantified in urine, feces and tissues from male and female mice and male rats. The routes, rates and extent of elimination appeared similar in male and female mice, with combined urinary and fecal elimination accounting for 60-74% of the dose at 14 hours and with urine accounting for 50-60% and approximately 25% of the dose excreted in urine and feces, respectively, at 72 hours. Approximately 10-12% was excreted as 14C-CO₂ in breath, cumulative to 72 hours; less than 0.12% was excreted as BCM in breath of male mice. Cumulative elimination via all routes accounted for greater than 90% of dose in each sex.

Tissue distribution in male and female mice was similar, and body burdens approximated less than 1% of the administered dose. After 24 hours, 14C in blood was unextractable. At 72 hours, blood concentrations of 14C (in BCM equivalents) were 162 ng/gram, and 118 ng/gram for male and female mice, respectively. For males, tissues with 14C concentrations higher (ratios of tissue:blood concentrations in parentheses) than blood included liver (2.88), kidney (2.48), thymus (1.72), skin (1.33), lung (1.31), spleen (1.29), and adipose (1.18). For female mice, tissues with 14C concentrations higher than blood included liver (3.10), thymus (2.72), kidney (2.61), adipose (1.86), ovaries (1.82), lung (1.56), spleen (1.41), and skin (1.05). Heart tissue contained concentrations of 14C approximating 85% that of blood for both sexes.

As in mice, BCM was rapidly eliminated from orally-exposed male rats, but higher rates and extent of elimination occurred via the urine; this route accounted for more than 50% of the dose at 8 hours, and for 90% of the dose at 72 hours. Feces accounted for approximately 0.4% of the dose at 72 hours. Exhalation of 14C-CO₂ accounted for approximately 7% of the dose, and exhaled BCM accounted for less than 0.2% of the administered dose. Less than 2.5% of the dose's 14C equivalent was retained in the body at 72 hours. Blood concentrations of 14C (in BCM equivalents) were 390 ng/gram. For male rats, tissues with 14C concentrations higher (ratios of tissue:blood concentrations in parentheses) than blood included liver (1.74) and thymus (1.69). While higher blood concentrations in the rat may lead to speculation that species differences in the apparent concentrations of BCM in blood may shift the pattern of blood:tissue distribution, most rat solid tissues also contained higher concentrations of BCM equivalents than their mouse counterparts.

Male mice were dosed with 1.0 mg/kg, i.v. and female mice were dosed i.v. with BCM at 0.1 and 1.0 mg/kg. For all mice, urinary and fecal elimination was characterized for 72 hours; tissue distribution was evaluated for male mice. Combined urinary and fecal elimination for all dose groups approximated 85 to 95% at 72 hours, with urine accounting for 65 to 72% of the administered dose. Sex-dependent differences in the fraction exhaled seemed evident for the 1 mg/kg mice. This route accounted for nearly 10% of the dose in males and approximately 5% of

dose in females. Males eliminated nearly twice as much of the dose unchanged in expired air than did females, and approximately three-fold more of the dose as CO₂ than did females. In females administered an i.v. dose of 0.1 mg/kg, a slightly lower fraction of the dose was eliminated in urine and feces, and a slightly higher fraction of the dose was eliminated as expired CO₂ when compared to females administered 1.0 mg/kg via i.v.

In male mice administered 1.0 mg/kg BCM i.v., at 72 hours, approximately 4% of the administered dose was retained in the body. Blood concentrations were approximately 19 ng equivalents/gram, and tissues with ¹⁴C concentrations higher (ratios of tissue:blood concentrations in parentheses) than blood included kidney (2.33), liver (1.94), adipose (1.45), thymus (1.24), and lung (1.08). Heart contained approximately 71% the concentration of BCM equivalents as blood.

In male and female rats administered 1.0 mg/kg BCM i.v., the time course profile demonstrated rapid and marked decline of BCM, where levels circulating dose approximated 2% of administered dose within 15 minutes. BCM equivalents demonstrated a biphasic decline with the terminal slope appearing largely defined by the proportion of unextractable ¹⁴C residues. For example, for males and females, total BCM equivalents decreased from 362 to 67 and from 283 to 45 ng equivalents/ gram blood between 15 minutes and 24 hours, respectively. During this time the percentage of blood ¹⁴C present as unextractable fraction increased from approximately 20% to approximately 75% for males and from approximately 22% to approximately 95% for females. Similar results were demonstrated in male mice administered 1.0 mg/kg BCM. In addition to blood, liver and thymus tissues were analyzed for extractable radioactivity. At 15 minutes post-dosing, less than 30% of the total radioactivity in liver was extractable, with the majority of extracted radiolabel represented by parent compound. At 8 hours post-dosing, less than 5% of the total radioactivity present in liver tissue was extractable, and virtually no parent BCM was demonstrated. Results in thymic tissue were qualitatively the same: at 8 hours approximately 85% of the total radioactivity was extractable, and approximately 45% of the extractable radioactivity represented parent BCM; at 8 hours post-dosing extractable radiolabel in thymus represented approximately 10% of total radioactivity, with parent BCM levels approaching zero. The high level of binding early in the time profile (15 minutes) seems inconsistent with incorporation of radiolabelled moiety into protein.

Mathews and Jeffcoat (2002) also reported the results of investigations of BCM metabolism. The results of an experiment in which cytochrome P4502E1 was inhibited demonstrated no change in the blood concentration-time profile in male mice administered 1.0 mg/kg BCM. Urine collected from rats administered 10 and 0.1 mg/kg BCM orally and male rats administered 1.0 mg/kg i.v. demonstrated three distinct peaks, accounting for 80 to 88% of urinary ¹⁴C, when analyzed by high performance liquid chromatography, and none of these peaks was altered when urine was incubated with sulfatase, acylase and beta-glucuronidase. One of these metabolites co-eluted with thiodiglycolic acid; subsequent gas chromatography/mass spectrometric analysis confirmed that metabolite as thiodiglycolic acid. This metabolite accounted for 49 to 51% of recovered ¹⁴C. The peak that co-eluted with the sulfoxide of thiodiglycolic acid accounted for 25-31% of urinary radiolabel. Combined recoveries for these two peaks accounted for between 74 and 82% of urinary radiolabel. With the preponderance of

radiolabel eliminated in urine, these data support thiodiglycolic acid as the major metabolite of BCM.

Oral-Dermal Dose Comparison. Data from 90-day studies conducted via the dermal route of exposure in rats (Battelle, 2002a) and mice (Battelle, 2002b) offer additional insights on the dose-response relationship for several toxicities. However, in order for advantage to be made from these results, some measure of absorbed, rather than applied dose is required. The relationship between dermally applied and absorbed dose can be developed from information from a distribution study also recently available (Mathews and Jeffcoat, 2002). Twenty-four hours after administration, 15.73 and 15.44% of dermally applied doses were absorbed by rats, and urinary elimination accounted for 91.1 and 88.1% of the absorbed dose in male rats receiving dermal doses of 0.1 and 10 mg/kg. The absorbed dose from these two exposures corrects to 0.016 and 1.5 mg/kg. In mice, 24 hours after application, approximately 9% of a 0.1 mg/kg dose was absorbed, and urinary elimination accounted for 95.5% of the absorbed dose. In mice dermally exposed to 10 mg/kg, approximately 18% of the dose was absorbed, and urinary elimination accounted for 68.8% of the absorbed dose. In mice, the study authors noted the confounding issue of cross contamination of urine and feces occurring in the metabolism cage. Combined urinary and fecal elimination in 10 mg/kg-dosed mice accounted for 78.3% of the absorbed dose. While the fraction of absorbed ^{14}C eliminated in urine is similar between oral and dermal exposures, there is no information on the comparative metabolism of ^{14}C -labelled BCM, and there is evidence that the thiodiglycolic acid metabolite may be responsible for the noted cardiotoxicity.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfDs FOR BIS(2-CHLOROETHOXY)METHANE

No pertinent data regarding the oral toxicity of bis(2-chloroethoxy)methane in humans are available. Only one subchronic study of oral administration of bis(2-chloroethoxy)methane to rats was located: a 90-day oral gavage study conducted by Bio/Dynamics (1990a), wherein Sprague-Dawley rats (10/sex/dose group) received oral doses of 0, 10, 20, 40, 80, or 120 mg/kg-day of bis(2-chloroethoxy)methane in corn oil. Subchronic and chronic oral RfDs for bis(2-chloroethoxy)methane can be derived using a NOAEL/LOAEL approach, based on liver lesions (centrilobular hepatocellular hypertrophy) in male rats receiving 20 mg/kg-day or more of bis(2-chloroethoxy)methane. This study identified a NOAEL of 10 mg/kg-day for the critical effect. The finding that the liver is a sensitive target for bis(2-chloroethoxy)methane is supported by the short-term range-finding study (Bio/Dynamics, 1990b).

To the rat NOAEL of 10 mg/kg-day for liver lesions established by Bio/Dynamics (1990a), a combined uncertainty factor of 300 was applied. The uncertainty factors included a 10 for interspecies extrapolation, a 10 for human variability, and a 3 for database deficiencies (including lack of reproductive and developmental toxicity tests), resulting in a combined uncertainty factor of 300. A provisional **subchronic oral RfD of 0.03 mg/kg-day** was calculated as follows:

$$\begin{aligned}
 \text{p-sRfD} &= \text{NOAEL} / \text{UF} \\
 &= 10 \text{ mg/kg-day} / 300 \\
 &= 0.03 \text{ mg/kg-day or } 3\text{E-}2 \text{ mg/kg-day}
 \end{aligned}$$

A provisional chronic oral RfD can also be derived by dividing the NOAEL of 10 mg/kg-day established by Bio/Dynamics (1990a) by a combined uncertainty factor of 3000. The uncertainty factors included a 10 for extrapolation from a subchronic study, a 10 for interspecies extrapolation, a 10 for human variability, and a 3 for database deficiencies, resulting in a combined uncertainty factor of 3000. A provisional **chronic oral RfD of 0.003 mg/kg-day** was calculated as follows:

$$\begin{aligned}
 \text{p-RfD} &= \text{NOAEL} / \text{UF} \\
 &= 10 \text{ mg/kg-day} / 3000 \\
 &= 0.003 \text{ mg/kg-day or } 3\text{E-}3 \text{ mg/kg-day}
 \end{aligned}$$

Confidence in the principal study is low. The principal study examined a number of relevant endpoints; however, the study used only minimally adequate group sizes, failed to conduct histopathology on all tissues at lower exposure doses, and appears to have used a dose that was too high based on the range-finding study (Bio/Dynamics, 1990b). Confidence in the database is also low: the database is lacking human data, supporting subchronic or chronic animal studies, and studies of developmental, reproductive, or neurological effects of exposure to bis(2-chloroethoxy)methane. Reflecting low confidence in the principal study and low confidence in the database, confidence in the provisional RfD is low.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC RfCs FOR BIS(2-CHLOROETHOXY)METHANE

Derivation of a provisional subchronic or chronic RfC for bis(2-chloroethoxy)methane is precluded by the absence of inhalation toxicity data.

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7-24-2007

Provisional Peer Reviewed Toxicity Values for

Chloroethane
(CASRN 75-00-3)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR CHLOROETHANE (CASRN 75-00-3)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

No reference dose (RfD) assessment is available for chloroethane (ethyl chloride) in the Integrated Risk Information System (IRIS) database (U.S. EPA, 2006a) or in the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997). The Chemical Assessments and Related Activities (CARA) list (U.S. EPA, 1991, 1994a) includes a Health Effects Assessment for Ethyl Chloride (U.S. EPA, 1987). Although an Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Chloroethane (ATSDR, 1998) is available, no oral minimal risk levels (MRLs) were derived for chloroethane because no relevant oral data were located.

The IRIS database (U.S. EPA, 2006a) includes a reference concentration (RfC) of 10 mg/m³ (verified 12/20/1990) for chronic exposure to ethyl chloride (chloroethane), based on a NOAEL of 4000 mg/m³ and a LOAEL of 13,000 mg/m³ for delayed fetal ossification in a mouse developmental inhalation study (Scortichini et al., 1986). The same critical effect (delayed ossification in mice) from the study of Scortichini et al. (1986) was used by ATSDR (1998) to derive an acute-duration inhalation MRL of 15 ppm (40 mg/m³) and by CalEPA (2006a) to derive a chronic reference exposure level (REL) of 30,000 µg/m³ (30 mg/m³).

A cancer assessment for chloroethane is not available on IRIS (U.S. EPA, 2006a) or in the HEAST (U.S. EPA, 1997). CalEPA (2006b) includes chloroethane in its List of Chemicals Known to the State to Cause Cancer or Reproductive Toxicity (updated December 2, 2005). The National Toxicology Program (NTP) performed a 2-year inhalation toxicity and carcinogenicity study of ethyl chloride in rats and mice, resulting in the conclusion that there was equivocal

evidence of carcinogenic activity in rats and clear evidence of carcinogenic activity in female mice (NTP, 1989). The International Agency for Research on Cancer (IARC) assigned chloroethane to Group 3 (not classifiable as to its carcinogenicity to humans), based on limited evidence for the carcinogenicity of chloroethane in animals and no available human data (IARC, 1991). NIOSH (2006) includes a warning to handle ethyl chloride with caution in the workplace due to structural similarity to other chloroethanes shown to be carcinogenic in animals. A carcinogenicity assessment for chloroethane is not available from the World Health Organization (WHO, 2006).

An Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Chloroethane is available (ATSDR, 1998). Update literature searches for more recent information were performed for the time period of 1993 to December, 2005 in TOXLINE, MEDLINE (plus PubMed cancer subset), and DART/ETICBACK. Update search of the TOXCENTER database was performed for the time period of August, 2000 to December, 2005. Databases searched without date limitations included TSCATS, RTECS, GENETOX, HSDB and CCRIS. Search of Current Contents encompassed July to December, 2005.

REVIEW OF PERTINENT DATA

Human Studies

Oral Exposure

No data were located regarding the oral toxicity or carcinogenicity of chloroethane in humans.

Inhalation Exposure

Chloroethane has been used as a general anesthetic in humans at inhaled concentrations in the same range (3-4.5%) as its explosive concentration of 4% (40,000 ppm (105,521 mg/m³))* in air. Blood levels required to achieve general anesthesia range from 20 to 30 mg% (mg/100 mL), whereas respiratory failure can be triggered by slightly higher blood levels (40 mg%) (Dobkin and Byles, 1971). Sublethal adverse effects of overexposure to inhaled chloroethane are predominantly neurological (Finch and Lobo, 2005; Hes et al., 1979; Nordin et al., 1988), but may include hepatic effects (Hes et al., 1979). Davidson (1925) reported the results of short-term inhalation exposure of human subjects to chloroethane. Exposure to 13,000 ppm (34,294 mg/m³) for 12 minutes resulted in feelings of intoxication and reduced reaction time. At an exposure level of 19,000 ppm (50,123 mg/m³), slight intoxication was reported within 1 minute and progressed to distinct intoxication and mild analgesia within 12 minutes. Higher concentrations (25,000 ppm for 15 minutes, 33,600 ppm for 8 minutes [5,957 and 88,638 mg/m³ respectively]) resulted in concentration-related incoordination. At the highest exposure level (33,600 ppm), unconsciousness was achieved within 13-17 minutes. Sayers et al. (1929)

* Concentrations were determined using the following formulae:

$$\text{xppm} = (\text{y mg/m}^3)(24.45)/(\text{molecular weight})$$

$$\text{ymg/m}^3 = (\text{xppm})(\text{molecular weight})/24.45$$

reported dizziness and abdominal cramping in two human subjects who inhaled two breaths of a 4% (40,000 ppm) concentration of chloroethane in air or three or four breaths of a 2% concentration.

No studies were located regarding the carcinogenicity of chloroethane in humans exposed by inhalation.

Animal Studies

Oral Exposure

Male and female Fischer 344 rats (10/sex/group) were given drinking water containing 0 or 5700 mg chloroethane/L water for 14 days (Dow Chemical Company, 1995). Estimated chloroethane doses to the male and female rats were 297 and 361 mg/kg-day, respectively. Rats were assessed for clinical signs of toxicity, body weight and food and water consumption. Other parameters evaluated included clinical chemistry [serum aspartate aminotransferase (AST), alanine aminotransferase (AST) and alkaline phosphatase (AP)], hematology, organ weights and gross and histopathological (liver only) examinations. There were no indications of chloroethane-induced toxicity from any of the parameters evaluated. Decreased water consumption (20-25% lower in chloroethane-treated rats, relative to controls) was considered to be the result of reduced palatability. Decreased food consumption and body weight were considered to be secondary to decreased water consumption. Slight changes in selected mean organ weights were within 10% of control values and were consistent with decreased water and food consumption.

In an early study in rabbits (Rowe et al., 1939), administration of 500 or 1000 mg/kg-day of chloroethane by gavage on work days through a total of 60 doses (assumed to be 5 days/week for 12 weeks) did not elicit clinical signs of toxicity or treatment-related effects on body weight gain. Histopathological examinations (tissues not specified) revealed no evidence of treatment-related effects. The available results of this study were limited to a summary statement.

Inhalation Exposure

Groups of male and female Fischer 344 rats (6/sex/exposure concentration) were exposed to chloroethane (99.7% pure) by inhalation at concentrations of 0, 1600, 4000 or 10,000 ppm (0, 4288, 10,720 or 26,800 mg/m³) for 6 hours/day, 5 days/week for 2 weeks and observed for clinical signs of toxicity (Landry et al., 1982). Body weights were monitored during the study and clinical chemistry, urinalysis, hematology and comprehensive histopathological examinations were performed. Significant increases in relative liver weight (4.9 and 7.5% greater than controls) were noted in male rats of the 4000 and 10,000 ppm groups, respectively, in the absence of liver histopathology or increased levels of serum enzymes. There were no other signs of chloroethane-induced effects. No effects were seen in male beagle dogs (2/group) subjected to the same exposure scenario and analysis.

Landry et al. (1989) exposed groups of B6C3F1 mice (7/sex/group) to chloroethane (99.9% pure) by inhalation at vapor concentrations of 0, 250, 1250 or 5000 ppm (0, 670, 3350 or

13,400 mg/m³) for 23 hours/day on 11 consecutive days. Body weights were recorded periodically during the study. On the day following the final exposure period, a neurobehavioral battery of tests was performed. Clinical chemistry, hematology and comprehensive histopathological analyses were conducted. The only indication of chloroethane-related effects consisted of significantly increased mean relative liver weight (approximately 9-12% greater than controls) and a minimal increase in degree of hepatocellular vacuolization in 4/7 of the 5,000-ppm male and female mice. These liver effects were not accompanied by increased serum enzyme levels and were considered to be an adaptive response.

In a study designed to assess chloroethane metabolism, Fedtke et al. (1994a) exposed groups of male and female F-344 rats and B6C3F1 mice (2 rats or 10 mice/sex/group) to chloroethane (>99% pure) by inhalation at exposure concentrations of 0 or 15,000 ppm (0 or 40,200 mg/m³) for 5 days (6 hours/day). At necropsy, livers, lungs, kidneys and uteri were removed and weighed. The only indication of chloroethane-induced toxicity was significantly decreased mean uterine weight (approximately 35% lower than controls, data not shown) in the female mice of the 15,000 ppm exposure level. Body weights and relative uterine weights were not reported, but all groups of mice, including sham-exposed controls, were stated to have lost weight, possibly due to exposure-related stress.

In an unpublished study, Scortichini et al. (1986) assessed the developmental toxicity of chloroethane in CF-1 mice. In a range-finding study, groups of 8-10 bred female CF-1 mice were exposed to 0, 5000, 10,000 or 15,000 ppm of chloroethane (99.9% pure) for 6 hours/day on gestation days 6 through 15. The study authors reported increased locomotor activity and significantly decreased body weight and body weight gain in all chloroethane-exposed dams, but did not include actual data for these findings. In the main study, groups of bred female mice (30/group) were exposed to chloroethane (99.9% pure) by inhalation at target concentrations of 0, 500, 1500 or 5000 ppm (analytical concentrations of 0, 491, 1504 and 4946 ppm; approximately 0, 1300, 4000 and 13,000 mg/m³) for 6 hours/day on gestation days 6 through 15. Dams were observed daily for clinical signs of chloroethane-induced toxicity. Maternal body weights and food and water consumption were monitored. At sacrifice on gestation day 18, maternal liver weights and gravid uterine weights were recorded. Numbers of pregnant dams, resorptions and live and dead fetuses were noted, as well as fetal weight and sex and gross external fetal alterations. Apparently nonpregnant mice were assessed for evidence of implantation sites. Examinations for signs of visceral alterations were performed on one-half of the fetuses from each litter. All fetuses were examined for evidence of cardiac and skeletal anomalies. Bones of the skull were examined for anomalies in approximately one-half of the fetuses; heads of the other fetuses were assessed for other effects.

There were no indications of chloroethane-induced maternal effects at any exposure level (Scortichini et al., 1986). Evaluation of reproductive parameters in pregnant mice revealed no indication of adverse effects on pregnancy rate, resorption rate, litter size, fetal sex ratios or fetal body weights. No significant exposure-related effects on incidences of fetal visceral anomalies were detected. A small, but significant ($p=0.05$) increase in the incidence of foramina of the skull bones (delayed fetal ossification) was noted in fetuses of the 4946 ppm exposure group (1/126, 1/142, 1/174 and 5/116 fetuses of the 0, 491, 1504 and 4946 ppm exposure groups, respectively). On a per litter basis, respective incidences were 1/22, 1/24, 1/25 and 5/22 litters.

Incidences of litters exhibiting supernumerary ribs were elevated at the higher exposure levels [2/22 (9%), 1/25 (4%), 5/26 (19%) and 4/22 (18%) in 0, 491, 1504 and 4946 ppm exposure groups, respectively]. The statistical significance of this effect was not indicated in the study report and did not appear to increase with increasing exposure concentration on a per fetus basis [2/257 (1%), 1/299 (0.3%), 6/311 (2%), and 2/242 (2%) in 0, 491, 1504, and 4946 ppm groups, respectively]. The authors considered this observed effect to be questionable. There were no other indications of chloroethane-induced fetal effects. Although the authors did not identify it as such, this study identified a NOAEL of 1504 ppm and a LOAEL of 4946 ppm for fetal effects (delayed fetal ossification).

A series of studies were conducted for the National Toxicology Program to assess the toxicity and carcinogenicity of inhaled chloroethane (99.5% pure) in male and female F344/N rats and B6C3F1 mice (NTP, 1989). Preliminary 4-hour and repeated 14-day and 13-week exposure studies (6 hours/day, 5 days/week) were performed prior to a 2-year toxicity and carcinogenicity study. In the 4-hour and repeated 14-day studies, no overt signs of chloroethane-induced toxicity were seen in the rats or mice (5/sex/species) exposed to chloroethane vapors at a concentration of 19,000 ppm (50,920 mg/m³). No gross or histopathological signs of chloroethane-induced toxicity were seen in rats or mice in the 14-day studies.

In the 13-week repeated exposure study, groups of 10 animals/sex/species were exposed to chloroethane concentrations of 0, 2500, 5000, 10,000 or 19,000 ppm (0, 6700, 13,400, 26,800 or 50,920 mg/m³) for 6 hours/day, 5 days/week for 13 weeks (NTP, 1989). Animals were observed daily for clinical signs of toxicity. Body weights were recorded weekly. Comprehensive gross and histopathological examinations were performed on each animal.

All rats survived until terminal sacrifice (NTP, 1989). No compound-related clinical signs of toxicity were observed in rats. Mean final body weights of all groups of chloroethane-exposed male and female rats ranged from 4 to 8% lower than respective controls, but were statistically significantly lower ($p < 0.01$) only in the 19,000-ppm male rats. Relative mean liver weight was significantly increased only in 19,000-ppm male rats (approximately 14% higher than controls). Gross and histopathologic examinations revealed no signs of chloroethane-related adverse effects in male or female rats.

Chloroethane-exposed mice also survived until terminal sacrifice, with the exception of a single male mouse of the 10,000-ppm exposure group (NTP, 1989). No compound-related clinical signs of toxicity were observed in mice. Final body weights of chloroethane-exposed mice were generally slightly higher than controls. Relative mean liver weight was significantly ($p < 0.01$) increased in 19,000-ppm female mice (approximately 18% higher than controls). There were no indications of chloroethane-induced gross or histopathologic effects. Observed nasal cavity hemorrhage of minimal severity in 3/10 male and 6/10 female mice of the 19,000 ppm exposure level was considered by NTP to be an artifact of necropsy in the absence of microscopic lesions in the nasal mucosa of these mice.

In the 2-year toxicity and carcinogenicity bioassay, groups of 50 animals/sex/species were exposed to chloroethane vapor concentrations of 0 (inhalation chamber controls) or 15,000 ppm (40,200 mg/m³) for 6 hours per day, 5 days per week for 102 weeks (rats) or 100 weeks

(mice). NTP (1989) conducted the 2-year studies using air-exposed controls and a single chloroethane exposure level in order to obtain structure-activity comparative data with results of a concurrent study of bromoethane. The 15,000 ppm level was selected for the 2-year study due to concerns about the potential flammability and explosion hazard of higher concentrations and because no effects were seen in the subchronic study at a slightly higher exposure level. All animals were observed twice per day for clinical signs. Body weights were recorded weekly for the first 12 weeks and monthly thereafter. Comprehensive gross and histopathological examinations were performed for each animal in the study.

No chloroethane-induced clinical signs of toxicity were observed in rats of either sex exposed for 2 years (NTP, 1989). No significant differences in survival were noted between exposed and control groups of rats of either sex, but survival of exposed and control male rats was unusually low at the end of the study. The authors reported that unusually high incidences of mononuclear cell leukemia in both control and exposed groups of male rats may have contributed to the high mortality. The authors also reported that survival for all groups was sufficient through weeks 90 and 95 to evaluate carcinogenicity. At the end of the study (102 weeks), survival for male rats was 16/50 (controls) and 8/50 (exposed) and for female rats was 31/50 (controls) and 22/50 (exposed); however, at 90 weeks, survival was 37/50 (control) and 31/50 (exposed) for respective male groups and 43/50 (control) and 33/50 (exposed) for females. Mean body weights of exposed male rats were 4%-8% lower than those of controls after week 33 and in exposed female rats body weights ranged from 5-13% lower than controls after week 11.

Three exposed female rats displayed uncommon astrocytomas (malignant glial cell tumors of the brain) (NTP, 1989). The authors reported that although the overall incidence of malignant glial cell tumors (3/50) was not statistically significantly different ($p > 0.05$) from the concurrent controls (0/50), it was statistically significantly increased ($p < 0.05$) relative to incidences for previous chamber control groups at the study laboratory (1/297) or for untreated control female F344/N rats from previous NTP studies (23/1969 = 1%). Primary tumors of glial cell origin were also observed in exposed male rats. One control male had a malignant oligodendroglioma. A benign oligodendroglioma and a malignant astrocytoma were observed in two exposed males.

There were five exposed male rats that had epithelial tumors of several types with similar characteristics (trichoepithelioma, sebaceous gland adenoma and basal cell carcinoma) (NTP, 1989). The combined overall incidence (5/50) was not significantly different from the concurrent control incidence (0/50), but statistical significance ($p < 0.05$) could be demonstrated when comparisons were made to historical incidences in chamber controls (2/300) at the study laboratory or in untreated controls (30/1936 = 1.5%) from NTP studies.

NTP (1989) concluded that the study provided equivocal evidence of carcinogenic activity in both male and female F344/N rats, even though comparisons with concurrent controls were negative, because comparisons with historical controls indicated significant differences.

In the 2-year study, chloroethane-exposed female mice, but not male mice or rats of either sex, were hyperactive during daily exposure, but returned to normal shortly after the exposure period ended (NTP, 1989). Survival of 15,000 ppm exposed mice was significantly

lower than that of control mice; statistical significance for reduced survival was demonstrated for exposed male mice after day 330 and for exposed female mice after day 574. All surviving mice were sacrificed at 100 weeks. Mean body weights of exposed male mice were up to 13% higher than control male mice. Mean body weights for exposed and control female mice were generally similar throughout the study.

Decreased survivability in exposed male mice was not related to tumor occurrences (NTP, 1989). The authors noted that greater than normal incidences of nonneoplastic urogenital lesions were observed in both control and exposed male mice and that this occurrence may have contributed to the reduced survival. The overall incidences of alveolar/bronchiolar adenomas (8/48) and of alveolar/bronchiolar adenomas and carcinomas (combined) (10/48) among exposed male mice were statistically significantly greater ($p < 0.05$) than respective incidences for control male mice (3/50 and 5/50). The authors, however, considered the study of male B6C3F1 mice inadequate to evaluate carcinogenic activity because of the reduced survival.

Most of the early mortalities in exposed female mice were associated with carcinomas of the uterus (NTP, 1989). The overall incidence of uterine carcinomas (all of endometrial gland origin) in exposed female mice (43/50) was significantly ($p < 0.001$) greater than that of the concurrent controls (0/49). Uterine carcinomas were first noted on day 469 of the study. The tumors were highly malignant, invasive and, in 34 animals, metastasized to other organs. Exposed female mice also displayed statistically significantly higher ($p < 0.05$, according to a logistic regression test) overall incidences of hepatocellular carcinomas (7/48) and hepatocellular carcinomas and adenomas (combined) (8/48) compared to respective incidences in control female mice (3/49 and 3/49).

Picut et al. (2003) re-evaluated the pathology and incidence data from the NTP (1989) study and confirmed the NTP findings of increased incidences of uterine cancer in female B6C3F1 mice exposed to 15,000 ppm of chloroethane vapors 6 hours/day, 5 days/week for 100 weeks (NTP, 1989).

Based on the results of the NTP (1989) study in which high incidences of uterine carcinomas were observed in mice chronically exposed to chloroethane vapors at a concentration of 15,000 ppm, Bucher et al. (1995) designed a study to assess the potential for chloroethane to induce early changes on sex hormones (estradiol and progesterone). Groups of virgin female B6C3F1 mice (30/group) were exposed to 0 or 15,000 ppm of chloroethane (99.7% pure) by inhalation 6 hours/day for 21 days after having been sham-exposed for an initial 21-day period. There were no clinical signs of chloroethane-induced toxicity and no exposure-related effects on weight gain. No changes were seen in weights of the liver, uterus, or ovary, or in histopathology of the ovaries, pituitary, uterus, or adrenal glands. Blood concentrations of sex hormones were not significantly affected by chloroethane exposure, but variability was high. Compared to the mean duration of estrous cycle during the 21 days of sham exposure (5.15 ± 0.15 days), the mean duration of estrous cycle during the subsequent 21 days of chloroethane exposure (5.52 ± 0.15 days) was slightly but significantly ($p < 0.05$) increased. There was also a significant difference ($p < 0.05$) in the proportion of time spent in the different estrous stages during exposure compared to the time period of sham exposure in both control and chloroethane-exposed mice. Thus, no

consistent exposure-related patterns of change were found in estrous cyclicity or circulating levels of sex hormones.

In an early study of rabbits (4/group) and rats (12/group) exposed to chloroethane vapors at a concentration of 26,400 mg/m³ (9847 ppm) for 7.5-8 hours/day, 5 days/week for 6.5 months, no exposure-related clinical signs or effects on weight gain, liver weights or histopathology were observed (Rowe et al., 1939).

Troshina (1964) reported adverse respiratory and liver effects in rats (sex and species unspecified) exposed to chloroethane by inhalation at a concentration of 14,000 mg/m³ (5222 ppm), 2 hours/day for 60 days. In a subsequent report, Troshina (1966) described several exposure-related effects including disturbed liver function, lowered blood pressure, fatty liver and apparent intraalveolar thickening in the lungs of rats exposed to chloroethane 4 hours/day, 6 days/week for 6 months at chloroethane concentration as low as 8.5 mg/m³ (3.17 ppm). Both reports are deficient in numerous study details and control groups, which preclude their usefulness for quantitative risk assessment.

Other Studies

Two reports provide evidence for the mutagenicity of chloroethane in the closed-desiccator *Salmonella typhimurium* test for reverse mutations. Riccio et al. (1983) observed mutations in strains TA98, TA100, TA1535 and TA1537 in both the presence and absence of metabolic activation. NTP (1989) observed mutagenic activity in strain TA1535 with or without activation and in strain TA100 only with activation, but no mutagenic activity was observed in strain TA98 with or without activation. Chloroethane was mutagenic to the HPRT (hypoxanthine-guanine phosphoribosyl transferase) locus of Chinese hamster ovary cells both with and without metabolic activation (Ebert et al., 1994). However, exposure of female B6C3F1 mice to chloroethane at a concentration of 25,000 ppm (6 hours/day for 3 days) did not induce unscheduled DNA synthesis; similar exposure of male and female B6C3F1 mice did not result in increased numbers of micronuclei in bone marrow cells (Ebert et al., 1994).

DERIVATION OF A PROVISIONAL SUBCHRONIC RfD FOR CHLOROETHANE

No information was found regarding the oral toxicity of chloroethane in humans. Information regarding repeated-dose oral toxicity of chloroethane in animals was restricted to the results of two limited studies in which no adverse effect levels were identified.

Rowe et al. (1939) reported no clinical or histopathological signs of toxicity, or treatment-related effects on body weight gain among rabbits administered chloroethane doses of 500 or 1,000 mg/kg-day by gavage for 60 doses (assumed to be 5 days/week for 12 weeks). However, available results for this study were limited to a summary statement reporting few details.

Chloroethane did not appear to be toxic to rats given the chemical in drinking water for 14 days at a concentration (5700 mg/L) resulting in doses estimated in the report (Dow, 1995) to be 297 mg/kg-day in male rats and 361 mg/kg-day in female rats. The authors considered decreased water consumption among treated animals (20-25% less than controls) to result from reduced palatability. They concluded that decreased food consumption was secondary to the decreased water consumption and that reduced weight gains and slightly reduced organ weights resulted from reductions in consumption of food and water.

Although Rowe, et al (1939) provide the highest oral NOAEL for the longest dosing period, an unacceptably low level of confidence in the study makes these data unacceptable for use as the point of departure for deriving an oral RfD. The Dow (1995) study reported free-standing drinking water NOAELs of 361 mg/kg-day among female rats and 297 mg/kg-day in male rats dosed for 14 days. The slightly higher dose in female rats was chosen as the point of departure (POD) for deriving a subchronic oral RfD, because both values were freestanding NOAELs. A composite uncertainty factor of 3,000 for a subchronic oral p-RfD was calculated from the following individual uncertainties:

- 10 - inter-human variability
- 10 - mouse to human extrapolation
- 10 - database deficiencies (e.g., no developmental or reproductive oral studies; inhalation developmental data are available)
- 3 - adjustment from 14-day study to subchronic RfD

Subchronic oral p-RfD = (361 mg/kg-day)/3,000 = **0.1 mg/kg-day**

Confidence in the key study, Dow (1995), is medium because the duration of exposure was less than subchronic and because a LOAEL was not identified. Confidence in the database is low because of the lack of studies of appropriate duration and the absence of reproductive and developmental toxicity studies. Consequently, confidence in the subchronic p-RfD is low.

The existing database does not support the derivation of a chronic oral p-RfD because of the lack of 90-day or chronic exposure studies.

DERIVATION OF A PROVISIONAL SUBCHRONIC RfC FOR CHLOROETHANE

As discussed earlier, chloroethane has been used as a general anesthetic in humans. Information regarding chloroethane-induced neurological effects is available for short-term high-level exposure (Davidson, 1925; Dobkin and Byles, 1971; Finch and Lobo, 2005; Hes et al., 1979; Nordin et al., 1988; Sayers et al., 1929). One case report described possible liver effects from repeated abuse of inhaled chloroethane (Hes et al., 1979). These limited human reports are not adequate for purposes of quantitative risk assessment for chloroethane.

Repeated inhalation exposure of adult male and female F344/N rats and B6C3F1 mice resulted in no evidence of exposure-related noncancer effects from exposures as high as 15,000

ppm (6 hours/day, 5 days/week) for up to 2 years, except for hyperactivity in female mice during exposure (NTP, 1989). Other reports (Landry et al., 1982, 1989; Rowe, 1939) found no adverse effects in adult rats, mice or rabbits repeatedly exposed to chloroethane vapors at the highest concentrations tested (10,000, 4843 and 9847 ppm, respectively). However, repeated inhalation exposure of female B6C3F1 mice to a chloroethane vapor concentration of 15,000 ppm (6 hours/day for 5 days) resulted in significantly decreased mean uterine weight (Fedtke et al., 1994a) and slightly increased duration of the estrous cycle (Bucher et al., 1995). Developmental effects (foramina of the skull bones) were noted in fetuses of CF-1 mice exposed to chloroethane at an analytical concentration of 4946 ppm for 6 hours/day on gestation days 6 through 15 (Scortichini et al., 1986). This study served as the basis for a chronic RfC of 10 mg/m³ for ethyl chloride (chloroethane), which is available on IRIS (U.S. EPA, 2006a).

A point of departure for the provisional subchronic RfC for chloroethane is derived by benchmark dose (BMD) analysis of delayed fetal ossification in the Scortichini et al. (1986) study. All dichotomous models in the EPA Benchmark Dose Modeling Software (BMDS; Version 1.3.2) were fit to the incidence data for foramina of the skull bones on a per litter basis (see Table 1). For each model, a benchmark response (BMR) of 10% extra risk (as recommended by U.S. EPA, 2000) was used to calculate an Effect Concentration (EC₁₀) and its lower 95% confidence limit (LEC₁₀). Table 2 shows the modeling results for each of the dichotomous models.

Table 1. Incidences of foramina of the skull bones in fetuses of CF-1 mouse dams exposed to chloroethane by inhalation for 6 hours/day on gestation days 6 through 15 (Scortichini et al., 1986).

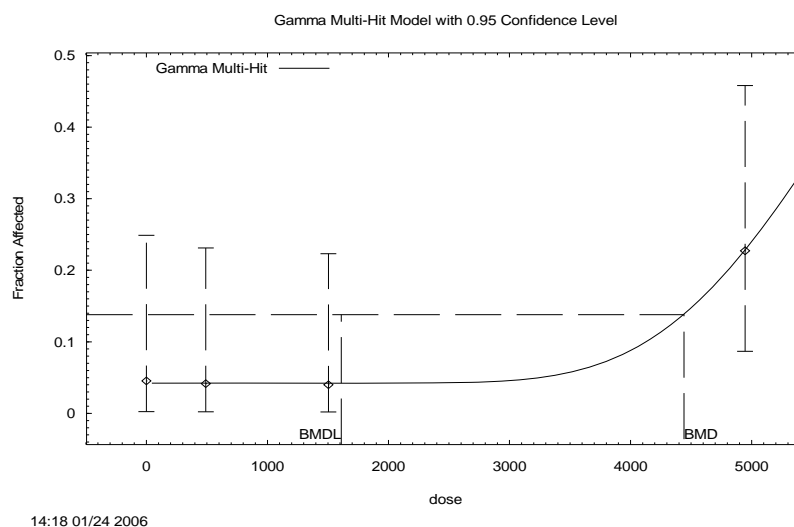
	Exposure level (ppm)		
	0	491	1504
			4946
1/22 ^a	1/24	1/25	5/22

^a Number of affected litters/number of litters examined

All models provided acceptable global goodness of fit (chi square p-value ≥ 0.1). As recommended by U.S. EPA (2000), the model with the lowest Akaike Information Criterion (AIC) value (Gamma model) was selected as the best fitting model, which yielded an EC₁₀ of 4442 ppm and an LEC₁₀ of 1609 ppm. A plot of the observed and expected fraction of affected litters versus exposure concentration from the results of the Gamma model is shown in Figure 1. The LEC₁₀ of 1609 ppm serves as the point of departure for the provisional subchronic RfC for chloroethane.

Table 2. BMD modeling results for foramina of the skull bones (number of litters affected) in fetuses of CF-1 mice (Scortichini et al., 1986).

MODEL	EC ₁₀ (ppm)	LEC ₁₀ (ppm)	χ^2 p-value	AIC
Gamma^a	4441.64	1609.46	0.996	52.438
Quantal quadratic	3489.38	2405.34	0.918	52.609
Logistic	3449.15	2462.81	0.856	52.747
Probit	2289.76	3319.11	0.828	52.820
Quantal linear	2868.95	1405.82	0.615	53.497
Multi-stage ^c	2868.95	1405.82	0.615	53.497
Log-logistic ^b	4635.81	1480.59	0.925	54.438
Log-probit ^b	4400.96	2078.46	0.925	54.438
Weibull ^a	4655.54	1609.46	0.925	54.438

^a Power restricted to ≥ 1 ^b Slope restricted to ≥ 1 ^c Betas restricted to ≥ 0 ; Degree of polynomial = 1**Figure 1. Exposure-response modeling of incidence data for foramina of the skull bones (fraction of litters affected) in fetuses of CF-1 mice exposed to chloroethane for 6 hours/day on gestation days 6 through 15 (Scortichini et al., 1986). BMD = EC₁₀; BMDL = LEC₁₀; Dose = Concentration (ppm)**

For extrapolation of developmental effects, the intermittent exposure is duration adjusted as follows:

$$\text{LEC}_{10[\text{ADJ}]} = \text{LEC}_{10} \times \frac{6 \text{ hrs/day}}{24 \text{ hrs/day}} = 1609 \text{ ppm} \times \frac{1}{4} = 402 \text{ ppm (1078 mg/m}^3\text{)}$$

Therefore $LEC_{10[ADJ]} = 402 \text{ ppm}$ (1078 mg/m^3). According to U.S. EPA (1994b) methodology for extrarrespiratory effects of a category three gas (such as chloroethane), the $LEC_{10[HEC]}$ (human equivalent concentration) is derived by multiplying the $LEC_{10[ADJ]}$ by the ratio of the blood:gas partition coefficients ($[H_{b/g}]_A/[H_{b/g}]_H$). A value of 1 is used for the ratio of the blood:gas partition coefficients if the animal blood:gas partition coefficient is greater than the human blood:gas partition coefficient, or if one or more of the blood:gas partition coefficients are not known. The value for humans is 2.69 (Gargas et al., 1989). In the absence of an available blood:gas partition coefficient for chloroethane in the mouse,

$$LEC_{10[HEC]} = LEC_{10[ADJ]} = 1078 \text{ mg/m}^3.$$

The **subchronic p-RfC of 4E+0 mg/m³** based on delayed fetal ossification (foramina of the skull bones) in the mouse study of Scortichini et al. (1986) is derived by dividing the $LEC_{10[HEC]}$ of 1078 mg/m^3 by a composite uncertainty factor (UF) of 300, as shown below. The subchronic p-RfC of chloroethane is lower than the chronic RfC for this chemical on IRIS (U.S. EPA, 2006a) because of the application of BMDS to derive the LEC_{10} .

$$\begin{aligned} \text{Subchronic p-RfC} &= LEC_{10[HEC]} / \text{UF} \\ &= 1078 \text{ mg/m}^3 / 300 \\ &= 4 \text{ mg/m}^3 \text{ or } 4\text{E}+0 \text{ mg/m}^3 \end{aligned}$$

The composite UF includes a factor of 3 ($10^{0.5}$) for animal-to-human extrapolation using dosimetric adjustment, 10 for interindividual variability and 10 for database deficiencies.

The interspecies UF of 3 ($10^{0.5}$) reflects a factor of one for pharmacokinetic differences across species (reduced from three due to application of the dosimetric equations) and a factor of 3 ($10^{0.5}$) for pharmacodynamic considerations.

The UF of 10 is used to account for variation in sensitivity within human populations because there is limited information on the degree to which humans of varying gender, age, health status, or genetic makeup might vary in the disposition of, or response to chloroethane.

The default UF of 10 for database deficiencies is selected due to the lack of multigeneration reproductive toxicity study and a developmental toxicity study in a second animal species.

Confidence in the critical study is medium. Although the principal study (Scortichini et al., 1986) was well-conducted, it did not establish a firm exposure-response relationship with an adverse effect and did not include a maternally-toxic exposure level. Confidence in the database is medium. Although well-conducted inhalation studies of repeated exposure of rats and mice are available, most studies did not identify a LOAEL in adult animals. This may be due to the explosion hazard of chloroethane in air at concentrations above 15,000 ppm. Although the selection of developmental toxicity as a critical effect for derivation of a p-RfC is protective of a group considered to be a sensitive subgroup, the database lacks some longer-term exposure studies which might be useful for derivation of both subchronic and chronic provisional values.

Other limitations of the database include the lack of multigeneration reproductive toxicity data and the lack of additional developmental toxicity data to support the results of the principal study. The application of a database deficiencies UF was considered to adequately compensate for the database limitations. Overall, confidence in the subchronic p-RfC is medium.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR CHLOROETHANE

Weight-of-Evidence Descriptor

No data were found on the carcinogenicity of chloroethane in humans. In NTP (1989) animal studies, a high incidence of malignant uterine tumors was observed in chloroethane-exposed female B6C3F1 mice. Inhalation exposure of B6C3F1 mice (but not F344/N rats) resulted in a high incidence of uterine carcinomas (43/50 in chloroethane-exposed mice versus 0/49 in controls), which demonstrates clear evidence of chloroethane carcinogenicity (NTP, 1989). The tumors were invasive and in 34 animals metastasized to a wide variety of organs. Exposed female B6C3F1 mice also displayed significantly increased incidences of hepatocellular carcinomas (7/48) and hepatocellular carcinomas and adenomas (combined) (8/48) compared to respective incidences in controls (3/49 and 3/49), according to a logistic regression test. Other neoplastic lesions that exhibited significantly increased incidences relative to historical (but not concurrent) controls included benign and malignant epithelial neoplasms of the skin in chloroethane-exposed male F344/N rats (trichoepithelioma, 1/50; sebaceous gland adenoma, 1/50; basal cell carcinoma, 3/50; squamous cell carcinoma 2/50) and malignant astrocytomas in the brain of chloroethane-exposed female F344/N rats (3/50). Thus, there is clear evidence for carcinogenicity in female B6C3F1 mice and equivocal evidence for carcinogenicity in male and female F344/N rats. In the absence of data to indicate otherwise, the finding of chloroethane-induced uterine carcinomas in female B6C3F1 mice is considered to be relevant to humans. Chloroethane has not been extensively tested for genotoxicity, but the available studies indicate that chloroethane may be mutagenic (Riccio et al., 1983; NTP, 1989; Ebert et al., 1994). The limited mechanistic data for chloroethane do not provide clear evidence of a specific carcinogenic mode of action.

Based on these observations and in accordance with the U.S. EPA (2005) cancer guidelines, chloroethane is classified as *likely to be carcinogenic to humans* based on two factors: 1) a lack of human data and; 2) animal data that demonstrate a high degree of malignancy in chloroethane-exposed female mice.

Mode of Action Discussion

Several investigators (Fedtke et al., 1994a,b; Gargas et al., 1989; Pottenger et al., 1992) have studied the metabolism of chloroethane in an effort to discern the mechanism for induction of rare uterine tumors in female mice (NTP, 1989). A high-dose dependent disposition and GSH-dependent metabolism in mice has been suggested to account for the development of tumors in mice and not in rats (Pottenger et al., 1992). Fedtke et al. (1994a,b) examined cytochrome P450-dependent and GSH-dependent metabolism in a series of *in vitro* and *in vivo*

experiments in groups of male and female rats and mice exposed to 15,000 ppm chloroethane or air for 6 hours/day for 5 days. The authors concluded that chloroethane may be oxidatively dechlorinated by cytochrome P450 to form acetaldehyde, which enters the 2-carbon pool and is further metabolized to ethanol and acetic acid and that species differences in oxidative metabolism were not significant. In addition, rate constants estimated for rats from these experiments were consistent with those estimated earlier by Gargas et al. (1989) in a PBPK model for chloroalkanes in the rat. In assessing GSH-dependent chloroethane metabolism in rats and mice, Fedtke et al. (1994b) noted the following: 1) chloroethane could be conjugated with glutathione, converted to the mercapturic acid and excreted in the urine as the mercapturic acid (S-ethyl-N-acetyl-L-cysteine) or the non-acetylated intermediate S-ethyl-L-cysteine (mice only); 2) the rate of hepatic glutathione conjugation of chloroethane (measured by GSH-transferase specific activity) was found to be higher in both sexes of mice compared with rats; 3) when GSH concentrations were measured in the lungs, liver, kidneys and uterus, GSH was decreased in the lung and uterus of mice after exposure to 15,000 ppm, 6 hours/day for 5 days, compared with GSH concentrations in these tissues after exposure to air; and 4) decreases in GSH levels in the lungs of rats were smaller than those in mice. These results suggested that tumor formation may be dependent on chloroethane metabolism, which might explain species-specific differences in susceptibility to chloroethane carcinogenicity.

The mode of action by which chloroethane produces uterine tumors in mice is unknown. A hormonally-mediated mode of action has been postulated, but testing for the impact of early exposure (21 days) on sex hormones and estrous cyclicity did not reveal consistent exposure-related effects in B6C3F1 mice (Bucher et al., 1995). In addition, no histopathological effects were seen in ovary, uterus, pituitary or adrenals.

Although chloroethane has not been extensively assessed for genotoxicity, a genotoxic mode of action is plausible because chloroethane has been shown to produce mutagenic effects in several bacterial strains (Riccio et al., 1983; NTP, 1989) and in Chinese hamster ovary cells (Ebert et al., 1994). This genotoxic mode of action for chloroethane carcinogenicity is discussed below within the context of the modified Hill criteria of causality as recommended in the most recent Agency guidelines (U.S. EPA, 2005).

Mutagenic Mode of Action for Uterine Tumors

Key events

This mode of action hypothesizes that, following appearance in the uterine cells, chloroethane or one of its reactive metabolites reacts directly with DNA, or indirectly via induction of oxidative stress, to produce DNA damage leading to mutations in critical genes for tumor initiation.

Strength, consistency, specificity of association

Information to support this hypothetical genotoxic mode of action for chloroethane is limited to chloroethane-induced mutagenicity in several bacterial strains (Riccio et al., 1983; NTP, 1989) and in Chinese hamster ovary cells (Ebert et al., 1994).

Dose-response concordance

The only available chronic cancer bioassay (NTP, 1989) included only one high exposure concentration (15,000 ppm), which resulted in a high incidence (43/50) of uterine carcinomas in female B6C3F1 mice, and there are no dose-response data for precursor events, thus precluding an assessment of dose-response concordance.

Temporal relationships

No data are available to assess the temporal relationship between exposure to chloroethane and development of uterine carcinomas in B6C3F1 mice.

Biological plausibility and coherence

Support to the plausibility and coherence of a hypothetical genotoxic mode of action for chloroethane-induced uterine carcinomas in mice is provided by a limited number of genotoxicity assays in which chloroethane induced a mutagenic response. In the NTP (1989) 2-year cancer bioassay, chronic exposure to a high concentration of chloroethane resulted in a high incidence of uterine carcinomas in mice, but not rats. The basis for this species-specific difference in response is not known, although there is some indication that metabolic differences may play a role. The human relevance of chloroethane-induced uterine carcinomas in mice is assumed in the absence of data to indicate otherwise.

Conclusions

Based on available information regarding the carcinogenicity of chloroethane, increased incidences of uterine carcinomas in chloroethane-exposed mice are considered relevant to human health and marginally suitable for quantitative cancer assessment of chloroethane. Although a mutagenic mode of action is plausible, the available data are inadequate to establish a mode of action. Consistent with U.S. EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), a linear (e.g., non-threshold) extrapolation is indicated when a mode of action is not established.

Quantitative Estimates of Carcinogenic Risk*Oral Exposure*

There are no human or animal oral data on which to base an oral cancer assessment for chloroethane.

Inhalation Exposure

The only available inhalation carcinogenicity bioassay (NTP, 1989) used a single chloroethane exposure level (15,000 ppm) at which a high proportion (86%) of female mice developed uterine tumors. Because a mutagenic mode of action cannot be discounted and no other mode of action has been proposed, a linear non-threshold dose-response model would be

appropriate. The U.S. EPA cancer guidelines (U.S. EPA, 2005) specify a linear extrapolation from a BMDL with a BMR in the 1% to 10% range, as determined from the multistage dose-response model. In this case, however, the lowest response (86%) is far from any BMR acceptable as a POD. Therefore, the data are deemed to be inadequate for the calculation of an inhalation unit risk.

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10-12-2006

Provisional Peer Reviewed Toxicity Values for

Chlorobenzene
(CASRN 108-90-7)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion

ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR CHLOROBENZENE (CASRN 108-90-7)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

IRIS (U.S. EPA, 2006) lists an RfD of 2×10^{-2} mg/kg-day for chlorobenzene based on a NOAEL of 27 mg/kg-day (adjusted dose of 19.6 mg/kg-day) and LOAEL of 54.5 mg/kg-day (adjusted dose of 39.3 mg/kg-day) for liver histopathology in dogs given gelatin capsules containing chlorobenzene for 13 weeks (Hazleton Laboratories, 1967a). The source document for this assessment is a Drinking Water Criteria Document for chlorobenzene (U.S. EPA, 1986). This RfD is also included on the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). The HEAST (U.S. EPA, 1997) indicates the availability of the chronic RfD on IRIS, but does not list a subchronic RfD. The CARA list (U.S. EPA, 1991a, 1994a) includes a Health Effects Assessment (HEA) for chlorobenzene (U.S. EPA, 1989) that derived a subchronic RfD of 0.3 mg/kg-day and chronic RfD of 0.03 mg/kg-day based on the same 13-week dog study, as well as an Ambient Water Quality Criteria Document (U.S. EPA, 1980) and Health Assessment Document (U.S. EPA, 1985) for chlorinated benzenes, neither of which included derivation of an RfD for chlorobenzene. ATSDR (1990) derived an intermediate duration MRL of 0.4 mg/kg-day for chlorobenzene based on a NOAEL of 60 mg/kg-day and LOAEL of 125 mg/kg-day for liver effects (increases in liver weight and serum biomarkers for hepatotoxicity) in rats and mice administered chlorobenzene for 13 weeks (NTP, 1985).

No RfC is available for chlorobenzene on IRIS (U.S. EPA, 2006). The HEAST (U.S. EPA, 1997) lists a chronic RfC of $2\text{E-}2 \text{ mg/m}^3$ for chlorobenzene based on a subchronic study in rats (Dilley, 1977); however, this RfC was prepared using outdated methodology. A subchronic RfC for chlorobenzene is not reported in the HEAST (U.S. EPA, 1997). The source document for the RfC in the HEAST was the HEA for chlorobenzene (U.S. EPA, 1989). An RfC for chlorobenzene was not included in the Ambient Water Quality Criteria Document (U.S. EPA, 1980) or the Health Assessment Document (U.S. EPA, 1985) for chlorinated benzenes. ATSDR (1990) has not derived inhalation-based Minimal Risk Levels (MRLs) for chlorobenzene. California EPA (OEHHA, 2006) has derived a chronic inhalation REL of 1 mg/m^3 based on the occurrence of liver, kidney, and testicular lesions in a multigeneration study in rats (Nair et al., 1987). ACGIH (2006) has adopted a TLV of 10 ppm (46 mg/m^3) based on liver effects in experimental animals (Dilley, 1977; Nair et al., 1987). The OSHA (2006) PEL is 75 ppm (350 mg/m^3). NIOSH (2006) has not established a REL for chlorobenzene, but has questioned whether the OSHA PEL is adequate to protect workers from the recognized health hazards.

The cancer assessment for chlorobenzene on IRIS (U.S. EPA, 2006) includes a classification of Group D, not classifiable as to human carcinogenicity. This classification is based on no human data, inadequate animal data, and predominantly negative genetic toxicity data in bacterial, yeast, and mouse lymphoma cells. A significant positive trend was observed in the incidence of hepatocellular neoplastic nodules in male (but not female) rats administered chlorobenzene by gavage for 103 weeks; no site-specific tumors or neoplastic pathology were observed in similarly-treated mice (NTP, 1985). Quantitative estimates of carcinogenic risk from oral or inhalation exposure were not made.

The toxicity of chlorobenzene was reviewed by WHO (1991). Updated literature searches for additional toxicity data for chlorobenzene were performed for the period from 1988 to June, 2003 in the following databases: TOXLINE (supplemented with NTIS and BIOSIS updates), CANCERLIT, MEDLINE, CCRIS, GENETOX, HSDB, EMIC/EMICBACK, DART/ETICBACK, RTECS, and TSCATS. The above listed documents and literature searches were used to identify relevant studies. Additional literature searches from June 2003 through October 2004 were conducted by NCEA-Cincinnati using MEDLINE, TOXLINE, Chemical and Biological Abstracts databases.

REVIEW OF PERTINENT DATA

Human Studies

Oral Exposure. No relevant data were located regarding the toxicity of chlorobenzene to humans following oral exposure.

Inhalation Exposure. Five human inhalation studies (Rosenbaum et al., 1947; Tarkhova, 1965; Ogata et al., 1991; Girard et al., 1969; and Syrovadko and Malysheva, 1977) were located. The Tarkhova (1965) and Ogata et al. (1991) studies are acute exposure studies and Rosenbaum et al. (1947), Girard et al. (1969), and Syrovadko and Malysheva (1977) are occupational exposure studies.

In a biological monitoring study conducted by Ogata et al. (1991), 4 humans were exposed once to 60.2 ppm (277 mg/m³) chlorobenzene for 3 hours in the morning and 4 hours in the afternoon, with a 1 hour break between the morning and afternoon exposure sessions. All of the subjects complained of a sensation of a disagreeable odor and of drowsiness, three of a heavy feeling in the head and/or headache, two of throbbing pain in the eyes, and one complained of a sore throat. The authors did not report the incidence of these effects in the control group, thus the significance of the reported symptoms is not known. A significant decrease, as compared to a non-exposed control group, in mean flicker-fusion value was observed (no further details on the control group were given). No significant alterations in pulse rate or systolic or diastolic blood pressure were found.

Tarkhova (1965) exposed 4 subjects to 0.1, 0.2, and 0.3 mg/m³ of chlorobenzene (0.02, 0.04, and 0.07 ppm) and measured changes in electroencephalographic (EEG) patterns in response to light flashes. All subjects were exposed to all three concentrations, but the author did not indicate how much time was allowed for recovery or the order of the exposures. It appears that the experiment was repeated at least "three times during three days for each subject". The subjects were exposed to chlorobenzene for 2½ minutes in each session. The exposure period was preceded by a 3 minute control period. No effects were observed at the 0.1 mg/m³ concentration. A response was observed in 2/4 subjects at 0.2 mg/m³ and in 3/4 subjects at 0.3 mg/m³.

Several occupational exposure studies suggest a neurotoxic effect in workers exposed to chlorobenzene; however, the results do not allow for a definitive conclusion because workers were exposed to other chemicals in addition to chlorobenzene. Rosenbaum et al. (1947; as reviewed by U.S. EPA, 1985 and ATSDR, 1990) examined 28 factory workers intermittently exposed to chlorobenzene for 1-2 years. Exposure concentrations were not reported. Headaches and signs of somnolence and dyspepsia were common among the workers. Tingling, numbness, and stiffness of the extremities and hyperesthesia of the hands were observed in 8 of the 28 workers and spastic contractions of the finger muscles were observed in 9 of 28 workers. Without comparative data from non-exposed workers, it is not clear that these symptoms were caused by chlorobenzene exposure. Girard et al. (1969) reported anemia and symptoms of central nervous system effects (headaches, numbness, and lethargy) and eye and respiratory tract irritation in workers exposed to chlorobenzene at unspecified concentrations. These workers, however, were also exposed to other unspecified chemicals in addition to chlorobenzene. Increased number of birth anomalies and hormonal disturbances were associated with occupational exposure of chlorobenzene and tricresol in female workers (Syrovadko and Malysheva, 1977). However, it is not possible to attribute these effects to chlorobenzene exposure because workers were exposed to tricresol in addition to chlorobenzene.

Overall, the human data suggest that chlorobenzene may affect the nervous system. However, none of the human data are adequate for use in risk assessment either because the effects cannot be definitively attributed to chlorobenzene exposure or because only acute exposures were used.

Animal Studies

Oral Exposure. Subchronic oral studies in dogs (Hazleton Laboratories, 1967a), rats (Hazleton Laboratories, 1967b; NTP, 1985; Irish, 1963; Varshavskaya, 1967), and mice (NTP, 1985), chronic studies in rats and mice (NTP, 1985), and an oral developmental toxicity study in rats (IBT, 1977) were located. These studies are described below.

Groups of 4 male and 4 female young adult beagle dogs were treated with 0, 0.025, 0.05, or 0.250 mL/kg (0, 27.5, 55.0, 275 mg/kg-day using a specific gravity of 1.1) of pure chlorobenzene via capsule 5 days/week for 13 weeks (duration-adjusted doses of 0, 19.6, 39.3 or 196.4 mg/kg-day) (Hazleton Laboratories, 1967a). The dogs were observed daily for appearance and behavior, and body weight and food consumption were determined weekly. Hematology, serum chemistry, and urine analyses were performed after 1 month of treatment and again after 3 months. The dogs were sacrificed after 3 months. All dogs, including those that died during the study, were examined for gross pathology. Organ weights were determined at necropsy. Histological examination was performed for 20 organs (brain, pituitary, thyroid, lung, heart, liver, gallbladder, spleen, kidney, adrenal, stomach, pancreas, duodenum, jejunum, ileum, colon, urinary bladder, ovaries, bone, and bone marrow) in the control and high-dose dogs, but only suspected target organs were examined in the low- and mid-dose dogs.

Four of the 8 high-dose dogs (2 males and 2 females) died or were sacrificed in moribund condition within the first 5 weeks of the study (Hazleton Laboratories, 1967a). Death was preceded by loss of appetite, weight loss, inactivity, and coma. High-dose dogs that survived had reduced appetite and loss of weight over the first 5-6 weeks of the study, but appetite returned and body weight held steady over the remainder of the experiment. Terminal weight loss in these dogs ranged from 0.7 to 2.0 kg. A number of changes in blood and urine parameters were observed in dogs from the high-dose group, including low blood sugar, high circulating levels of immature leukocytes, increased urinary concentrations of acetone and bilirubin, and slight-to-marked increases in serum alkaline phosphatase, alanine aminotransferase, bilirubin, and cholesterol. Gross pathology in high-dose dogs included grey-yellow discoloration of the hepatic parenchyma, distended gallbladder, and red discoloration of the renal medulla. Increases in relative weight of the liver, kidney, adrenals, heart, and thyroid were observed among high-dose dogs, reflecting the poor physical condition of dogs in this group.

Histopathological examination of high-dose dogs revealed moderate-to-severe vacuolation, formation of fatty cysts and bile stasis in the liver, glomerular swelling and swelling and vacuolation of tubular epithelium in the kidney, variations in mucus content of the gastrointestinal mucosa, and leukocytosis and moderate-to-high cellularity in the bone marrow (Hazleton Laboratories, 1967a). Incidence data for the liver and kidney lesions are reported in Table 1. Although the small group sizes in this study limit the power of statistical tests to detect changes, statistically significant increases were shown for several of the liver lesions in the high-dose group (males and females combined, Fisher exact test conducted for this review). No liver or kidney lesions were observed in control animals. Histopathological changes in the liver and kidney were the only effects observed in mid-dose dogs. These changes included slight bile duct proliferation, slight swelling and vacuolation and leukocytic infiltration in the liver, and swelling of tubular epithelium and variations in cellularity in the kidney. No effects of any type were

Table 1. Incidence of Liver and Kidney Lesions in Male and Female Dogs (Combined) Administered Chlorobenzene Daily for 13 Weeks^a (Hazleton Laboratories, 1967a)

Organ, Lesion	Dose (mg/kg-day)			
	0	19.6	39.3	196.4
Liver, bile stasis	0/8	0/8	0/8	4/8
Liver, pigment deposition	0/8	0/8	0/8	3/8
Liver, centrilobular degeneration	0/8	0/8	0/8	8/8 ^b
Liver, vacuolation	0/8	0/8	1/8	6/8 ^b
Liver, cytologic changes	0/8	0/8	1/8	4/8
Liver, bile duct hyperplasia	0/8	0/8	3/8	7/8 ^b
Kidney, tubular dilation	0/8	0/8	2/8	4/8
Kidney, proximal convoluted tubule swelling	0/8	0/8	0/8	2/8
Kidney, proximal convoluted tubule vacuolation	0/8	0/8	1/8	4/8
Kidney, tubule epithelial degeneration	0/8	0/8	1/8	4/8
Kidney, terminal proximal tubule vacuolation	0/8	1/8	0/8	3/8
Kidney, epithelial pigment deposition	0/8	0/8	0/8	3/8

^a Data reported as number of animals observed with the lesion/total number of animals in the dose group

^b Incidence significantly greater than controls using the Fisher exact test ($p < 0.05$) performed for this review

observed in low-dose dogs. This study, therefore, identified a LOAEL of 39.3 mg/kg-day for liver and kidney effects (histopathological changes) and a NOAEL of 19.6 mg/kg-day.

In a companion study to Hazleton Laboratories (1967a), Charles River CD rats (18/sex/group) were given 12.5, 50, 100, or 250 mg/kg-day of pure chlorobenzene by gavage in corn oil, daily for 93-99 days (Hazleton Laboratories, 1967b; Knapp et al., 1971). An additional group of 18 males and 18 females served as an untreated control group. The rats were observed daily for appearance and behavior. Rats in the test groups were weighed daily, while those in the control group were weighed weekly. Food consumption was determined weekly. Hematology, clinical chemistry and urine analyses were performed at 30 and 90 days using 5 males and 5 females from each group. Sacrifice was performed after 93-99 days of treatment. All animals, even those that died during the study, received a gross necropsy. Organ weights were determined at necropsy. Histopathological examination (on 5 males and 5 females from each group) included 17 organs (brain, pituitary, thyroid, lung, heart, liver, spleen, kidney, adrenal, stomach, pancreas, intestines, urinary bladder, gonads, femur, and bone marrow) in the high-dose and control groups, but only the thyroid, heart, liver, kidney and adrenals were examined from the other groups. Although a few deaths occurred during the course of the study, there was no clear relationship between treatment and mortality. There was a statistically significant decrease in body weight gain among high-dose males (terminal body weight reduced approximately 7%), but growth was not affected in other groups. Food consumption did not differ from controls. The only clinical sign clearly related to treatment was salivation following dosing throughout the first week of the study. Salivation generally occurred in about half of the rats exposed to 50 mg/kg-day, a majority of those exposed to 100 mg/kg-day and all of those exposed to 250 mg/kg-day. Hematology, clinical chemistry and urinalysis results were unremarkable. The only gross pathological observation of interest was a high incidence of mottled and discolored livers in rats exposed to 50, 100, or 250 mg/kg-day, that did not, however, increase in incidence or intensity as dose increased from 50 to 250 mg/kg-day. Absolute and relative liver weights were significantly increased in females exposed to 100 or 250 mg/kg-day and males exposed to 250 mg/kg-day. Absolute and relative kidney weights were also significantly increased at these doses. Histopathological examination failed to detect any compound-related effects in rats of any dose group. NOAEL and LOAEL values of 50 and 100 mg/kg-day, respectively, may be derived from this study based on weight increases in the liver and kidney. Although the organ weight increases were not accompanied by histopathological changes or other clear indicators of toxicity, the results of the companion study on dogs (Hazleton Laboratories, 1967a) showed that these organs are targets of chlorobenzene toxicity.

Supporting data also come from subchronic and chronic studies in rats and mice conducted by NTP (1985). In the subchronic studies, groups of 10 F344/N rats and 10 B6C3F1 mice of each sex were given chlorobenzene at 0, 60, 125, 250, 500, or 750 mg/kg-day, 5 days/week for 13 weeks by gavage in corn oil. Duration-adjusted doses were 0, 43, 89, 179, 357 or 536 mg/kg-day, respectively. Clinical signs of toxicity were noted daily and body weights were determined weekly. Urine samples for analysis were obtained during the 13th week of treatment. Blood samples for hematology and clinical chemistry analyses were collected prior to sacrifice. The major organs were weighed at necropsy. Comprehensive histopathological examinations were given to rats from the two highest dose groups and mice from the three highest dose groups, as well as controls. Only suspected target organs were examined

microscopically in the other groups. Chlorobenzene produced death in rats exposed to 500 mg/kg-day (4/10 males and 3/10 females) and 750 mg/kg-day (9/10 males and 8/10 females). Body weight gain was reduced about 20% in both males and females from these groups. The most frequent histopathological lesions in these groups were moderate centrilobular hepatocellular necrosis, mild-to-moderate nephrosis (characterized by degeneration and necrosis of the proximal tubule) and minimal-to-moderate myeloid depletion of the bone marrow. Other lesions observed were hepatic degeneration and lymphoid depletions of the thymus and spleen. Further effects observed in rats from these dose groups included decreased white blood cell count, increased reticulocytes, increased serum alkaline phosphatase and gamma glutamyl transpeptidase, increased urinary output, increased urinary excretion of uroporphyrin and coproporphyrin, increased absolute and/or relative liver and kidney weights and decreased absolute and relative spleen weight. Effects in rats exposed to 250 mg/kg-day included reduced body weight gain (>20%, males only), increased absolute and relative liver weight, decreased absolute and relative spleen weight and a few observations of minimal hepatic necrosis and nephropathy. The only effects at lower doses were increased absolute and relative liver weight in females exposed to 125 mg/kg-day and decreased absolute and relative spleen weight in males exposed to 60 or 125 mg/kg-day. Taken together, the liver was identified as the most sensitive target organ. Increased liver weight was observed at ≥ 125 mg/kg-day, and hepatic necrosis occurred at ≥ 250 mg/kg-day. Although spleen weights were decreased at all doses, microscopic lesions (lymphoid depletion) were only observed at the high dose (750 mg/kg-day). Therefore, these results suggest a NOAEL and LOAEL in rats of 60 and 125 mg/kg-day, respectively (43 and 89 mg/kg-day, respectively, when adjusted for a 5 day/week dosing schedule).

Results in mice were similar in pattern to those in rats, although mice appeared to be more sensitive to chlorobenzene toxicity, as indicated by an increase in mortality in this species at 250 mg/kg-day (5/9 males and 4/10 females) and above (37/40 mice) (NTP, 1985). Body weight gain was reduced 50-80% in these groups. Histopathological lesions were generally limited in occurrence to these same dose groups; lesions included severe hepatic necrosis, moderate renal tubular necrosis, myeloid depletion of the spleen and bone marrow, lymphoid depletion of the spleen and thymus, and necrosis of the thymus. Absolute and relative liver weights were significantly increased in surviving males and females from these groups. Other changes in these dose groups were increased urinary output and increased urinary excretion of coproporphyrins. The only effect in mice exposed to 125 mg/kg was significantly increased absolute and relative liver weight in males. Based on liver toxicity in mice, NOAEL and LOAEL values of 60 and 125 mg/kg-day (duration adjusted doses of 43 and 89 mg/kg-day) can be derived.

In the chronic studies, groups of 50 rats of each sex and 50 female mice were administered chlorobenzene by gavage in corn oil at 0, 60 or 120 mg/kg-day 5 days/week for 103 weeks (NTP, 1985). Duration-adjusted doses were 0, 43, and 86 mg/kg-day, respectively. Groups of 50 male mice were similarly treated with 0, 30 or 60 mg/kg-day (duration adjusted doses of 0, 21, 43 mg/kg-day, respectively). Survival was significantly reduced in male rats in the 120 mg/kg group, but not in lower dose male rats or female rats. Body weight gain was not affected in rats of any group. From the original microscopic examination, there appeared to be a slightly increased incidence of hepatic necrosis in treated rats of both sexes (males at 60 mg/kg and females at 120 mg/kg), but a second independent review did not support these findings.

Other than effects on immune system at substantially high doses, no other chemical-related non-carcinogenic effects were identified in the rats. In mice, survival was marginally reduced in males at 30 and 60 mg/kg; however, survival trend did not follow a dose-response relationship and no effect was noted in females. Body weight gain was similar in treated and control mice and no treatment-related non-neoplastic lesions were identified.

Irish (1963) briefly reported the results of an unpublished Dow Chemical study, in which rats were treated orally with chlorobenzene 5 days per week for approximately 6 months. Doses of 144 and 288 mg/kg-day (duration adjusted doses of 103 and 206 mg/kg-day) produced significant increases in liver and kidney weight and slight liver pathology. No effects were detected in rats treated with 14.4 mg/kg-day (duration adjusted dose of 10.3 mg/kg-day). Further details regarding this study were not provided.

In contrast to the results of the studies described above, toxicity was reported at much lower doses by Varshavskaya (1967). Groups of 7 male albino rats weighing 180-200 g were treated with 0, 0.001, 0.01 or 0.1 mg/kg-day of chlorobenzene in sunflower oil by stomach tube for 9 months. Effects reported at 0.1 mg/kg-day included inhibition of higher nervous system activity (i.e., prolonged formation and accelerated loss of conditioned reflexes), a statistically significant inhibition of erythropoiesis (i.e., decreased red blood cell count and hemoglobin), increased serum alkaline phosphatase and aminotransferase levels, and immune system effects (increased leukocytes and gamma globulin). Many of these endpoints were also marginally affected by exposure to 0.01 mg/kg-day. No effects were reported at 0.001 mg/kg-day. Although some of the effects reported in this study are consistent with those observed in other studies, the effective doses are much lower. Varshavskaya (1967) also reports effects for o-dichlorobenzene that are over 3 orders of magnitude lower than other published values. Therefore, U.S. EPA (1980, 1985) considered the results of this study to be questionable.

Chlorobenzene was the subject of a developmental toxicity study in rats (IBT, 1977). Pregnant Charles River albino rats (20-22 per dose) were administered chlorobenzene at 100 or 300 mg/kg-day on gestation days 6-15 via oral gavage. Maternal body weight, mortality, and clinical signs of toxicity were recorded at regular intervals throughout exposure. All dams were sacrificed on gestation day 20 and were administered via Caesarian section. Implantation sites and the number of corpora lutea were determined, and the number of viable fetuses was recorded. All fetuses were removed from the uterus, weighed, and examined for external malformations. Two-thirds of the fetuses were examined for skeletal effects; the remaining fetuses were evaluated for internal development. No treatment-related effects were noted at any dose; however, the study did not test up to maternally toxic doses. The results of this study do not rule out developmental effects at high doses, but indicate that developmental toxicity is not likely a sensitive toxicological endpoint for chlorobenzene toxicity.

Inhalation Exposure. Several studies have examined the subchronic toxicity of inhaled chlorobenzene in animals (IBT, 1979; Roloff, 1980; Dilley, 1977; Irish, 1963; Zub, 1978). John et al. (1984) and Nair et al. (1987) have examined the developmental and reproductive toxicity, respectively, of inhaled chlorobenzene.

In a study conducted by IBT (1979), groups of male and female rats (15/sex/group) and beagle dogs (4/sex/group) were exposed to 0, 0.76, 1.47, or 2 mg/L (0, 760, 1470, or 2000 mg/m³) of chlorobenzene 6 hours/day, 5 days/week for 90 days (62 exposure days). Controls were exposed to “clean air.” All animals were observed for mortality and clinical signs of toxicity daily throughout the exposure period, and body weights were recorded weekly. Blood was taken from all surviving dogs at Day 28 (blood was taken from several dogs earlier than Day 28 because they were expected to be sacrificed moribund prior to the bleed) and from 5 control and high-concentration rats per sex at Days 39 and 91. Hematology, clinical chemistry, and urinalysis examinations were conducted at each bleed. At scheduled sacrifice, all animals were subjected to a gross pathology evaluation, the adrenal glands (dogs only), brain (cerebrum, cerebellum, and pons), lungs, pancreas, pituitary gland (dogs only), spleen, and thyroid gland (dogs only) were weighed (absolute weights and organ weight relative to the brain and terminal body weight were determined), and 29-32 tissues were microscopically examined from the control and high-concentration groups. Tissues from low- and mid-concentration animals were examined only if “significant pathologic findings” were observed at the high concentration.

No effects on rats were observed for any of the parameters evaluated (IBT, 1979). In dogs, however, a number of potential treatment-related effects were observed. An apparent concentration-related increase in mortality was observed. Mortality rates in dogs exposed at 0, 760, 1470, and 2000 mg/m³, respectively, were 0/4, 0/4, 1/4, and 2/4 in males and 0/4, 0/4, 1/4, and 3/4 in females. Hypoactivity was observed in 0/4, 1/4, and 4/4 dogs at the low-, mid-, and high-concentrations, respectively, in both males and females (control incidences were not reported). Conjunctivitis occurred at the same incidence rates in both males and females. Also, one high-concentration female dog was observed with glazed eyes. There were no clear effects on body weight, although final mean body weights of high-concentration dogs were less than controls. No chlorobenzene-related alterations in hematological, serum clinical chemistry, or urinalysis parameters were observed. A number of statistically significant changes in absolute and relative organ weights were found; however, only pancreas weights of female dogs appeared to show a concentration-response relationship (although the lack of several organ weights from the low- and mid-concentration groups precludes a full evaluation of potential treatment-related effects). The toxicological relevance of the change in pancreas weight, however, is not clear because no microscopic lesions were observed in the pancreas.

Icterus (characterized by yellow discoloration of the aorta) and enlarged hardened livers were observed in dogs that were killed *in extremis* (IBT, 1979). Microscopic lesions were observed in the liver, kidney, testes, and bone marrow in treated dogs. At 2000 mg/m³, slight to moderate vacuolation of the liver (2/4 males, 3/4 females), aplastic bone marrow (2/4 males, 3/4 females), epithelial cytoplasmic vacuolation in the kidneys (1/4 males, 3/4 females), and atrophy of the seminiferous epithelium in the testes (2/4 males) were observed. At 1470 mg/m³, vacuolation of the liver (1/4 males) and juvenile testes (1/4 males) were observed. These lesions were not seen in controls. No tissues from low-concentration males or females were microscopically examined. This study was not peer-reviewed, and statements from the researchers highlighted that only a limited quality assurance review was given to this report. Therefore, reliable NOAEL or LOAEL values cannot be derived from this study. However, the study does provide suggestive evidence that the liver, kidneys, bone marrow, and testes may be target organs for chlorobenzene in dogs.

As a follow-up study, Roloff (1980) exposed beagle dogs (6 per sex and concentration) to chlorobenzene (96.5% pure) 6 hours/day, 5 days/week for 6 months at 0, 0.79, 1.59, or 2.06 mg/L (0, 790, 1590, or 2060 mg/m³). Clinical signs of toxicity were recorded at regular intervals during the 6-hour exposure periods, detailed physical examinations were conducted weekly, and body weight was recorded weekly. After six months of exposure, animals were sacrificed, the adrenals, brain, heart, kidney, liver, pituitary, and testes were weighed, and 24 tissues were microscopically examined. A number of hematology, clinical chemistry, and urinalysis parameters were determined twice prior to study initiation, twice during the first four weeks of exposure, monthly thereafter, and at terminal sacrifice.

Body weight, food consumption, and general health of the dogs were unaffected by chlorobenzene exposure (Roloff, 1980). A concentration-related, statistically significant increase in the number of dogs observed to vomit ($p \leq 0.01$) or pass abnormal stools ($p \leq 0.01$) was reported, suggesting gastrointestinal irritation at all concentrations. However, histopathology did not reveal any treatment-related lesions of the GI tract, and other studies have not observed gastrointestinal effects. Therefore, the toxicological significance of this observation is not clear. A statistically significant ($p \leq 0.05$) increase in liver-to-body weight ratio was observed in mid- and high-concentration females and a significant ($p \leq 0.05$) decrease in absolute adrenal weight was observed in the mid- and high-concentration males. Kidney weight was not affected by treatment. In the absence of microscopic lesions in these tissues, the biological significance of the organ weight changes is not clear. Although chlorobenzene exposure has been shown to affect the liver in other studies, only relative weights in females were significantly increased compared with controls and no microscopic lesions were observed in the liver in this study. Also, relative liver weights in females did not show a clear concentration-related increase. Relative liver weights at 0, 790, 1590, and 2000 mg/m³, respectively, were 2.4%, 3.2%, 3.1%, and 3.1%. Therefore, it does not appear that the increase in relative liver weight in female dogs was related to treatment. Statistically significant changes in various clinical chemistry parameters were observed; however, these changes appeared to be random and not related to chlorobenzene exposure. A clear LOAEL was not observed in this study.

Taken together, these 90 day and 6-month studies in dogs resulted in contradictory results and, therefore, do not allow for reliable NOAEL or LOAEL derivations. In one study, (IBT, 1979), an apparent concentration-related increase in mortality and effects on the kidney, liver, and testes were observed. In a follow-up study (Roloff, 1980), however, no effects were observed in dogs at comparable concentrations. Therefore, a clear NOAEL or LOAEL in dogs was not established.

In a study reported by Irish (1963), groups of rats, rabbits, and guinea pigs were exposed to 0, 200, 475, or 1000 ppm (0, 920, 2189, or 4604 mg/m³) of chlorobenzene for 7 hours/day, 5 days/week for 44 days. In the guinea pigs exposed at 4604 mg/m³, increased mortality was observed. Unspecified histological alterations were observed in the liver, kidney, and lungs in exposed animals at 4604 mg/m³ (the study report did not specify which effects were associated with each species tested). At 2189 mg/m³, slight histological alterations were also observed in the liver. No effects were observed at 920 mg/m³. Additional details were not available.

Zub (1978) exposed male and female white Swiss mice (5 per sex and concentration) to chlorobenzene vapors at 100 mg/m³ daily (7 hours per day) for 3 months or 2500 mg/m³ daily for 3 weeks. Additional experimental design parameters were not reported. Five of 10 mice exposed at 2500 mg/m³ died. Loss of appetite, general emaciation, marked somnolence, decreased body weight, fatty degeneration and atrophy in the liver were also observed at 2500 mg/m³. Slight leukopenia and lymphocytosis were the only haematological effects in mice exposed to 100 mg/m³ for 3 months. Although these data support the conclusion that chlorobenzene may affect the liver, the data are limited because sufficient detail on experimental methods and results were not reported in the published article to permit critical evaluation of the study.

Dilley (1977) exposed groups of 32 male Sprague-Dawley rats and 32 male rabbits (strain not specified) to 0, 73, or 248 ppm (0, 336, or 1142 mg/m³, respectively) of chlorobenzene for 7 hours/day, 5 days/week for 24 weeks. Groups of 10 rats and 10 rabbits were killed after 5, 11, or 24 weeks of exposure. Animals were weighed weekly for 5 weeks, every 2 weeks for the next 4 weeks and monthly thereafter. All animals were observed daily for clinical signs of toxicity. The brain, heart, lungs, liver, spleen, kidneys, and gonads were weighed. These tissues and the adrenal glands, bone marrow, eye, skin, and abnormal tissues were microscopically examined. A number of hematology and clinical chemistry parameters were evaluated.

In rats, no deaths, unusual clinical observations or changes in body weight gain were observed (Dilley, 1977). The kidney and liver weights generally increased with increasing concentration (Table 2). Significant increases in absolute and relative liver weights were observed in male rats exposed to 248 ppm for 24 weeks (Table 2) compared with controls. Relative kidney weights were also significantly greater than controls after 24 weeks.

Hematology evaluations found decreased hematocrit and mean corpuscular volume, and increased mean corpuscular hemoglobin concentration, in rats exposed to chlorobenzene at ≥ 73 ppm after 11 weeks of exposure, consistent with microcytic anemia; however, similar effects were not observed at 24 weeks (Dilley, 1977). Therefore, the biological significance of this observation is not clear. The only consistent and significant change in the rat clinical chemistry profile was reduced serum aspartate aminotransferase (AST) activity in the high-dose group at all three sacrifice times. The toxicological significance of this observation is not clear. Histopathology revealed no consistent concentration-related increase in the incidences of any lesions. Chronic respiratory disease was observed in 8-10 rats in all treatment and control groups. It is not known if the chronic respiratory disease made the animals unusually sensitive to the toxicity of chlorobenzene or masked some aspects of chlorobenzene toxicity. Therefore, a NOAEL and LOAEL suitable for RfC derivation cannot be identified from this study. However, the organ weight data provide supportive evidence that the liver and kidneys are possible targets of chlorobenzene toxicity.

Table 2. Selected Organ Weights in Male Rats Exposed to Chlorobenzene via Inhalation for 24 Weeks (Dilley, 1977)

Organ	Absolute or Relative Weight	0 ppm	73 ppm	248 ppm
Liver	Absolute (g)	16±0.8 ^a	18±0.9	21±2.0 ^b
	Relative, body	34±0.6	38±1.0	44±1.9 ^c
	Relative, brain	7.3±0.4	8.0±0.4	9.7±0.8
Kidneys	Absolute (g)	3.5±0.2	3.7±0.1	4.1±0.2
	Relative, body	7.5±0.2	7.9±0.2	8.5±0.2 ^b
	Relative, brain	1.6±0.08	1.6±0.06	1.8±0.08
^a mean ± standard deviation ^b statistically significant (p≤0.05) ^c statistically significant (p≤0.01)				

In rabbits, no treatment-related deaths, unusual clinical observations, or changes in body weight gain were observed (Dilley, 1977). Overall, there were no consistent concentration-related changes in hematology, clinical chemistry, or gross or microscopic lesions. Encephalitozoonosis (caused by *Escherichia cuniculi* infection) and respiratory illness associated with atelectasis and emphysema; lymphocytic foci near bronchi and bronchioles, focal edema and congestion were observed in a number of treated and control animals that may have affected the rabbits' sensitivity to chlorobenzene-induced toxicity. Therefore, these data are not suitable for RfC derivation.

Nair et al. (1987) conducted a two-generation reproductive study in rats. In this study, groups of 30 male and 30 female CD Sprague-Dawley rats were exposed to chlorobenzene (>99.9% pure) in a dynamic air chamber at target concentrations of 0, 50, 150, or 450 ppm (0, 230, 691, or 2072 mg/m³) for 6 hours/day, 7 days/week for 10 weeks before mating, and during mating, gestation, and lactation. The male and female F₀ rats were sacrificed after the lactation period. Groups of 30 male and 30 female F₁ rats were exposed to the same concentrations of chlorobenzene (beginning 1 week post-weaning) for 11 weeks before mating and during mating, gestation, and lactation. The F₁ rats were also sacrificed after the lactation period. The F₂ pups were sacrificed after weaning. Mortality and clinical signs of toxicity were recorded twice each day, detailed physical examinations were conducted weekly, body weights were recorded weekly except that female body weights were also recorded at additional regular intervals throughout gestation and lactation, and food consumption was recorded weekly during the growth period. Complete gross postmortem examinations were conducted on all sacrificed animals. Liver and brain weights of F₀ and F₁ adults were recorded. Liver, kidneys, pituitary gland, and reproductive organs (males: testes, epididymides, seminal vesicle, and prostate; females: vagina, uterus, and ovaries) were examined microscopically for all F₀ and F₁ adult animals in the control and high-concentration groups. Liver, kidneys, and testes of male rats in the low- and mid-

concentration groups were microscopically examined. Hematology or clinical chemistry parameters were not evaluated.

No deaths were observed in the F₀ or F₁ groups, and no significant alterations in body weight gain were observed (Nair et al., 1987). No apparent alterations in the mating, pregnancy (number of pregnant females/number mated), fertility, pup viability, pup survival, or litter survival indices were observed in the F₀ or F₁ rats. Absolute and relative liver weights were clearly and significantly increased in F₀ and F₁ male rats exposed to ≥ 150 ppm and F₀ and F₁ female rats exposed to ≥ 450 ppm (Table 3). Much smaller, but still statistically significant, increases in relative liver weight at lower doses were consistent with the observed trend, but do not themselves indicate a toxicologically significant effect at the lower doses. Histopathology examinations identified the liver, kidneys, and testes as target organs for chlorobenzene in male rats. Table 4 shows incidence data reported by the investigators and the results of statistical tests conducted for this review (statistical tests of the incidence data were not performed by the original investigators). In the liver, the incidence of centrilobular hepatocellular hypertrophy was significantly increased in the 150 and 450 ppm F₀ males in a dose-related manner, and marginally increased in the 450 ppm F₁ males. In the kidneys, significant increases in the incidences of tubular dilation, chronic interstitial nephritis, and foci of regenerative epithelium were observed at 150 and 450 ppm in the F₀ males, but primarily at 450 ppm in the F₁ males. The incidence of small and flaccid testes was significantly increased in the F₁ males at 450 ppm, and was also observed in both F₀ and F₁ males at 150 ppm. Degeneration of the testicular germinal epithelium was seen in F₀ and F₁ males at 150 and 450 ppm, and appears to have been treatment-related. Although incidence levels were low at 150 ppm and just approached statistical significance at 450 ppm, the lesion was graded as moderate or severe in 1 F₀ and 2 F₁ males at 150 ppm and in 3 F₀ and 5 F₁ males at 450 ppm. The two observations of this lesion in controls were both graded as minimal. No concentration-related microscopic lesions were observed in female rats.

The kidney lesions observed in this study included tubular dilation, chronic interstitial nephritis, and foci of regenerative epithelium (Nair et al, 1987). Because the lesions observed in this study only occurred in male rats and are consistent with those typical of alpha-_{2u}-globulin accumulation (U.S. EPA, 1991b), and because other chlorobenzene derivatives have been shown to cause alpha-_{2u}-globulin accumulation (WHO, 1991), it is possible that the kidney effects observed in this study may not be relevant to human health risk assessment. However, there is insufficient evidence to attribute the kidney effects observed in this study to alpha-_{2u}-globulin accumulation. The presence of alpha-_{2u}-globulin was not tested for in the Nair et al. (1987) study, and other studies have demonstrated the occurrence of kidney effects in animals other than male rats. Hazleton Laboratories (1967a) reported kidney effects in orally treated dogs (including tubule dilation, vacuolation, and leukocytic infiltration), and NTP (1985) reported kidney effects in male and female mice (tubular necrosis) and male and female rats (degeneration and necrosis of the proximal tubule) orally administered chlorobenzene in subchronic studies. It is possible that the absence of kidney lesions in female rats in the Nair et al. (1987) study was due to generally lower sensitivity of the females to chlorobenzene toxicity, as liver lesions were also observed only in males in this study. Therefore, there is insufficient evidence to attribute the kidney lesions observed in this study to alpha-_{2u}-globulin accumulation, and the kidney

Table 3. Mean Absolute and Relative Liver Weights of F₀ and F₁ Rats Exposed to Chlorobenzene Vapor (Nair et al., 1987)				
Concentration (ppm)	Males		Females	
	Absolute Liver Weight (g)	Relative Liver Weight (g)	Absolute Liver Weight (g)	Relative Liver Weight (g)
F ₀ Animals				
0	19.3±2.2 ^a	3.6±0.35	11.5±1.3	3.8±0.30
50	19.0±3.1	3.6±0.34	12.0±1.3	3.9±0.23
150	21.5±2.3 ^c	4.1±0.30 ^c	12.1±1.1	4.0±0.21 ^b
450	21.9±3.8 ^c	4.1±0.61 ^c	13.3±1.5 ^c	4.4±0.33 ^c
F ₁ Animals				
0	18.3±2.2	3.5±0.32	12.4±2.3	4.2±0.60
50	19.5±2.6	3.7±0.36 ^b	12.7±1.6	4.2±0.35
150	21.7±3.5 ^c	4.2±0.46 ^c	13.1±1.6	4.4±0.41
450	23.4±4.1 ^c	4.4±0.40 ^c	14.0±2.0 ^c	4.6±0.37 ^c
^a mean ± standard deviation ^b statistically significant (p≤0.05) ^c statistically significant (p≤0.01)				

Table 4. Incidences of Liver, Kidney, and Testicular Lesions Observed in Adult Male Rats Exposed to Chlorobenzene via Inhalation in a 2-Generation Reproductive Toxicity Study (Nair et al., 1987)^a					
Organ, Lesion	Generation	Concentration (ppm)			
		0	50	150	450
Liver, hepatocellular hypertrophy	F ₀	0/30 ^b	0/30	5/30 ^c	14/30 ^d
	F ₁	2/30	0/30	3/30	7/30
Kidney, tubular dilation/eosinophilic material (unilateral or bilateral)	F ₀	0/30	4/30	6/30 ^c	18/30 ^d
	F ₁	8/30	7/30	14/30	22/30 ^d
Kidney, chronic interstitial nephritis (unilateral or bilateral)	F ₀	1/30	2/30	7/30 ^c	10/30 ^d
	F ₁	1/30	3/30	7/30 ^c	11/30 ^d
Kidney, foci of regenerative epithelium (unilateral or bilateral)	F ₀	0/30	1/30	5/30 ^c	8/30 ^d
	F ₁	1/30	0/30	5/30	11/30 ^d
Testes, small and flaccid	F ₀	0/30	0/30	1/30	3/30
	F ₁	0/30	0/30	1/30	5/30 ^c
Testes, degeneration of germinal epithelium	F ₀	1/30	0/30	2/30	6/30
	F ₁	1/30	0/30	3/30	6/30
^a statistical analysis (Fisher Exact test) performed for this review and not by original investigators ^b number of animals with lesion/total number of animals exposed ^c statistically significant ($p \leq 0.05$) ^d statistically significant ($p \leq 0.01$)					

effects are considered relevant to human health risk assessment until conclusive evidence is obtained indicating otherwise.

Nair et al. (1987) demonstrated dose-related effects on the liver, kidney, and testes. Male rats were more sensitive than females. In all three organs, there was some evidence for an effect at 150 ppm, and more clear evidence at 450 ppm. In the liver, significant increases in liver weight and the incidence of hepatocellular hypertrophy were seen at 150 and 450 ppm. In the kidneys, the incidences of tubular dilation, chronic interstitial nephritis, and foci of regenerative epithelium were increased at both 150 and 450 ppm. The kidney effects are considered relevant to human health risk assessment, as previously discussed. In the testes, degeneration of the germinal epithelium was not statistically increased in incidence even in the 450 ppm group, but appeared to be related to treatment in both the 150 and 450 ppm groups based on severity of the lesions observed. Although the testes appeared to be a target for chlorobenzene, reproductive

performance was not affected at any exposure level. Based on these endpoints, this study identified a LOAEL of 150 ppm (691 mg/m³) and NOAEL of 50 ppm (230 mg/m³).

Chlorobenzene was the subject of several developmental toxicity studies. John et al. (1984) exposed groups of 32-33 pregnant Fischer 344 rats to 0, 75, 210, or 590 ppm (0, 345, 967, or 2716 mg/m³) of chlorobenzene 6 hours/day on gestation days (GDs) 6-15. The dams were sacrificed on GD 21. At necropsy, the uterine horns were examined for (1) number and position of fetuses; (2) number of live and dead fetuses; (3) number and position of resorption sites; (4) number of corpora lutea; (5) the sex, body weight, and crown-rump length of each fetus; and (6) gross external abnormalities. One half of each litter was examined under a dissecting microscope for soft tissue alterations, and the heads of these animals were also examined by sectioning. All fetuses were examined for skeletal alterations.

No maternal deaths or changes in general appearance or behavior were observed in the chlorobenzene-exposed rats (John et al., 1984). In dams exposed to 590 ppm, significant decreases in body weight gain were observed on GDs 6-8 (Table 5); however, weight gains over subsequent intervals and total weight gains over GDs 6-20 were not significantly affected. Significant increases in absolute and relative liver weights were observed at 590 ppm (Table 5). Mean litter size and incidence of resorptions were not affected by chlorobenzene exposure, and no alterations in the incidence of malformations were observed in the rat fetuses. The incidences of some minor skeletal variations were altered in some groups, but no consistent concentration-related changes were observed. Therefore, chlorobenzene was not considered a developmental toxicant in this study. A maternal NOAEL and LOAEL of 210 ppm and 590 ppm, respectively, was identified from this study based on increased maternal liver weight and decreased body weight. The developmental NOAEL was 590 ppm, the highest concentration tested.

Table 5. Body and Liver Weights of Female Rats Exposed to Chlorobenzene (John et al., 1984)			
Concentration (ppm)	Body weight gain GD 6-8 ^a (g)	Liver weight (absolute) (g)	Liver weight (relative)
0	3 ± 2 ^b	9.8 ± 1.1	3.8 ± 0.28
75	4 ± 3	10.0 ± 0.81	3.9 ± 0.28
210	2 ± 3	10.1 ± 0.54	3.9 ± 0.21
590	-2 ± 5 ^c	11.0 ± 0.83 ^c	4.3 ± 0.42 ^c
^a body weight gains at other time periods were comparable to controls and are not reported ^b mean ± standard deviation ^c statistically significant (p ≤ 0.05)			

John et al. (1984) conducted two developmental toxicity studies in rabbits. In the first study, groups of 30 pregnant New Zealand white rabbits were exposed to 0, 75, 210, or 590 ppm (0, 345, 967, or 2716 mg/m³) of chlorobenzene for 6 hours/day on GDs 6-18 and sacrificed on GD 29. Other details of the protocol were the same as described for rats (John et al. 1984). No

effect on body weight or weight gain was observed in the does. Absolute and relative liver weights in the does were reported to be significantly increased at 210 and 590 ppm, but the data were not shown. No effects on reproductive or fetal parameters were found. There was a statistically significant ($p \leq 0.05$) increase in the incidence of fetuses with extra rib at 590 ppm. The number of litters affected, however, was comparable to controls (Table 6). There were also several observations of historically rare malformations (head/facial anomalies, heart defects, spina bifida, acephaly) in treated rabbits that were not seen in controls (Table 6). Because it was not clear that any of these effects were directly related to chlorobenzene treatment, a second experiment was conducted in rabbits at 0, 10, 30, 75, and 590 ppm.

Table 6. Fetal Alterations in Chlorobenzene Exposed Rabbits - Experiment 1 (John et al., 1984)					
Concentration (ppm)	Extra Rib	Head/Facial Anomalies	Heart Anomalies	Spina Bifida	Acephaly
0	79 (24) ^a	0	0	0	0
75	68 (19)	1 (1)	0	0	0
210	92 (33)	0	1 (1)	1 (1)	0
590	113 ^b (26)	1 (1)	2 (2)	1 (1)	1 (1)
^a number of fetuses affected (number of litters affected in parentheses) ^b statistically significant ($p \leq 0.05$)					

In the second study (John et al., 1984), groups of 30-32 pregnant New Zealand White rabbits were exposed to 0, 10, 30, 75, or 590 ppm (0, 46, 138, 345, or 2716 mg/m³) of chlorobenzene 6 hours/day on GDs 6-18. An increase in maternal liver weight was observed in the 590 ppm group. No significant alterations in the number of litters, number of fetuses per litter, or the number of implantations resorbed were observed; however, there was a significant increase in the number of litters with resorptions at 590 ppm. This observation, however, was not considered to be related to chlorobenzene exposure by the researchers because the incidence was within the range of historical controls (details on the historical controls were not reported) and because this effect was not observed in the first rabbit study. The incidence of malformations was not altered in the chlorobenzene-exposed groups. The malformations observed in the first rabbit study were either not observed at all in the second study or were seen at comparable incidence in the control group. An increased incidence of fetuses with extra ribs was found at 10 ppm, but the number of affected litters was similar to controls. No increases in extra ribs were seen at ≥ 30 ppm. Overall, no consistent developmental effects were observed in the two studies conducted in rabbits. The NOAEL for developmental toxicity was the high concentration of 590 ppm. Increased liver weights were observed at ≥ 210 ppm in maternal animals in the first study; the second study did not test any concentration between 75 and 590 ppm. Therefore, the maternal NOAEL for these studies was 75 ppm (345 mg/m³) and the maternal LOAEL was 210 ppm (967 mg/m³), based on increased liver weights.

Tarkhova (1965) exposed groups of 15 male white rats (strain not specified) to 0, 0.1, or 1.0 mg/m³ (0, 0.02, 0.2 ppm) for an "uninterrupted" 60-day period. No alterations in body weight or appearance were observed. In the 1.0 mg/m³ group, the conduction speeds of nerve impulses to sets of flexor and extensor muscles had changed on day 39. The ratios of chronaxias of the flexor and extensor muscles in the 1 mg/m³ exposed animals were measured every 9-10 days as the experiment progressed. Ninety-nine percent (99%) reliability of changes by comparison to the control were observed starting day 39. A significant increase in blood cholinesterase and changes in the ratio of albumin:α-globulin ratio (direction of the change can not be determined) was also observed in the 1.0 mg/m³ group. The rise in blood cholinesterase activity was observed in the 1 mg/m³ exposed groups of animals on the 36th day of the treatment.

Aranyi et al. (1986) tested the immunotoxicity of a number of potentially hazardous air contaminants, including chlorobenzene. Female CD₁ mice (135/group) 4-5 weeks old were exposed to either 0 or 75 ppm (0 or 345 mg/m³) of chlorobenzene for 3 hours/day for 5 days. The mice were exposed simultaneously to an aerosol of viable *Streptococcus zooepidemicus*, and deaths over a 14-day observation period were recorded. Pulmonary bactericidal activity of *in vivo* alveolar macrophages was also monitored in animals (23/group) simultaneously exposed to ³⁵S-*Klebsiella pneumonia* and either chlorobenzene or air. The ratio of viable bacterial counts to radiolabeled bacteria was used to determine bactericidal activity. Exposure to chlorobenzene resulted in no significant increase in mortality in female CD₁ mice due to *S. zooepidemicus* challenge after 5 days of simultaneous exposure for 3 hours/day, in comparison with filtered-air controls. There was also no evidence of any adverse effect on the bactericidal activity of alveolar macrophages due to chlorobenzene exposure. The data indicate that immunotoxicity is not likely a sensitive toxicological endpoint for chlorobenzene.

DERIVATION OF A PROVISIONAL SUBCHRONIC RfD FOR CHLOROBENZENE

No relevant data were located regarding the subchronic or chronic toxicity of chlorobenzene to humans following oral exposure. Subchronic studies in dogs (Hazleton Laboratories, 1967a), rats (Hazleton Laboratories, 1967a; NTP, 1985; Irish, 1963; Varshavskaya, 1967), and mice (NTP, 1985) and chronic studies in rats and mice (NTP, 1985) were located. Overall, the data indicate that the liver and kidneys are the most sensitive target organs of orally administered chlorobenzene in experimental animals, and that the dog is the most sensitive species evaluated to chlorobenzene toxicity. In dogs, increased incidence of liver and kidney pathology was reported by Hazleton Laboratories (1967a) at ≥39.3 mg/kg-day. Effects on the bone marrow and GI tract were observed at higher chlorobenzene doses. In rodents, increased liver and kidney weights and liver pathology was observed at ≥≈100 mg/kg-day of chlorobenzene (Hazleton Laboratories, 1967b; NTP, 1985; Irish, 1963). The kidney, bone marrow, thymus, and spleen were affected by treatment at higher chlorobenzene doses (NTP, 1985). The available data indicate that the developing fetus is not a sensitive target of orally administered chlorobenzene (IBT, 1977). Although no reproductive toxicity data from oral studies were located, the available inhalation data indicate that reproductive toxicity is not the most sensitive toxicological endpoint for chlorobenzene toxicity (Nair et al., 1987). Effects on immune system tissues were observed in the study conducted by NTP (1985); however, these

effects were only observed at substantially higher doses than those that induced liver toxicity. An inhalation exposure immune function assay indicated that the immune system is not likely a sensitive indicator of chlorobenzene toxicity (Aranyi et al., 1986). No neurotoxicity studies using oral exposure were located. Inhalation data appear to indicate that neurotoxicity data could be a sensitive endpoint of chlorobenzene toxicity in humans (Rosenbaum et al., 1947; Tarkhova, 1965; Ogata et al., 1991; Girard et al., 1969; and Syrovadko and Malysheva, 1977), although none of these studies were sufficient to definitively conclude that chlorobenzene causes adverse effects on the nervous system.

The 13-week study in dogs (Hazleton Laboratories, 1967a) was chosen as the basis for the subchronic RfD because this study demonstrated that the dog is the most sensitive species that has been evaluated in subchronic studies. In this study, chlorobenzene was administered to male and female dogs (4 per sex and dose) in gelatin capsules containing 0, 27.5, 55.0, 275 mg/kg-day of chlorobenzene 5 days per week for 13 weeks (duration-adjusted doses of 0, 19.6, 39.3 or 196.4 mg/kg-day). This study revealed treatment-related effects in the liver, kidneys, GI tract, and bone marrow at 196.4 mg/kg-day. At 39.3 mg/kg-day, effects on the liver (slight bile duct proliferation, slight swelling and vacuolation and leukocytic infiltration) and kidneys (swelling of tubular epithelium and variations in cellularity) were observed. No effects were observed in dogs administered 19.6 mg/kg-day chlorobenzene. Although none of the increased incidences at 39.3 mg/kg-day were significantly greater than controls, the study used only 8 dogs (4 per sex) per dose and the small number of animals resulted in low power of statistical analysis to detect a change. The study did show a clear increase in the incidence and severity of liver and bile duct hyperplasia with increasing dose. Therefore, the marginal increase in liver lesions and bile duct hyperplasia observed at 39.3 mg/kg-day was considered related to chlorobenzene treatment. This study, then, identified a NOAEL of 19.6 mg/kg-day and a LOAEL of 39.3 mg/kg-day for liver and bile duct hyperplasia.

The provisional **subchronic RfD of 7E-2 mg/kg-day** is derived from the NOAEL of 19.6 mg/kg-day by applying an uncertainty factor of 300 (10 to extrapolate from dogs to humans, 10 to protect sensitive subpopulations, and 3 for database deficiencies, including the lack of reproductive and neurological oral toxicity studies), as follows:

$$\begin{aligned} \text{subchronic p-RfD} &= \text{NOAEL} \div \text{UF} \\ &= 19.6 \text{ mg/kg-day} \div 300 \\ &= 0.07 \text{ or } 7\text{E-2 mg/kg-day} \end{aligned}$$

Confidence in the principal study is medium. This study demonstrated a progression of effects with increasing dose, enabling identification of both a NOAEL and a LOAEL. However, the study was limited by small group sizes, lack of statistical analysis and only marginally adequate reporting of results. Confidence in the database is medium. Supporting oral toxicity data are available, but reproductive effects have been studied only by inhalation exposure, and neurotoxicity, which has been identified as a potential effect of chlorobenzene in humans exposed by inhalation, has not been systematically studied by any route. Medium confidence in the p-RfD follows.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfCs FOR CHLOROBENZENE

Human data suggest that the nervous system may be a target for chlorobenzene toxicity (Rosenbaum et al., 1947; Girard et al., 1969; Tarkhova, 1965; Ogata et al., 1991). Headaches and drowsiness have been reported by workers and experimental subjects (Rosenbaum et al., 1947; Girard et al., 1969; Ogata et al., 1991), and tingling, numbness, and stiffness of the extremities have been observed in workers (Rosenbaum et al., 1947). However, none of these studies reported control data for these effects and the workers may have been exposed to other chemicals in addition to chlorobenzene. Thus, the observations may not have been related to chlorobenzene exposure. Tarkhova (1965) found alterations in the EEG pattern in response to rapid light flashes in humans exposed at 0.2 mg/m^3 ; however, the toxicological significance of these alterations is not known. Tarkhova (1965) also reported effects of unclear relevance (changes in the conduction speeds of nerve impulses to sets of flexor and extensor muscles) in rats exposed to chlorobenzene at 1.0 mg/m^3 for 60 days. The reliability of these data is uncertain because these effects have not been confirmed by other studies. Taken together, the data suggest that chlorobenzene may affect the nervous system. However, none of the data are adequate for use in risk assessment, either because the effects cannot be definitively attributed to chlorobenzene exposure, only single exposures were used, or the toxicological relevance of the effects is not clear.

The available animal data indicate that the liver and kidneys are the most sensitive target organs for chlorobenzene toxicity. Liver effects included increased weight, hepatocellular hypertrophy, fatty change, and other unspecified microscopic lesions (IBT, 1979; Nair et al., 1987; Dilley, 1977; Irish, 1963; Zub, 1978). Kidney effects included increased weights, cytoplasmic vacuolation, tubule dilation, inflammation of the interstitial cells, and regeneration of the epithelium in male rats (IBT, 1979; Irish, 1963; Nair et al., 1987; Dilley, 1977). The NOAEL and LOAEL for both liver and kidney effects were 50 and 150 ppm (230 and 691 mg/m^3 , respectively) in the only adequately conducted and reported study (Nair et al., 1987). Kidney lesions have only been reported in male rats (or rats of unspecified sex) in the available inhalation studies, which suggests that the observed kidney effects may be related to alpha- 2_u -globulin accumulation, a male rat-specific effect that is not predictive for health effects in humans (U.S. EPA, 1991b). Such an effect is known for other chlorinated benzene compounds (WHO, 1991). However, there does not appear to be sufficient evidence to attribute the kidney lesions observed by Nair et al. (1987) to alpha- 2_u -globulin accumulation. Although the lesions were consistent with those associated with alpha- 2_u -globulin, Nair et al. (1987) did not test for the presence of alpha- 2_u -globulin directly. Chlorobenzene produced kidney lesions, including tubule dilation, vacuolation, and leukocytic infiltration, in dogs treated by oral exposure (Hazleton Laboratories, 1967a). NTP (1985) reported kidney effects in male and female mice (tubular necrosis) and male and female rats (degeneration and necrosis of the proximal tubule) in oral subchronic studies on chlorobenzene. The absence of kidney lesions in females (Nair et al., 1987) may reflect general lower sensitivity of females to chlorobenzene toxicity, as liver lesions were also observed only in males in this study. For these reasons, there is insufficient evidence to attribute the kidney lesions observed in this study to alpha- 2_u -globulin accumulation, and the kidney effects are considered potentially relevant to human health risk assessment.

The testis was also identified as a target for chlorobenzene in male rats. Possible effects on the testes were observed in male rats exposed to chlorobenzene at 150 or 450 ppm (Nair et al., 1987). However, it does not appear that the testes are as sensitive a target as the liver and kidney because the incidence of the testicular lesions was only marginally increased in rats exposed at chlorobenzene concentrations that induced significant increases in the incidences of animals with microscopic liver and kidney lesions, and because reproductive performance was not affected.

Other studies suggested that the blood may be a potential target for chlorobenzene. Effects on the blood were observed by Zub (1978), who reported slight leukopenia and lymphocytosis in mice exposed to 100 mg/m³ for 3 months. Dilley (1977) reported microcytic anemia in rats exposed at ≥ 336 mg/m³. However, neither of these studies was adequate to base a definitive conclusion regarding effects on the blood, either because sufficient detail was not available to allow for an independent evaluation of study adequacy (Zub, 1978) or because the animals were sick during exposure (Dilley, 1977). Anemia was also reported in workers potentially exposed to unspecified concentrations of chlorobenzene; however, the workers were also exposed to other chemicals (Girard et al., 1969). Clear effects on the blood were not observed in dogs exposed to chlorobenzene for 6 months (IBT, 1979; Roloff, 1980) and blood effects have not been consistently reported in chlorobenzene exposed animals in subchronic studies; therefore, the data suggest that the blood is not likely a sensitive indicator of chlorobenzene toxicity.

Developmental toxicity studies in two species were located, which indicate that chlorobenzene is not a developmental toxicant (John et al., 1984). In a 2-generation reproductive toxicity study in rats (Nair et al., 1987), marginal increases in testicular lesions were associated with chlorobenzene exposure at concentrations that induced significant increases in the incidences of microscopic liver and kidney lesions. Reproductive impairment was not observed at any concentration. Therefore, it does not appear that reproductive toxicity is a sensitive endpoint for chlorobenzene toxicity.

Although a number of subchronic inhalation studies in animals were located (IBT, 1979; Roloff, 1980; Dilley, 1977; Irish, 1963), none of these studies were considered adequate for RfC derivation for the following reasons: a clear LOAEL was not established (combined data from IBT, 1979 and Roloff, 1980); infection occurred in the test animals during exposure (Dilley, 1977); sufficient detail on the experimental design and results were not reported (Irish, 1963; Zub, 1978); or only one concentration was used (Zub, 1978). The only available study suitable for RfC derivation was the 2-generation study conducted by Nair et al. (1987). In this study, Sprague-Dawley rats (30 per sex and dose) were exposed to chlorobenzene (>99% pure) in a dynamic air chamber at target concentrations of 0, 50, 150, or 450 ppm (0, 230, 691, or 2072 mg/m³) for 10 weeks before mating, then during mating, gestation, and lactation. Their offspring (F₁ rats) were exposed for 11 weeks beginning 1 week after weaning. Clear treatment-related effects were observed in the kidneys and liver of chlorobenzene exposed rats, and possible effects on the testes were observed. Kidney effects included increased weights, tubule dilation, inflammation of the interstitial cells, and regeneration of the epithelium in male rats; liver effects included increased organ weight and hepatocellular hypertrophy. The NOAEL and LOAEL for these effects was 50 and 150 ppm (230 and 691 mg/m³, respectively). A marginal increase in the incidence of degeneration of the germinal epithelium was also observed at 150 ppm.

In order to derive the point of departure for derivation of the RfC, the LED₁₀ (lower bound on dose estimated to produce a 10% increase in the extra risk of the modeled effects over background) was estimated for all kidney and liver lesions reported by Nair et al. (1987) using the U.S. EPA (2000) benchmark dose methodology. A 10% response level was modeled, as recommended for dichotomous endpoints by U.S. EPA (2000). The sensitivity of the study does not appear to warrant the use of a different response level (e.g., 1% or 5%). All available models for dichotomous data in the EPA Benchmark Dose Software (version 1.3.2) were fit to the incidence data for all treatment-related kidney and liver lesions observed in Nair et al. (1987) (incidence data reported in Table 7 below). Because each of the lesions were considered potentially relevant in human health risk assessment, the lesion that resulted in the lowest LED₁₀ that was adequately described by modeling was chosen as the point of departure for the RfC. As illustrated in Table 7, renal tubular dilation resulted in the lowest LED₁₀. Tubular dilation can be caused by alpha-_{2u}-globulin accumulation in male rats. However, tubular dilation was observed in dogs orally administered chlorobenzene, and tubular necrosis, vacuolation, and/or regeneration was observed in male and female rats and mice orally administered chlorobenzene for 13 weeks. Therefore, tubular dilation is not necessarily a result of alpha-_{2u}-globulin accumulation and is potentially relevant to human health risk assessment; it was chosen as the point of departure for RfC derivation.

The dichotomous models estimated concentrations between 17 and 125 ppm associated with a 10% extra risk (ED₁₀) for tubular dilation (Table 8). As assessed by Akaike's Information Criterion (AIC), the best fitting models were the gamma, quantal linear, and Weibull models. Each of these models calculated ED₁₀ values of 53.8 ppm and a lower 95% confidence interval (LED₁₀) of 39.7 ppm. Therefore, 39.7 ppm was selected as the point of departure to derive the p-RfC.

The LED₁₀ of 39.7 ppm (183 mg/m³) was converted to a human equivalent concentration using the following equations (U.S. EPA, 1994b):

$$\begin{aligned}\text{LED}_{10 \text{ ADJ}} &= \text{LED}_{10} \times \text{duration adjustment} \\ \text{LED}_{10 \text{ ADJ}} &= 183 \text{ mg/m}^3 \times 6 \text{ hours}/24 \text{ hours} \times 7 \text{ days}/7 \text{ days} \\ \text{LED}_{10 \text{ ADJ}} &= 46 \text{ mg/m}^3\end{aligned}$$

$$\text{LED}_{10 \text{ HEC}} = \text{LED}_{10 \text{ ADJ}} \times L_R/L_H$$

where,

$$\begin{aligned}L_R/L_H &= \text{rat to human blood:air partition coefficient ratio} \\ L_R/L_H &= \text{default ratio of 1, because } L_R (59.4; \text{Gargas et al., 1989}) \text{ is greater than } L_H \\ &\quad (30.0; \text{Gargas et al., 1989})\end{aligned}$$

$$\begin{aligned}\text{LED}_{10 \text{ HEC}} &= 46 \text{ mg/m}^3 \times 1 \\ \text{LED}_{10 \text{ HEC}} &= 46 \text{ mg/m}^3\end{aligned}$$

Table 7. LED₁₀ Values Calculated for Chlorobenzene Based on Liver and Kidney Lesions in Nair et al. (1987)^a

Organ, Lesion	Generation	Concentration (ppm)				LED ₁₀ (ppm)
		0	50	150	450	
Liver, hepatocellular hypertrophy	F ₀	0/30 ^b	0/30	5/30 ^c	14/30 ^d	97.3
	F ₁	2/30	0/30	3/30	7/30	NA
Kidney, tubular dilation/eosinophilic material (unilateral or bilateral)	F ₀	0/30	4/30	6/30 ^c	18/30 ^d	39.7
	F ₁	8/30	7/30	14/30	22/30 ^d	55.0
Kidney, chronic interstitial nephritis (unilateral or bilateral)	F ₀	1/30	2/30	7/30 ^c	10/30 ^d	55.9
	F ₁	1/30	3/30	7/30 ^c	11/30 ^d	49.5
Kidney, foci of regenerative epithelium (unilateral or bilateral)	F ₀	0/30	1/30	5/30 ^c	8/30 ^d	73.0
	F ₁	1/30	0/30	5/30	11/30 ^d	116.7
^a statistical analysis (Fisher Exact test) performed for this review and not by original investigators ^b number of animals with lesion/total number of animals exposed ^c statistically significant (p≤0.05) ^d statistically significant (p≤0.01) NA not assessed because statistical significance was not observed at any concentration						

Table 8. ED₁₀, LED₁₀, and Selected Goodness of Fit Parameters from Modeled Incidence of Tubular Dilation/Eosinophilic Material (Unilateral or bilateral) Observed in Adult Male Rats Exposed to Chlorobenzene via Inhalation (Nair et al., 1987)

MODEL	ED ₁₀ (ppm)	LED ₁₀ (ppm)	χ^2 statistic	AIC
Gamma ^a	53.8	39.7	0.786	97.0
Quantal linear	53.8	39.7	0.786	97.0
Weibull ^a	53.8	39.7	0.786	97.0
Multi-stage ^b	56.6	39.8	0.586	99.0
Log-logistic ^c	51.33	17.0	0.501	99.4
Log-probit ^c	96.3	66.1	0.137	102.4
Probit	131.5	103.5	0.230	102.5
Logistic	143.4	111.6	0.210	102.9
Quantal quadratic	152.7	125.2	0.133	103.7

^a Restrict power ≥ 1
^b Restrict betas ≥ 0 , Degree of polynomial = 2
^c Slope restricted to >1

The LED_{10 HEC} of 46 mg/m³ was divided by an uncertainty factor of 100 (3 to account for interspecies extrapolation using dosimetric adjustments, 10 to protect sensitive subpopulations, and 3 for database uncertainties [including the lack of adequate neurotoxicity data and the absence of a study that examined the entire respiratory tract]) to yield a provisional **subchronic RfC of 5E-1 mg/m³**, as follows:

$$\begin{aligned}
 \text{subchronic p-RfC} &= \text{LED}_{10 \text{ HEC}} \div \text{UF} \\
 &= 46 \text{ mg/m}^3 \div 100 \\
 &= 0.5 \text{ or } 5\text{E-1 mg/m}^3
 \end{aligned}$$

Because no chronic inhalation toxicity studies were located in the literature, an additional subchronic-to-chronic uncertainty factor of 10 was applied to the provisional subchronic RfC to derive the provisional **chronic RfC of 5E-2 mg/m³**, as follows:

$$\begin{aligned}
 \text{p-RfC} &= \text{subchronic p-RfC} \div \text{UF} \\
 &= 5\text{E-1 mg/m}^3 \div 10 \\
 &= 5\text{E-2 mg/m}^3
 \end{aligned}$$

One area of uncertainty in the inhalation toxicity database for chlorobenzene is the lack of a study in which the entire respiratory tract was examined. None of the studies discussed in this issue paper examined the upper respiratory tract. Dilley (1977) examined the lungs, but the high incidence of chronic respiratory disease observed in the controls and chlorobenzene-exposed animals limited the ability of this study to detect chlorobenzene-related lung effects.

Data reported by Irish (1963) indicate that the lungs are not more sensitive than the liver or kidneys to chlorobenzene effects, although data from this study were not adequately reported and, therefore, cannot be independently assessed. In the Ogata et al. (1991) human study, none of the subjects complained of nose or eye irritation, although one of the subjects did complain of a sore throat following a 7-hour exposure to 60.2 ppm (277 mg/m³). Another area of uncertainty is the lack of neurological testing. The available human data (Ogata et al., 1991) suggest that the nervous system may be a sensitive target of chlorobenzene toxicity. Headaches and drowsiness were reported by experimental subjects during exposure to 60.2 ppm (277 mg/m³). The subchronic and chronic RfCs that were derived from Nair et al. (1987) (0.5 and 0.05 mg/m³, respectively) are substantially lower than concentrations associated with these effects. Although Tarkhova (1965) reported changes in electroencephalographic (EEG) patterns in response to light flashes in 2/4 human subjects exposed at 0.2 mg/m³, the toxicological relevance of this effect is not clear, and the reliability of these data is uncertain. Other studies have reported potential neurological effects in exposed humans (Rosenbaum et al., 1947; Girard et al., 1969); however, there is some uncertainty whether these effects were related to chlorobenzene exposure because neither of these studies reported data from unexposed controls. None of the repeated-dose animal studies observed overt signs of neurological effects. Tarkhova (1965) reported potential effects in rats at 0.2 ppm (1 mg/m³); however, the toxicological significance of the reported effect (changes in the conduction speeds of nerve impulses to sets of flexor and extensor muscles on Day 39) is not clear, and these results have not been confirmed by other studies.

Confidence in the principal study (Nair et al. 1987) is high. It is a well designed two-generation study examining relevant endpoints with an adequate number of animals. Confidence in the database is low. As discussed above, the database lacks a study that adequately examined the entire respiratory tract, and also lacks an adequate neurotoxicity study. Because the available data suggest that neurotoxicity may be a sensitive toxicological endpoint for chlorobenzene, confidence in the provisional chronic and subchronic RfC is low.

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Provisional Peer Reviewed Toxicity Values for
Cobalt
(CASRN 7440-48-4)

Derivation of Subchronic and Chronic Inhalation RfCs

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
COBALT AND COMPOUNDS (CASRN 7440-48-4)
Derivation of Subchronic and Chronic Inhalation RfCs**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

IRIS (U.S. EPA, 2000) does not report an RfC for cobalt. The HEAST (U.S. EPA, 1997) likewise does not list an RfC for cobalt. The CARA lists (U.S. EPA, 1991, 1994) report a HEA for cobalt (U.S. EPA, 1987), which derived a subchronic inhalation RfD of $9\text{E-}5 \text{ mg/m}^3$ based on a LOAEL of 0.1 mg/m^3 for respiratory effects in a 3-month study in swine (Kerfoot et al., 1975). A chronic inhalation RfD of $9\text{E-}6 \text{ mg/m}^3$ was derived from the same study. An updated HEA prepared by SRC (1990) for U.S. EPA, but never finalized, dropped the RfC because, it was argued, such an RfC would not be protective for individuals already sensitized to cobalt. ATSDR (1992) has published a Toxicological Profile for cobalt and compounds. A chronic inhalation MRL was not derived, due to a lack of a suitable chronic inhalation study. A subchronic inhalation MRL of $3 \times 10^{-5} \text{ mg cobalt/m}^3$ was derived based on a LOAEL of $0.11 \text{ mg cobalt/m}^3$ for metaplasia of the larynx in rats exposed for 13 weeks (6 hours/day, 5 days/week)

(NTP 1991). ACGIH (2000) has set a TLV-TWA of 0.02 mg/m³ for cobalt and inorganic cobalt compounds, expressed as cobalt, based on respiratory and cardiovascular effects. OSHA (1989, 1993) established a PEL of 0.05 mg/m³ for cobalt in its 1989 ruling, which has since reverted to the previous PEL of 0.1 mg/m³ following the court-ordered vacating of the 1989 ruling; the values are based on effects in the respiratory system. The NIOSH (2000) REL (TWA) for cobalt is 0.05 mg/m³, also based on respiratory effects. An IARC Monograph on cobalt and compounds (IARC, 1991) and the NTP Status Reports (NTP, 2000) were also searched for relevant information. The WHO (2000) has not published an Environmental Health Criteria document about cobalt. Literature searches were conducted from 1991 to November, 2000 for studies relevant to the derivation of an RfC. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, and TSCATS.

REVIEW OF PERTINENT DATA

Overview

Respiratory effects, including respiratory irritation, wheezing, asthma, pneumonia and fibrosis, have been widely reported in humans exposed to cobalt by inhalation (ATSDR, 1992). Cardiomyopathy has also been reported, although this effect is better known from oral exposure. Other effects of oral exposure in humans are polycythemia and thyroid effects. Cobalt is a sensitizer in humans by any route of exposure. Sensitized individuals may react to inhalation of cobalt by developing asthma; ingestion or dermal contact with cobalt may result in development of dermatitis. Several studies have suggested that cross-sensitization may occur between cobalt and nickel (Bencko et al., 1983; Lammintausta et al., 1985; Rystedt and Fisher, 1983; Shirakawa et al., 1990).

Animal studies have generally found similar effects to those observed in humans. One additional target found in animal studies that has not been identified as a target in humans is the reproductive system.

Urinary cobalt levels are a widely used and well accepted biomarker of exposure to cobalt in humans and animals, with many studies demonstrating good correlations between cobalt concentrations in the urine and ambient air (ATSDR, 1992; Gennart and Lauwerys, 1990; Nemery et al., 1992; NTP, 1991; Swennen et al., 1993). Some researchers have concluded that measurement of urinary cobalt is a more effective and more accurate means to monitor worker exposure than monitoring of air concentrations (Cugell, 1992; Nemery et al., 1992).

Human Studies

Numerous studies have investigated health effects in workers occupationally exposed to cobalt dust (Auchincloss et al., 1992; Cugell, 1992; Davison et al., 1983; Demedts et al., 1984; Gennart and Lauwerys, 1990; Kusaka et al., 1986a,b; Meyer-Bisch et al., 1989; Nemery et al., 1992; Prescott et al., 1992; Raffn et al., 1988; Shirakawa et al., 1988, 1989; Sprince et al., 1988; Swennen et al., 1993). However, many of these studies are of limited utility for risk assessment due to inadequate characterization of exposure and/or effects. In addition, many studies were of hard metal workers exposed to tungsten carbide as well as cobalt. There is substantial evidence from animal studies that tungsten, although it acts as an inert dust by itself, can potentiate the effects of cobalt on the respiratory tract (Lasfargues et al., 1992; Swennen et al., 1993). Therefore, studies of hard metal workers were not given further consideration. Four studies were considered to be potentially suitable bases for RfC derivation. Two of these focused exclusively on respiratory effects, one studied only thyroid effects, and one considered multiple endpoints. The populations studied included diamond-cobalt saw manufacturers, diamond polishers, plate painters and cobalt production workers. All four studies were cross-sectional in design.

Gennart and Lauwerys (1990) studied ventilatory function in workers at a plant producing diamond-cobalt circular saws. The exposed population consisted of 48 workers (34 males and 14 females) who agreed to participate in the study (an additional 27 workers declined). Exposure duration for these workers ranged from 0.1 to 32 years, with an average of about 6 years. The work involved weighing and mixing cobalt powder and microdiamond particles (and possibly small amounts of other undisclosed substances), cold pressing, heating and hot pressing. After sintering, the pieces were welded onto steel disks. These operations were performed in two rooms called the mixing room and the oven room, where all the examined workers spent most of their time. Controls were 23 workers (11 males and 12 females) from other factories in the same area who were not exposed to known pneumotoxic chemicals. Personal air samples were collected at different workplaces during half of a workshift. Subjects filled out a questionnaire regarding occupational and medical histories, smoking habits and pulmonary symptoms, gave a urine sample for cobalt determination and submitted to lung function tests. Cobalt concentrations varied from 9.4-2875 $\mu\text{g}/\text{m}^3$ in the mixing room (geometric mean = 135.5 $\mu\text{g}/\text{m}^3$) and 6.2-51.2 $\mu\text{g}/\text{m}^3$ (geometric mean = 15.2 $\mu\text{g}/\text{m}^3$) in the oven room. The prevalence of respiratory symptoms, such as cough, sputum and dyspnea, were significantly increased in the exposed workers. Mean predicted values of FEV₁ (forced expiratory volume in one second) and FVC (forced vital capacity) were significantly reduced, and the prevalence of abnormal values increased, in the exposed workers (both smokers and non-smokers). Among non-smokers, the spirometric effects were greater in workers exposed for 5 years or more than in those exposed for a shorter period of time.

Nemery et al. (1992) conducted a cross-sectional study of cobalt exposure and respiratory effects in diamond polishers. The study group was composed of 194 polishers working in 10

different workshops. In two of these workshops (#1,2), the workers used cast iron polishing disks almost exclusively, and in the others they used cobalt-containing disks primarily. The number of subjects from each workshop varied from 6-28 and the participation rate varied from 56-100%. The low participation in some workshops reflects the fact that only workers who used cobalt disks were initially asked to be in the study, rather than a high refusal rate (only 8 refusals were documented). More than a year after the polishing workshops were studied, an additional three workshops with workers engaged in sawing diamonds, cleaving diamonds or drawing jewelry were studied as an unexposed control group (n=59 workers). Subjects were asked to fill out a questionnaire regarding employment history, working conditions, medical history, respiratory symptoms and smoking habits, to give a urine sample for cobalt determination, and to undergo a clinical examination and lung function tests. Both area air samples and personal air samples were collected (always on a Thursday). Sampling for area air determinations started 2 hours after work began and continued until 1 hour before the end of the work day. Personal air samples were collected from the breathing zone of a few workers per workshop for four successive one-hour periods. Air samples were analyzed for cobalt and iron. In addition, personal air samplers were used to sample the air 1 cm above the polishing disks. These samples were analyzed for the entire spectrum of mineral and metallic compounds. Air samples were not obtained at one of the polishing workshops (#4), but this workshop was reported to be almost identical to an adjoining workshop (#3) for which samples were obtained. Urinary cobalt levels were similar between workers in these two workshops, so exposure was considered to be similar as well.

There was a good correlation ($R=0.92$) between the results of area and personal air sampling, with area air sampling reporting lower concentrations than personal air samples in all workshops except one (#9) (Nemery et al., 1992). In this workshop, personal air samples appeared to be artificially low in comparison to area air samples and urinary cobalt levels of the workers. When this workshop was excluded, there was a good correlation ($R=0.85-0.88$) between urinary cobalt and cobalt in the air. Based on urinary cobalt levels, the concentration of cobalt expected in personal air samples from workshop #9 was about $45 \mu\text{g}/\text{m}^3$ (the mean value actually reported was $6 \mu\text{g}/\text{m}^3$). The polishing workshops were divided into two groups: those with low exposure to cobalt (#1-5, n=102) and those with high exposure to cobalt (#6-10, n=91). Mean cobalt exposure concentrations were 0.4, 1.6 and $10.2 \mu\text{g}/\text{m}^3$ by area air sampling and 0.4, 5.3 and $15.1 \mu\text{g}/\text{m}^3$ by personal air sampling in the control, low-exposure and high-exposure groups, respectively. The inclusion of the apparently biased personal air samples from workshop #9 means that the reported mean cobalt exposure in the high-exposure group obtained by personal air sampling ($15.1 \mu\text{g}/\text{m}^3$) may be lower than the true value. Air concentrations of iron were highest in the two polishing workshops that used iron disks and the sawing workshop (highest value = $62 \mu\text{g}/\text{m}^3$), and were not correlated with cobalt levels. Analysis of samples taken near the disks showed the presence of cobalt, with occasional traces of copper, zinc, titanium, manganese, chromium, silicates and silicon dioxide. No tungsten was detected. There is a possibility that some workers had previously been exposed to asbestos, since pastes

containing asbestos had been used in the past to glue the diamonds onto holders. However, the degree of asbestos exposure had apparently been insufficient to produce functional impairment. The researchers considered cobalt to be the only relevant exposure. Smoking habits were similar in workers from the high-exposure, low-exposure and control groups. Duration of exposure was not discussed.

Workers in the high-exposure group were more likely than those in the other groups to complain about respiratory symptoms; the prevalences of eye, nose and throat irritation and cough, and the fraction of these symptoms related to work, were significantly increased in the high-exposure group (Nemery et al., 1992). Workers in the high-exposure group also had significantly reduced lung function compared to controls and low-exposure group workers, as assessed by FVC (forced vital capacity), FEV₁ (forced expiratory volume in one second), MMEF (forced expiratory flow between 25 and 75% of the FVC) and mean PEF (peak expiratory flow rate), although the prevalence of abnormal values did not differ significantly between exposure categories. Results in the low-exposure group did not differ from controls. Two-way analysis of variance was used to show that the effect on spirometric parameters in the high exposure group was present in both men and women. Women seemed to be affected more than men, but the interaction between exposure and sex was not significant. Smoking was found to exert a strong effect on lung function, but lung function level remained negatively correlated with exposure to cobalt, independently of smoking.

Swennen et al. (1993) conducted a cross-sectional study of workers exposed to metallic cobalt and inorganic cobalt compounds at a cobalt plant producing these materials from cobalt metal cathodes and scrap metal. The study group included 82 male workers from the cobalt plant who had not suffered from lung disease prior to employment and who had never been exposed to other pneumotoxic chemicals. It was not reported how this group was chosen from the pool of workers meeting these criteria or how many workers meeting these criteria refused to participate in the study. The control group comprised 82 age-matched workers from the mechanical workshop of a nearby plant owned by the same company. Workers filled out a questionnaire regarding occupational history, respiratory complaints and smoking habits, received a routine clinical examination, submitted to lung function tests, had a chest radiograph taken, and gave blood and urine samples (before and after working on Monday and Friday) for determination of cobalt content as well as hematological and serum chemistry analyses. Exposure was monitored by personal air samplers worn by each cobalt worker for 6 hours on both Monday and Friday.

Workers in the cobalt plant were exposed to cobalt concentrations ranging from 1 µg/m³ to 7772 µg/m³ (Swennen et al., 1993). The geometric mean exposure concentration was 125 µg/m³. Exposure duration ranged from 0.3-39.4 years, with an average exposure of 8.0 years. On the questionnaire, a significantly higher number of exposed workers reported dyspnea than controls. The increase occurred primarily among smokers, although no significant interaction was found between smoking and exposure to cobalt. A logistic regression model was used to

show that the probability of dyspnea during exercise increased as a function of cobalt concentration in the air and urine. The clinical examinations detected significantly increased prevalence of skin disorders (eczema, erythema) (51% vs 25%) and wheezing (16% vs 6%) in the exposed group compared to controls. Lung function tests did not differ between the two groups, but a few significant trends were noted: the FEV₁/VC (forced expiratory volume in one second/vital capacity) ratio decreased with increasing concentration of cobalt in the air and urine, and the RV (residual volume) and TLC (total lung capacity) increased with increasing duration of exposure. No lung abnormalities were found by chest radiographs in either group. The researchers concluded that these results demonstrate "airway involvement" in workers exposed only to cobalt and suggest there may have been thyroid effects as well. Blood analyses did not show polycythemia, and in fact there were slight, but significant, decreases in red blood cell count, hemoglobin, and hematocrit in the exposed workers. White blood cell was significantly increased. Serum levels of the thyroid hormone T3 (triiodothyronine) were slightly (7%), but significantly, decreased in the exposed group, while T4 (thyroxine) and TSH (thyrotropin) were not affected. Serum markers for cardiomyopathy were unchanged.

Prescott et al. (1992) conducted a cross-sectional study to investigate the effects of cobalt exposure on thyroid volume and function in female plate painters. The test group included 61 female plate painters exposed to cobalt blue dyes in two porcelain factories. The control group consisted of 48 unexposed women working at the same factories. The dyes used in the two factories differed; factory I (36 workers) used cobalt aluminate, which is insoluble, and factory II (25 workers) used cobalt-zinc silicate, which was reported to be "semi-soluble." Workers were exposed to cobalt during the painting procedure when the plates were spray-painted (under a fume hood) two or three times with the water-based cobalt blue underglaze and when the excess color was removed with a brush after drying. Cobalt concentrations were reported to be around 0.05 mg/m³ in the workplaces, but no further details were provided. Duration of exposure averaged 14.6 years in group I workers and 16.2 years in group II workers. Subjects were required to fill out a questionnaire regarding health, use of medicines, day of menstrual cycle, employment information and smoking habits, to give blood and urine samples for determination of thyroid hormones and cobalt, and to undergo ultrasonography to determine volume of the thyroid gland.

Urinary cobalt levels were similar in group I exposed workers and controls (Prescott et al., 1992). Group II workers had urinary cobalt levels that were roughly ten-fold higher than controls. Group I workers did not differ from controls for any of the thyroid parameters measured, but Group II workers had a significant 22% increase in serum T4 (thyroxine) levels. Thyroid volume appeared to be reduced in this group as well, although the difference from controls (16.1 ml in group II vs 19.2 ml in controls and 18.7 ml in group I) was not statistically significant. The occurrence of respiratory effects in these workers was not discussed.

Animal Studies

In a subchronic inhalation study, groups of 10 F344/N rats and 10 B6C3F1 mice of each sex were exposed to cobalt sulfate heptahydrate aerosol (MMAD=0.83-1.10 μm) at concentrations of 0, 0.3, 1, 3, 10 or 30 mg/m^3 (0, 0.11, 0.38, 1.14, 3.8 or 11.4 $\text{mg Co}/\text{m}^3$) 6 hours/day, 5 days/week for 13 weeks (Bucher et al., 1990; NTP, 1991). Animals were monitored for body weight and observed for clinical signs during the exposure period. Urine samples for urinalysis and cobalt determination were collected from rats prior to sacrifice. Following termination of exposure, all animals were sacrificed and necropsied. Blood samples were collected and analyzed for hematological parameters (rats and mice) and serum chemistry and thyroid function parameters (rats only). The major organs were weighed. Animals from the control and high-dose groups received comprehensive histopathological examinations, while those from the lower dose groups received more limited examinations focused on the respiratory tissues.

All rats survived until scheduled necropsy (Bucher et al., 1990; NTP, 1991). Gross evidence of toxicity was noted only in rats exposed to 30 mg/m^3 , which displayed clinical signs of toxicity (ruffled fur, hunched posture) and reduced body weights. Polycythemia, indicated by significant increases in red blood cell count, hemoglobin and hematocrit, was noted in males exposed to $\geq 3 \text{ mg}/\text{m}^3$ and females exposed to $\geq 10 \text{ mg}/\text{m}^3$. In addition, platelets were significantly reduced in rats of both sexes at $\geq 10 \text{ mg}/\text{m}^3$ and reticulocytes were increased in females at 30 mg/m^3 . Leukocyte counts and differentials were unaffected. Serum cholesterol was significantly reduced in males at $\geq 10 \text{ mg}/\text{m}^3$ and females at 30 mg/m^3 . No other serum chemistry parameters were affected, including creatine kinase isozymes indicative of damage to cardiac muscle cells. Among the thyroid hormones, T3 (triiodothyronine) was significantly reduced in females at $\geq 10 \text{ mg}/\text{m}^3$ and TSH (thyrotropin) was significantly reduced in males at 30 mg/m^3 , but T4 (thyroxin) was not affected in either sex at any dose and the researchers concluded that thyroid function was not consistently affected in this study. Urinalysis revealed a dose-related increase in the number of epithelial cells in the urine of male rats exposed to $\geq 3 \text{ mg}/\text{m}^3$ and granular casts in the urine of many exposed male rats (3-7 per group), but no controls. The researchers interpreted this finding to indicate minimal nephropathy in exposed male rats, although histopathological lesions were not detected in the kidney. No effects on sperm counts, sperm motility or the incidence of abnormal sperm were noted. Average estrus cycle of females exposed to 30 mg/m^3 was slightly longer than controls, but the difference was not significant. Absolute and relative lung weight were significantly increased in both male and female rats at $\geq 1 \text{ mg}/\text{m}^3$. Other organ weights were not affected by treatment. Compound-related lesions were found only in the respiratory tissues of exposed rats. Degenerative, inflammatory and regenerative lesions were found throughout the respiratory tract. The lesions were concentration-related and similar in incidence and severity in males and females. The most sensitive tissue was the larynx, with squamous metaplasia present at all exposure levels.

Among mice, 2/10 males exposed to 30 mg/m³ died during the study (Bucher et al., 1990; NTP, 1991). The only clinical signs of toxicity observed were rapid breathing and skin discoloration in one of the mice that died. Body weights were reduced throughout the study in both males and females exposed to 30 mg/m³. No dose-related hematological effects were found. Absolute and relative lung weight were significantly increased in male and female mice exposed to ≥ 10 mg/m³. Respiratory lesions were similar to those observed in rats. As with rats, the most sensitive tissue was the larynx, with squamous metaplasia present at all exposure levels. Reproductive system effects were more prominent in mice than rats. Males had significantly decreased testicular and epididymal weight, testicular atrophy consisting of loss of germinal epithelium in the seminiferous tubules and foci of mineralization, and an increased percentage of abnormal sperm at 30 mg/m³, and reduced sperm motility at ≥ 3 mg/m³ (lower doses not tested). Females had a significantly increased length of the estrus cycle at 30 mg/m³.

In a subsequent chronic inhalation carcinogenicity study by the same researchers, groups of 50 F344/N rats and 50 B6C3F1 mice of each sex were exposed to cobalt sulfate heptahydrate aerosol (MMAD=1.4-1.6 μ m) at concentrations of 0, 0.3, 1, or 3 mg/m³ (0, 0.11, 0.38, or 1.14 mg Co/m³) 6 hours/day, 5 days/week for 105 weeks (Bucher et al., 1999; NTP, 1998). Animals were monitored for body weight and observed for clinical signs during the exposure period. Following termination of exposure, all animals were sacrificed and necropsied. At necropsy, all organs and tissues were examined for gross lesions, trimmed, and examined histologically.

In F344 rats, there were no changes in survival or mean body weights in males or females of any exposure group (Bucher et al., 1999; NTP, 1998). Irregular breathing was noticed more frequently in female rats exposed to 3 mg/m³ than in controls or other groups; no changes in clinical signs were noted in any of the treated male rats. Both male and female rats in all exposure groups showed a high incidence (94% or greater) of squamous metaplasia of the alveolar epithelium, fibrosis of the pulmonary interstitium, and granulomatous inflammation, with all lesions increasing in severity with increasing exposure. Likewise, both sexes of rats showed dose-related increases in metaplasia of the larynx, atrophy of the olfactory epithelium, and hyperplasia of the lateral nasal wall. Significant increases in alveolar/bronchiolar adenomas or carcinomas were seen in high-dose male rats, while significant increases in alveolar/bronchiolar adenomas, alveolar/bronchiolar carcinomas, or squamous cell carcinomas were seen in the mid- and high-dose female rats. Increased incidence of pheochromocytomas were seen in the mid-dose male rats, but not the high-dose males, and in high-dose female rats.

In B6C3F1 mice, no changes in survival were noticed in any exposure group (Bucher et al., 1999; NTP, 1998). Male mice exposed to 3 mg/m³ showed a decreased mean body weight relative to controls from week 96 through the end of the study. Mean body weights of exposed female mice were generally greater than those of controls throughout the study. Irregular breathing was noted slightly more frequently in female mice exposed to 1mg/m³ than in controls or other exposed groups. A dose-related increase in the occurrence of cytoplasmic vacuolization

of the bronchus was seen in both sexes of mice, with incidences at all exposure levels being significantly different from controls. Mice of both sexes showed significantly increased incidences of squamous metaplasia of the larynx at all exposure levels examined. The incidence of alveolar/bronchiolar adenoma or carcinoma was significantly increased in high-dose male mice, and in mid- and high-dose female mice.

Other studies in animals have also reported respiratory lesions produced by inhaled cobalt. Johansson et al. (1987) observed effects in the alveolar region of the respiratory tract in rabbits exposed to 0.4 mg Co/m³, 6 hours/day, 5 days/week for 1-4 months. Kyono et al. (1992) observed mild pulmonary lesions in rats exposed to 2.12 mg/m³ of cobalt aerosols 5 hours/day for 4 days. Kerfoot et al. (1975) reported decreased pulmonary function, lethargy, and wheezing following exposure of groups of 5 miniature swine to 0, 0.1, or 1.0 mg/m³ of cobalt dust for 6 hours/day, 5 days/week for 3 months.

No developmental toxicity studies were located following inhalation exposure to cobalt. Oral developmental toxicity studies did not find evidence that cobalt is a potent developmental toxicant; one study in rats and one in mice reported no developmental effects, while a second study in rats reported developmental effects (reduced number of litters and average litter weight, increased number of dead pups/litter) that were considered by the researchers to be secondary consequences of maternal toxicity (ATSDR, 1992). The NTP (1991) study (described above) demonstrated that cobalt can produce reproductive effects in male and female mice following inhalation exposure, although the effects were produced at relatively high dose levels. Oral studies have also identified the testes as a target for cobalt toxicity. No multi-generation reproduction studies were located by inhalation or oral exposure.

DERIVATION OF PROVISIONAL RfC

Of the four epidemiology studies discussed above, the study by Nemery et al. (1992) provides the best basis for derivation of an RfC. Workers in this study were exposed to far lower concentrations of cobalt, over a much more constrained range of exposures, than in the studies by Gennart and Lauwerys (1990) and Swennen et al. (1993). As a result, discrete NOAEL and LOAEL values can be derived from the Nemery et al. (1992) study, which would not have been possible using the Gennart and Lauwerys (1990) or Swennen et al. (1993) studies. The Prescott et al. (1992) study was not considered for RfC derivation because evidence of an effect on the thyroid was marginal (22% increase in serum levels of T4) and respiratory endpoints were not investigated. The human and animal database strongly suggests that respiratory effects are the most sensitive endpoints of cobalt toxicity. Respiratory effects have been widely reported in workers exposed to cobalt, while effects on the thyroid and other tissues have not (ATSDR, 1992). In addition, Swennen et al. (1993) found only marginal evidence of thyroid effects (7% decrease in T3) and no evidence of cardiomyopathy or polycythemia in workers clearly

displaying evidence of respiratory effects. The animal data also support the conclusion that the respiratory tract is the critical target for inhaled cobalt. NTP (1991) observed respiratory lesions in rats and mice at the lowest exposure concentration tested, while polycythemia and reproductive effects were seen only at higher concentrations, thyroid function was not consistently affected at any concentration, and cardiomyopathy was undetected even at the highest concentration.

Assuming the personal air samples to be more representative of worker exposure than the area air samples, the study by Nemery et al. (1992) identified a NOAEL of $5.3 \mu\text{g}/\text{m}^3$ and a LOAEL of $15.1 \mu\text{g}/\text{m}^3$ for effects on lung function. Although the LOAEL may be biased low due to inclusion of data from workshop #9, this does not affect the RfC derivation. The NOAEL for occupational exposure is adjusted to continuous exposure as follows:

$$5.3 \mu\text{g}/\text{m}^3 (10 \text{ m}^3/\text{d} / 20 \text{ m}^3/\text{d}) (5 \text{ d} / 7 \text{ d}) = 1.9 \mu\text{g}/\text{m}^3$$

Dividing the NOAEL_{ADJ} of $1.9 \mu\text{g}/\text{m}^3$ by an uncertainty factor of 100 (3 to account for the fact that exposure duration may have been subchronic in some workers, 3 for lack of inhalation developmental toxicity studies and a multi-generation reproduction study, and 10 for human variability) yields an **RfC of $2\text{E-}5 \text{ mg}/\text{m}^3$** for cobalt. This RfC may not be protective for people with hypersensitivity to cobalt.

STATEMENT OF CONFIDENCE

Confidence in the key study is low. This was a cross-sectional study that looked at only respiratory endpoints, included a control group that was studied more than one year after the exposed population, included a study group exposed to iron and diamond dust in addition to cobalt (and possibly to asbestos in the past), had no discussion of duration of exposure, and encountered a number of procedural difficulties during its course. Confidence in the database is medium; choice of the critical endpoint is well supported by other studies in humans and animals, but reproductive and developmental effects have not been adequately studied. Medium-to-low confidence in the RfC follows.

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Provisional Peer Reviewed Toxicity Values for
Cobalt and Compounds
(CASRN 7440-48-4)

Derivation of a Carcinogenicity Assessment

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
COBALT AND COMPOUNDS (CAS NO. 7440-48-4)
Derivation of a Carcinogenicity Assessment**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

IRIS (U.S. EPA, 2000a) does not report a cancer classification, slope factor, or unit risk for cobalt. The HEAST (U.S. EPA, 1997) and Drinking Water Standards and Health Advisories list (U.S. EPA, 2000b) likewise do not report carcinogenicity assessments for cobalt. The CARA lists (U.S. EPA, 1991, 1994a) include a HEA for cobalt (U.S. EPA, 1987), which did not report a cancer classification or an estimate of the carcinogenic potency of stable cobalt compounds due to a lack of pertinent data. A draft of an updated HEA prepared by SRC (1990) for U.S. EPA, but never finalized, was also located, which likewise found insufficient data to assess the carcinogenicity of stable cobalt. An IARC Monograph on cobalt and compounds (IARC, 1991) classified cobalt and compounds as "possibly carcinogenic to humans." ACGIH (2000) has classified cobalt in category A3 - confirmed animal carcinogen with unknown relevance to humans. The ATSDR Toxicologic Profile for cobalt and compounds (ATSDR, 1992) and the NTP Status Reports (NTP, 2000) were also searched for relevant information. The WHO (2000) has not published an Environmental Health Criteria document about cobalt. Literature searches were conducted from 1991 to November, 2000 for studies relevant to the derivation of a provisional carcinogenicity assessment for cobalt. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, and TSCATS.

REVIEW OF PERTINENT DATA

Human Studies

Morgan (1983) investigated the health and causes of death of 49 men occupationally exposed to cobalt salts and oxides in a manufacturing plant in South Wales. During the study period, 33 men died, 5 with lung cancer and 3 with cancer at other sites. The expected number of deaths was 0.3 for lung cancer and 4.1 for all cancers based on national statistics, resulting in mortality ratios of 16.7 and 0.73, respectively. U.S. EPA (1987) concluded that the scope of this study was too limited to demonstrate the carcinogenicity or noncarcinogenicity of occupational exposure to cobalt compounds.

Mur et al. (1987) analyzed the mortality of a cohort of 1143 workers in a plant that refined and processed cobalt and sodium. An increase in deaths resulting from lung cancer was observed in workers (SMR = 4.66 [1.46-10.64]) based on four cases observed in the exposed group and 1 case expected based on French national statistics. In a study within the cohort that controlled for age and smoking habits, 44% (four workers) in the exposed group and 17% (three workers) in the control group died of lung cancer. The authors indicated that the differences were not statistically significant and that the workers were exposed to arsenic and nickel in addition to cobalt. The exposure levels of cobalt were not reported.

Tuchsen et al. (1996) analyzed the cancer incidence of a cohort of 874 women (382 from one factory, 492 from a second factory) who worked in one of two factories applying a cobalt-based plate underglaze. 520 referents were selected from unexposed areas of factory 1. All groups were compared to statistics for all Danish women in the same calendar year. During the follow-up period, the overall cancer incidence was only slightly elevated in exposed workers, while the incidence of lung cancers was significantly increased (SIR 2.35, 95% CI 1.01-4.6). The incidence of lung cancers in the referents (not exposed to cobalt) was greater than that of all Danish women, but the difference was not significant. Exposure characterization prior to 1980 was not described, while exposures after 1980 were variable and reported as a mean concentration for a given year. Exposures were generally in the range of 0-1 mg cobalt/m³ except for two years, during which they were greater.

Several studies examining the effects of hard metal, a mixture containing approximately 20% cobalt with the remainder being primarily tungsten carbide. Exposure of humans to hard metal has been shown to result in an increase in cancer mortality (Lasfargues et al., 1994; Moulin et al., 1998) as well as a number of other diseases, including asthma and fibrosis (for reviews, see Barceloux, 1999; Lison, 1996). There is substantial evidence from animal studies that tungsten, although it acts as an inert dust by itself, can potentiate the effects of cobalt on the respiratory tract (Lasfargues et al., 1995; Lison et al., 1995, 1996; Swennen et al., 1993). For this reason, studies of hard metal were not given further consideration.

Animal Studies

Wehner et al. (1977, 1979) exposed groups of 51 month old male Syrian golden hamsters by inhalation to cobalt oxide at 0 or 10 mg/m³, 7 hours/day, 5 days/week for their lifetimes. The

incidence of tumors in treated hamsters was not statistically different from controls. There was “limited” histopathologic and ultrastructural examination in the study.

In a chronic inhalation carcinogenicity study, groups of 50 F344/N rats and 50 B6C3F1 mice of each sex were exposed to cobalt sulfate heptahydrate aerosol (MMAD=1.4-1.6 μm) at concentrations of 0, 0.3, 1, or 3 mg/m^3 (0, 0.11, 0.38, or 1.14 $\text{mg Co}/\text{m}^3$) 6 hours/day, 5 days/week for 105 weeks (Bucher et al., 1999; NTP, 1998). Animals were monitored for body weight and observed for clinical signs during the exposure period. Following termination of exposure, all animals were sacrificed and necropsied. At necropsy, all organs and tissues were examined for gross lesions, trimmed, and examined histologically.

Mortality in either sex of F344/N rats was not affected at any exposure level, nor did treatment result in alterations in body weight (Bucher et al., 1999; NTP, 1998). The combined incidence of alveolar/bronchiolar neoplasms (adenoma and carcinoma) in males were 1/50, 4/50, 4/48, and 7/50 in the control, 0.3, 1, and 3 mg/m^3 groups, respectively, while in females the incidences were 0/50, 3/49, 15/50, and 15/50, respectively. The incidences in the 3 mg/m^3 male rats and the 1 and 3 mg/m^3 female rats were significantly greater than those in controls animals, and a significant linear trend occurred in both sexes. A significant increase in the incidence of pheochromocytoma in 3 mg/m^3 females was also noted (2/48, 1/49, 4/50, and 10/50 in control, 0.3, 1, and 3 mg/m^3 groups, respectively). A marginally increased incidence of pheochromocytoma in males exposed to 1 mg/m^3 , but not in those exposed to 3 mg/m^3 , was not considered by the study authors to be related to treatment.

In B6C3F1 mice, no changes in survival were noticed in any exposure group (Bucher et al., 1999; NTP, 1998). Male mice exposed to 3 mg/m^3 showed a decreased mean body weight relative to controls from week 96 through the end of the study (~10 weeks). Mean body weights of exposed female mice were generally greater than those of controls throughout the study. As in rats, both sexes of mice showed a significant linear trend toward increased alveolar/bronchiolar tumors, with the 3 mg/m^3 male and the 1 and 3 mg/m^3 female groups attaining statistical significance. Incidence of combined alveolar/bronchiolar adenoma or carcinoma were 11/50, 14/50, 19/50, and 28/50 in males and 4/50, 7/50, 13/50, and 18/50 in females in the control, 0.3, 1, and 3 mg/m^3 groups, respectively. In male mice, but not in females, the incidence of hemangiosarcoma was significantly elevated in animals exposed to 1 mg/m^3 , but not in other exposure groups (2/50, 4/50, 8/50, and 7/50 in the control, 0.3, 1, and 3 mg/m^3 groups, respectively).

Other Studies

Heath (1956) injected groups of 10 male and 20 female rats with a single intramuscular 28 mg dose of powdered cobalt in the thigh. Injection site sarcomas appeared in 18 (60%) of the treated rats within 5-12 months. Similar results were observed in Wistar rats by Gilman (1962) and Gilman and Ruckerbauer (1962), with single intramuscular doses of 20 mg of cobalt oxide and cobalt sulfide. Cobalt oxide and cobalt sulfide given intramuscularly at doses twice those used in rats did not induce sarcomas in mice (Gilman and Ruckerbauer, 1962). Shabaan et al. (1977) observed a high incidence of fibrosarcomas in rats given subcutaneous injections of cobalt chloride at 40 mg/kg -day for 10 days. Tumors developed in 8-12 months. Stoner et al. (1976) tested cobalt acetate in the strain A mouse pulmonary tumor test. Groups of 20 mice/sex

received three times per week intraperitoneal injections for a total of 19 cumulative doses of 0, 95, 237, or 475 mg/kg. Survival was high over the 30 week observation period, and the incidence of lung tumors in treated mice was not statistically different from controls.

The genetic toxicity of cobalt was reviewed by Beyersman and Hartwig (1992). Cobalt compounds have generally tested negative in bacterial mutagenicity assays, with the occasional positive result occurring only with the addition of an exogenous metabolic system. By contrast, cobalt compounds have generally tested positive in yeast and plant cells. In mammalian cell systems, cobalt has been shown to induce DNA strand breaks, sister-chromatid exchanges, and morphological cell transformation. Single oral exposure of male Swiss mice to 0, 4.96, 9.92, or 19.8 mg cobalt/kg as cobalt chloride resulted in significantly increased percentages of both chromosomal breaks and chromosomal aberrations, with significant linear trends toward increasing aberrations with increased exposure (Palit et al., 1991a,b,c,d). Thirty hours following single intraperitoneal injection of cobalt(II) chloride in BALB/c mice, an increase in micronucleus formation was seen at 12.4 or 22.3 mg cobalt/kg (as cobalt chloride), but not at 6.19 mg cobalt/kg (Suzuki et al., 1993). Single injection of 12.4 mg/kg $\text{CoCl}_2 \cdot 6\text{H}_2\text{O}$ resulted in significantly increased micronucleus formation at 24 hours post-injection, but not at 12, 48, 72, or 96 hours. Pedigo and Vernon (1993) reported that treatment with 400 ppm cobalt (99 mg cobalt/kg-day) in the drinking water of mice for 10 weeks resulted in an increase in dominant lethal effects.

While the precise mechanism of action of cobalt has not been determined, a number of potential mechanisms have been identified. The most likely mechanism for the carcinogenic effects of cobalt involves the generation of cobalt-induced oxidative stress. Exposure to cobalt compounds increases indices of oxidative stress, including diminished levels of reduced glutathione, increased levels of oxidized glutathione, increased levels of oxygen radicals, and increased free-radical-induced DNA damage (Kadiiska et al., 1989; Kawanishi et al., 1994; Lewis et al., 1991; Moorhouse et al., 1985; Zhang et al., 1998). The tungsten in hard metal is thought to enhance the generation of oxidants by cobalt, thus explaining the increased activity of hard metal relative to cobalt alone.

PROVISIONAL WEIGHT OF EVIDENCE CLASSIFICATION FOR COBALT

Under the 1986 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1986), cobalt is classified as group **B1 (Probable Human Carcinogen)**, based on limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals, as evidenced by increased incidence of alveolar/bronchiolar tumors in both sexes of rats and mice. Under the proposed cancer guidelines (U.S. EPA, 1999), cobalt is considered likely to be carcinogenic to humans.

While available studies in humans have been suggestive of a possible association between exposure to cobalt and respiratory tumors (Morgan et al., 1983; Mur et al., 1987; Tuchsén et al., 1996), limitations of the studies, including small numbers of subjects, inadequate exposure assessment and/or potential exposure to other chemicals, make them inadequate for assessing the carcinogenic potential of cobalt. Available chronic animal studies have demonstrated the carcinogenic potential of inhaled cobalt in male and female rats and mice, with alveolar and

bronchiolar tumors being the most prevalent (Bucher et al., 1999; NTP, 1998). No studies suitable for evaluation of the oral carcinogenic potential for cobalt were located.

The precise mechanism of cobalt-induced carcinogenicity has not been fully determined. There is evidence that cobalt is capable of eliciting genotoxic effects. While evaluations for mutagenic effects in bacteria have generally yielded negative results, results in mammalian cell systems have suggested that cobalt may be genotoxic in mammalian cells. Limited data from *in vivo* animal studies have also suggested genotoxic effects of cobalt, including chromosomal breaks, chromosomal aberrations, and micronucleus formation. The most likely mechanism for the carcinogenic effects of cobalt involves the generation of cobalt-induced oxidative stress.

QUANTITATIVE ESTIMATES OF CARCINOGENIC RISK

Provisional Oral Slope Factor for Cobalt

No human or animal studies examining the carcinogenicity of cobalt following oral exposure were located. Therefore, derivation of an oral slope factor is precluded.

Provisional Inhalation Unit Risk for Cobalt

As available human studies were not sufficiently detailed, particularly with regards to analysis of exposure, the NTP (1998; Bucher et al. 1999) 2-year carcinogenicity study in rats and mice was chosen as the key study for the derivation of an inhalation unit risk. The concentrations were first adjusted to continuous exposure as follows:

$$Conc_{[adj]} = Conc \times \frac{5 \text{ days / week}}{7 \text{ days / week}} \times \frac{6 \text{ hours / day}}{24 \text{ hours / day}}$$

This adjustment resulted in duration-adjusted concentrations of 0, 0.020, 0.068, and 0.20 mg cobalt/m³ for the control, 0.3, 1, and 3 mg/m³ groups, respectively. Using the RDDR computer program, as specified in the RfC guidelines (U.S. EPA, 1994b), human equivalent concentrations (HECs, in mg cobalt/m³) were calculated at each exposure level for each species and sex, assuming particulate effects (MMAD=1.5 µm, σ_g=2.2) in the thoracic portion of the respiratory tract. The results are displayed below.

	Male Rat	Female Rat	Male Mouse	Female Mouse
RDDR Multiplier	0.83	0.79	1.48	1.44
Control	0	0	0	0
Low	0.017	0.016	0.030	0.029
Medium	0.056	0.054	0.10	0.098
High	0.17	0.16	0.30	0.29

The GLOBAL86 computer program was then used to fit the incidence data to the HEC for each species and sex. Calculations were based on extra risk. The results of the analysis are presented in the table below:

Species	Sex	Control	Low	Med	High	q_1^* (mg/m ³) ⁻¹	LEC ₁₀ (mg/m ³)	P-value [#]
Rat	M	1/50	4/50	4/48	7/50	1.5	7.2×10^{-2}	0.50
Rat	F	0/50	3/49	15/50	15/50	4.4	2.4×10^{-2}	0.0085
Rat	F	0/50	3/49	15/50	***	8.1	1.3×10^{-2}	0.40
Mouse	M	11/50	14/50	19/50	28/50	2.9	3.6×10^{-2}	0.95
Mouse	F	4/50	7/50	13/50	18/50	2.1	5.1×10^{-2}	0.55

- P value describing the goodness of fit of the model. A greater p value describes a better fit, with $p > 0.05$ being a statistically significant fit.

*** - Because of a poor fit, the group was re-analyzed without the data from the high-exposure animals.

The model did not accurately fit the data from female rats, which appeared to be the most sensitive species and gender. If the high-dose group is dropped, the model better fits the data, but likely overestimates the risk, since the incidences of tumors in both the mid- and high-dose groups were the same. The most conservative (health-protective) value obtained with adequate model fit using all dose groups, calculated from male mice, was therefore selected.

In accordance with the proposed Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1999), the unit risk was also calculated by drawing a straight line between the LEC₁₀ and the origin. The linear dose-response model was utilized due to the lack of understanding of the mode(s) of action of cobalt-induced carcinogenesis. This method results in unit risk for pulmonary carcinogenesis of 2.8×10^{-3} (ug Co/m³)⁻¹, calculated as follows:

$$\begin{aligned}
 IUR &= \text{Incidence} / \text{Dose} \\
 &= 10\% \div \text{LEC}_{10} \\
 &= 0.1 \div 3.6 \times 10^{-2} (\text{mg Co} / \text{m}^3) \\
 &= 2.8 (\text{mg Co} / \text{m}^3)^{-1} \\
 &= 2.8 \times 10^{-3} (\text{ug Co} / \text{m}^3)^{-1}
 \end{aligned}$$

This value is similar to that generated by directly calculating the q_1^* , which was 2.9×10^{-3} (ug/m³)⁻¹. From these values, the following air concentrations were calculated for the specified risk levels:

<u>Risk Level</u>	<u>Concentration</u>
E-4 (1 in 10,000)	4E-2 ug Co/m ³
E-5 (1 in 100,000)	4E-3 ug Co/m ³
E-6 (1 in 1,000,000)	4E-4 ug Co/m ³

In summary, cobalt is provisionally classified as group B1 (Probable Human Carcinogen), based on limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals, as evidenced by increased incidence of alveolar/bronchiolar tumors in both sexes of rats and mice, under the 1986 cancer guidelines (U.S. EPA, 1986). Under the proposed cancer guidelines (U.S. EPA, 1999), cobalt is considered likely to be carcinogenic to humans. A provisional **inhalation unit risk of $2.8 \times 10^{-3} (\mu\text{g Co}/\text{m}^3)^{-1}$** was derived for cobalt, based on the increased incidence of respiratory tumors in male B6C3F1 mice. Derivation of the unit risk under the proposed guidelines (U.S. EPA, 1999) and the 1986 guidelines (U.S. EPA, 1986) resulted in very similar values. Data were inadequate to derive an oral slope factor for cobalt.

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Provisional Peer Reviewed Toxicity Values for

Cobalt

(CASRN 7440-48-4)

Derivation of Subchronic and Chronic Oral RfDs

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
COBALT AND COMPOUNDS (CASRN 7440-48-4)
Derivation of Subchronic and Chronic Oral RfDs**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

IRIS (U.S. EPA, 2000a) does not report an RfD for cobalt. The HEAST (U.S. EPA, 1997) and Drinking Water Standards and Health Advisories (U.S. EPA, 2000b) likewise do not list an RfD for cobalt. The CARA lists (U.S. EPA, 1991, 1994) report a HEA for cobalt (U.S. EPA, 1987). The 1987 HEA derived a chronic RfD of 0.005 mg cobalt/kg-day based on a NOAEL of 5 mg cobalt/kg-day for testicular effects in a subchronic rat study (Nation et al., 1983). An updated HEA prepared by SRC (1990) for U.S. EPA, but never finalized, dropped the RfD because, it was argued, such an RfD would not be protective for individuals already sensitized to cobalt. ATSDR (1992) has published a Toxicological Profile for cobalt and compounds, but no oral MRLs were derived due to the absence of suitable data. The WHO (2000) has not published an Environmental Health Criteria document about cobalt. An IARC Monograph on cobalt and compounds (IARC, 1991) and the NTP Status Reports (NTP, 2000)

were searched for relevant information. Literature searches were conducted from 1991 to November, 2000 for studies relevant to the derivation of an RfD. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, and TSCATS.

REVIEW OF PERTINENT DATA

Cobalt has been found to stimulate the production of red blood cells in humans and, therefore, has been used as a treatment for anemia. In 12 anemic, anephric patients undergoing dialysis, treatment with 0.18 mg cobalt/kg-day as cobalt chloride for 12 weeks resulted in a significant rise in hemoglobin (Duckham and Lee, 1976). Taylor et al. (1977) reported similar effects in 8 anephric patients treated with 0.16-0.32 mg cobalt/kg-day as cobalt chloride for 12-32 weeks. In both studies, hemoglobin levels rapidly returned to pre-treatment levels following the cessation of treatment. Similar effects were reported in nonanemic humans and animals (Davis and Fields, 1958; Krasovskii and Fridlyand, 1971). Reversible polycythemia was reported in 6 normal male subjects following treatment with 1 mg cobalt/kg-day as cobalt chloride for 25 days (Davis and Fields, 1958). In normal rats, treatment with 0.5 mg cobalt/kg-day, but not 0.05 mg cobalt/kg-day, as cobalt chloride resulted in polycythemia and an increase in hemoglobin (Krasovskii and Fridlyand, 1971). An increase in hematocrit and hemoglobin levels was not observed, however, in pregnant women treated with 0.5-0.6 mg cobalt/kg-day for 90 days in an attempt to alleviate the anemia often found during pregnancy (Holly, 1955).

Much of the oral data in humans deals with the cardiomyopathy seen in people who drank large quantities of beer containing cobalt chloride (previously used to stabilize the foam) (Alexander, 1969, 1972; Morin et al., 1971). The people ingested 0.04-0.14 mg cobalt/kg-day (approximately 8-30 pints of beer daily) over a period of years (Alexander, 1969, 1972; Morin et al., 1971). The cardiomyopathy in the beer-drinkers, termed "beer-cobalt cardiomyopathy," was fatal to 43% of the subjects within several years, with approximately 18% of these deaths occurring within the first several days following diagnosis. The beer-cobalt cardiomyopathy appeared to be similar to alcoholic cardiomyopathy and beriberi, but the onset of the beer-cobalt cardiomyopathy was much more abrupt. The practice of adding cobalt to beer to stabilize the foam has been discontinued. It should be noted, however, that the cardiomyopathy may also have been due to the fact that the beer-drinkers had protein-poor diets and may have had prior or concurrent cardiac and hepatic damage from alcohol abuse. Treatment of anemic patients, either resulting from pregnancy or other causes, with comparable or much higher doses of cobalt (0.09-1 mg cobalt/kg-day, ranging from 3-32 weeks) did not result in effects on the heart (Duckham and Lee, 1976; Davis and Fields, 1958; Holly, 1955; Taylor et al., 1977).

Studies in animals have noted cardiac effects following cobalt exposure (Haga et al., 1996; Mohiuddin et al., 1970; Pehrsson et al., 1991), though at higher exposure levels than examined in human studies. Pehrsson et al. (1991) exposed male rats (12/group) to protein-

restricted diets containing 8.4 mg Co/kg-day. Treated rats showed a significant decrease in body weight, but no differences in left ventricular function, relative to animals treated with protein-restricted diets without cobalt. In a followup study (Haga et al., 1996), in which groups of 12-16 rats were similarly exposed to 8.4 mg Co/kg-day for 16 or 24 weeks, animals exposed to cobalt showed reduced relative left ventricular weights, as well as diminished left ventricular function at both time points, with animals at the later time point demonstrating greater impairment. Groups of guinea pigs exposed to 20 mg cobalt/kg-day as cobalt sulfate in the diet for 5 weeks resulted in decreased absolute and relative heart weights and a greater incidence of abnormal electrocardiograms compared to animals not exposed to cobalt (Mohiuddin et al., 1970). Cellular alterations (observed at the light and electron microscopic level) in cardiac tissues included pericardial thickening and inflammation, myocardial degeneration and vacuolization, endocardial thickening, and myofibrillar damage.

The thyroid also appears to be a target for cobalt. Treatment of patients with 1 mg cobalt/kg/day as cobalt chloride for 2 weeks resulted in a greatly reduced uptake of radioactive iodine by the thyroid in 1 week, with uptake nearing 0 by the second week (Roche and Layrisse, 1956). When the cobalt treatment ended, the uptake values returned to normal. The decrease of radioactive iodine uptake found in patients administered 0.54 mg cobalt/kg/day for 10–25 days was found to result from cobalt blocking the organic binding of iodine (Paley et al., 1958).

Cobalt has been found to be a sensitizer in humans. Individuals are sensitized following dermal or inhalation exposure, but flares of dermatitis may be triggered following cobalt ingestion. One study was located that orally challenged cobalt-exposed workers in order to assess sensitization (Veien et al., 1987). In this study, several patients with eczema of the hands were challenged orally with 1 mg cobalt (0.014 mg cobalt/kg-day as cobalt sulfate) in tablet form once per week for 3 weeks; 28/47 patients had a flare of dermatitis following the oral challenge (Veien et al., 1987). All forty-seven patients had positive dermal patch tests to cobalt (13 to cobalt alone and 34 to nickel and cobalt) and 7 of the 13 patients that had patch-tested positive to cobalt alone reacted to the oral challenge. Comparing the results of the oral challenge and dermal patch tests, it was deduced that cobalt allergy was systemically induced. The exposure levels associated with sensitization to cobalt following inhalation or dermal exposure were not established.

Interrelationships have been found to exist between cobalt and nickel sensitization (Bencko et al., 1983; Rystedt and Fisher, 1983; Veien et al., 1987). In guinea pigs, nickel and cobalt sensitization appear to be interrelated and mutually enhancing (Lammintausta et al., 1985). Therefore, it is possible that in people sensitized by nickel, exposure to cobalt may result in an allergic reaction.

Four studies were located examining developmental effects of orally administered cobalt (given as cobalt chloride) in rodents (Domingo et al., 1985; Paternain et al., 1988; Pedigo and

Vernon, 1993; Seidenberg et al., 1986). Domingo et al. (1985) treated pregnant female rats with 5.4 to 21.8 mg cobalt/kg-day from gestation day 14 through lactation day 21. Maternal effects were not reported. Fetal effects at 5.4 mg cobalt/kg-day included stunted growth of the pups of both sexes, decreased body length and tail length in male offspring, and decreased spleen and liver weight in female offspring. Effects at the 10.9 mg cobalt/kg-day dose included decreased body weight in female pups, while at 21.8 mg cobalt/kg-day decreased survival was seen. These effects were at levels that were maternally toxic (authors did not specify the effects); therefore, the fetal effects at these levels may be a result of maternal toxicity rather than a direct effect of cobalt treatment.

No significant effects on fetal growth or survival were found in rats exposed to 6.2 to 24.8 mg cobalt/kg-day during gestation days 6-15 (Paternain et al., 1988), although a nonsignificant increase in the incidence of stunted fetuses was found in the animals treated with 12.4 or 24.8 mg cobalt/kg-day. Maternal effects, including reduced body weight gain and food consumption and altered hematological parameters, were reported at all exposure levels. No fetal effects were reported in mice exposed to 81.7 mg cobalt/kg-day during gestation days 8-12 (Seidenberg et al., 1986), but a significant decrease in maternal weight was found. Pedigo and Vernon (1993) exposed male rats to 93 mg cobalt/kg-day as cobalt chloride in the drinking water for 10 weeks, after which the males were mated with control females to examine for dominant lethal effects. Cobalt treatment resulted in a decreased percentage of pregnant females, decreased implantations per female, and increased preimplantation losses relative to controls. Recovery of reproductive function was seen by 8 weeks post-exposure.

Several studies reported testicular degeneration and atrophy in rats exposed to 6.1 to 24.4 mg cobalt/kg-day as cobalt chloride for 2-3 months in the diet or in the drinking water (Anderson et al., 1992, 1993; Corrier et al., 1985; Domingo et al., 1984; Mollenhauer et al., 1985; Nation et al., 1983; Pedigo et al., 1988). Pedigo et al. (1988) exposed male CD-1 mice to 100, 200, or 400 ppm of cobalt chloride (~6.1, 12.2, or 24.4 mg cobalt/kg-day, respectively) in the drinking water for 13 weeks. High-dose animals showed a significantly decreased testicular weight beginning at week 9 of treatment and a decreased epididymal sperm concentration by week 11 of treatment. All dose groups showed significantly decreased testicular weight and epididymal sperm concentration, and increased serum testosterone levels, by week 12 of exposure. Anderson et al. (1992, 1993) exposed groups of male CD-1 mice to 400 ppm of cobalt chloride (~24.4 mg cobalt/kg-day) in the drinking water for up to 13 weeks. A decrease in testicular weight and a progressive degeneration of the seminiferous tubules were seen beginning at 9 weeks of exposure, with no recovery seen after a 20-week non-exposure recovery period. Co-administration of 800 ppm of zinc chloride provided a partial protection against the effects of cobalt. Similar histology (degeneration of the testes, particularly the seminiferous tubules) was noted in Sprague-Dawley rats exposed to 20 mg cobalt/kg-day in the diet for up to 98 days (Corrier et al., 1985; Mollenhauer et al., 1985). Decreased testicular weight was seen in Sprague-

Dawley rats exposed to 500 ppm cobalt chloride (~17 mg cobalt/kg-day) for 3 months (Domingo et al., 1984).

Nation et al. (1983) exposed groups (n=6) of male Sprague-Dawley rats, 200-210 g, to diets containing 0, 5, or 20 mg cobalt/kg-day for a total of 69 days. Following 14 days of exposure, animals were trained for schedule (operant) or conditioned suppression neurobehavioral tests. Other than two seizures in the same high-dose animal, no overt signs of neurotoxicity were reported at any exposure level. A trend toward a decreased response rate in the schedule training behavior was observed in both the exposed groups, but only attained statistical significance in the high-dose animals near the end of the operant testing period (sessions 28-35, on exposure days 44-51). A trend toward decreased conditioned suppression behavior did not attain statistical significance in either group. Animals exposed to 20 mg cobalt/kg-day, but not 5 mg cobalt/kg-day, showed a significantly decreased weight of the testes following 69 days of exposure. This study established a NOAEL of 5 mg cobalt/kg-day and a LOAEL of 20 mg cobalt/kg-day for decreased testicular weight and changes in operant behavior in male Sprague-Dawley rats.

Several other studies have examined the effects of cobalt on neurobehavioral parameters (Bourg et al., 1985; Krasovskii and Fridlyand, 1971; Singh and Junnarkar, 1991). In groups of male Sprague-Dawley rats (n=8) exposed to 20 mg cobalt/kg/day as cobalt chloride for 57 days in the drinking water, cobalt enhanced behavioral reactivity to stress (the animals were less likely to descend from a safe platform to an electrified grid) (Bourg et al., 1985). Singh and Junnarkar (1991) reported a moderate reduction in spontaneous activity and mild hypothermia in rats exposed orally to 1/10 the LD50 of cobalt chloride or cobalt sulfate. Krasovskii and Fridlyand (1971) exposed groups of rats (number and sex not specified) to 0.05, 0.5, or 2.5 mg cobalt/kg-day for up to 7 months. Neurobehavioral tests showed that treatment with cobalt as cobalt chloride resulted in a significant increase in the latent reflex period at 0.5 mg cobalt/kg and above, and a pronounced neurotropic effect (disturbed conditioned reflexes) at 2.5 mg cobalt/kg.

DERIVATION OF A PROVISIONAL RfD FOR COBALT

The only known nutritional function of cobalt is as a vital component of vitamin B₁₂. All vitamin B₁₂ is derived from bacterial synthesis, so inorganic cobalt can be considered essential for animal species, such as ruminants, that depend totally on their bacterial flora for their vitamin B₁₂. This may apply to some degree also to humans with strict vegetarian diets, whose intake of pre-formed vitamin B₁₂ is severely limited (animal products are the primary source of vitamin B₁₂ in the diet). However, there is no evidence that the intake of cobalt is ever limiting in the human diet, and therefore no RDA is deemed necessary for cobalt (NRC, 1989). Recent data based on a 1984 FDA Total Diet Study (Pennington and Jones, 1987) have suggested that daily cobalt intakes in humans are in the 0.003-0.011 mg cobalt/day range. A recent survey of 5 Canadian

cities also suggested dietary intakes in the range of 0.007-0.015 mg cobalt/day for all age groups (Dabeka and McKenzie, 1995). Using body weights from NRC (1989), both of these studies would suggest daily intake levels on the order of 10^{-4} mg cobalt/kg-day. Older studies suggested that the average daily intake of cobalt in humans was higher, ranging from approximately 0.002 to 0.008 mg cobalt/kg-day in adults (Tipton et al., 1966; Schroeder et al., 1967) and 0.01 to 0.06 mg cobalt/kg-day in children (NRC, 1989; Murthy et al., 1971).

The most sensitive indicators of the effects of cobalt following oral exposure appear to be the increase of hemoglobin in both humans and animals and the elicitation of dermatitis in sensitized individuals. Cardiomyopathy is an endpoint of concern for cobalt in humans, but it is highly likely that alcohol consumed in “beer-cobalt cardiomyopathy,” as well as other factors, played a role in the effects that were seen. Other effects, including neurobehavioral, developmental, and testicular toxicity, were observed only in animals and at relatively high doses, and so were not considered critical for the risk assessment.

The elicitation of an allergic response in cobalt-sensitized workers was evaluated as a potential critical endpoint for the derivation of an oral RfD. However, the available data provide no information on the dose-response relationship of cobalt sensitization, nor is a NOAEL for the elicitation of the allergic response in humans defined. Interrelationships also exist between cobalt and nickel sensitization, so that people sensitized by nickel may have an allergic reaction following cobalt exposure. Sensitization was, therefore, not selected as the critical endpoint for RfD derivation.

Hematological effects of cobalt treatment (increased hemoglobin) have been reported in anemic dialysis patients (Duckham and Lee, 1976) and anephric patients (Taylor et al., 1977). In these patients, hemoglobin levels increased from levels clinically described as anemic to levels at or near “normal.” Thus, the effect of cobalt administration in these patients was clinically beneficial to these patients, and not adverse. However, hematologic effects of cobalt were also found in studies of normal humans (Davis and Fields, 1958) and rats (Krasovskii and Fridlyand, 1971), indicating that the effect is not limited to anephric individuals. Davis and Fields (1958) reported hemoglobin increases of 6-11% over “normal” in healthy volunteers given 0.96 mg cobalt/kg-day as cobaltous chloride. In contrast to the situation in the anemia patients studied by Duckham and Lee (1976) and Taylor et al. (1977), polycythemia in normal people is considered to be an adverse effect. Therefore, this is considered to be a suitable endpoint for derivation of an RfD.

The study by Duckham and Lee (1976) was used as the critical study for derivation of the RfD. The study by Taylor et al. (1977) reported similar effects at similar exposure levels, but the Duckham and Lee (1976) study described the study results and methods in greater detail. The study by Davis and Fields (1958) in normal humans used a higher dose level. While the cobalt in the Duckham and Lee (1976) study was used therapeutically, it represents an effect on the most

sensitive endpoint in a sensitive population of humans, and is, therefore, appropriate for use as the critical study for derivation of an RfD. The provisional RfD was derived from the LOAEL of 0.18 mg cobalt/kg-day as follows:

$$\text{RfD} = \text{LOAEL/UF} \times \text{MF}$$

Where:

$$\text{LOAEL} = 0.18 \text{ mg/kg-day}$$

$$\text{UF} = 10 \quad \text{Made up of component factors accounting for the use of a LOAEL (3) and deficiencies in the database (3), primarily the absence of chronic oral data leading to the use of a subchronic study.}$$

$$\text{MF} = 1 \quad \text{Default value for modifying factor.}$$

Therefore:

$$\begin{aligned} \text{RfD} &= 0.18/10 \times 1 \\ &= \mathbf{0.02 \text{ or } 2E-2 \text{ mg/kg-day}} \end{aligned}$$

An uncertainty factor to protect sensitive individuals was not considered to be necessary since the critical study was performed in a sensitive human population. The RfD of 0.02 mg Co/kg-day is higher than most estimates of dietary intake of cobalt, which range from 0.0001 mg/kg-day in the most recent studies (Pennington and Jones, 1987; Dabeka and McKenzie, 1995) to 0.002-0.008 mg/kg-day in older studies in adults (Tipton et al., 1966; Schroeder et al., 1967) and 0.01-0.06 in one study in children (Murthy et al., 1971). This RfD may not be protective for people with hypersensitivity to cobalt.

STATEMENT OF CONFIDENCE

Confidence in the critical study is low-to-medium. The study examined a small number of subjects over a subchronic duration, but examined what appears to be a sensitive endpoint in a group of sensitive humans. Confidence in the database is medium. There are supporting studies in both anemic and normal humans, and also in animals. However, there are no chronic oral data and only limited data on developmental effects. Low-to-medium confidence in the provisional RfD results.

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May 13, 2008

Michael Sivak
U.S. EPA, Region 2

ASSISTANCE REQUESTED: PPRTVs for Dibenzofuran, Endosulfan, Hexachlorobutadiene and Iron
(*Onondaga Lake*)

ENCLOSED INFORMATION:

Attachment 1: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR DIBENZOFURAN (CASRN 132-64-9)**

Attachment 2: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR ENDOSULFAN (CASRN 115-29-7)
Derivation of an Oral Slope Factor**

Attachment 3: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR HEXACHLOROBUTADIENE (CASRN
87-68-3)**

Attachment 4: **PROVISIONAL PEER REVIEWED TOXICITY
INFORMATION FOR IRON (CASRN 7439-89-6) AND
COMPOUNDS Derivation of Subchronic and Chronic
Oral RfDs**

Attachment 5: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR IRON (CASRN 7439-89-6) AND
COMPOUNDS Derivation of an Inhalation RfC**

Attachment 6: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR IRON (CASRN 7439-89-6) AND
COMPOUNDS Derivation of a Carcinogenicity
Assessment**

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (6)

cc: STSC Files

6-11-2007

Provisional Peer Reviewed Toxicity Values for

Dibenzofuran
(CASRN 132-64-9)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor

p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR DIBENZOFURAN (CASRN 132-64-9)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

This document has passed the STSC quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

INTRODUCTION

RfD and RfC values for dibenzofuran (DBF) were not available on IRIS (U.S. EPA, 2007) or in the HEAST (U.S. EPA, 1997). There is a Class D cancer assessment on IRIS (U.S. EPA, 2007). Dibenzofuran was included in a Drinking Water Toxicity Profile from 1992 (U.S. EPA, 1992), although no oral toxicity value was listed. The Office of Water did not include dibenzofuran on the latest Drinking Water Regulations (U.S. EPA, 2006a) or the Drinking Water Contaminant Candidate List (U.S. EPA, 2006b). The CARA list (U.S. EPA, 1991, 1994)

included a Health Effects Assessment (HEA) (U.S. EPA, 1987) and a Reportable Quantity Document (U.S. EPA, 1989) for Dibenzofuran. The HEA concluded that additional toxicity testing was necessary and did not derive a toxicity value due to the lack of data (U.S. EPA, 1987). The 1987 HEA for Dibenzofuran neither identified nor included discussion of Thomas et al. (1940), the primary source of data used in this PPRTV document. By contrast, the 1989 Reportable Quantity Document for Dibenzofuran (U.S. EPA, 1989) used Thomas et al. (1940) as the basis for derivation of composite scores and the corresponding reportable quantities for dibenzofuran.

ATSDR had not published a Toxicological Profile for dibenzofuran (ATSDR, 2006). NTP did not study the toxicity of dibenzofuran (NTP, 2006). WHO (2006) provided no relevant information. Available data on carcinogenicity, mutagenicity, metabolism, and other biological effects were summarized for dibenzofuran by the National Cancer Institute (NCI, 2000). Data on the adverse health effects of various halogenated dibenzofurans were available; however, the biological activity varies greatly among these congeners. U.S. EPA (1986a) did not recommend risk assessment by analogy to any of these more widely studied chemicals. NCI (2000) reported that the most structurally related chemical was dibenzo-p-dioxin. NCI (1979) reported that no excess tumors were induced in rats or mice fed dibenzo-p-dioxin up to 10,000 ppm in the diet.

Updated literature searches for noncancer and cancer data were conducted for data available through April 2006. The databases searched included: TOXLINE, MEDLINE, CANCERLIT, CCRIS, TSCATS, HSDB, RTECS, GENETOX, DART/ETICBACK, and EMIC/EMICBACK. Inhalation RfC values were not derived for dibenzofuran, because no human or animal inhalation data were found and the marginal ingestion data seemed inadequate to consider for inter-route extrapolation. However, a subchronic oral p-RfD value was derived, based on a LOAEL point of departure (POD) in Thomas et al. (1940). Chronic toxicity of dibenzofuran is discussed in the appendix. No data were identified from which to derive cancer risk values.

REVIEW OF PERTINENT DATA

Human Studies

Two cross-sectional studies of exposed workers were identified in the OPPT TSCATS database (Koppers 1980a,b). However, these studies reported exposures to dibenzofuran only in complex mixtures of coal tar products. Neither report noted adverse health effects that could be attributed to dibenzofuran exposure. Existing review documents and a detailed literature search identified no other data regarding the toxicity of dibenzofuran in humans.

Animal Studies

The only long-term toxicity data available for dibenzofuran were from a 200-day rat feeding study reported by Thomas et al. (1940). However, this document also will address the NCI (1979) data for dibenzo-p-dioxin, which NCI (2000) considered to be the chemical most structurally related to dibenzofuran.

NCI (1979) reported that unsubstituted dibenzo-p-dioxin, a structural analog of dibenzofuran, exhibited very low toxicity and no evidence of carcinogenicity in Osborne-Mendel rats and B6C3F1 mice, even when the maximum tolerated dose was approached (10,000 ppm in diet). Groups of 35 rats of each gender ingested dibenzo-p-dioxin at 5000 or 10,000 ppm in diet for 110 weeks. Groups of 50 mice of each gender ingested the same doses for 87 or 90 weeks. Controls consisted of groups of 35 untreated rats of each gender and 50 untreated mice of each gender. Mean body weights of the dosed male and female rats and mice were lower than those of the corresponding controls; the depression in the amount of weight gained in the dosed male mice was, however, relatively slight. Except for the male rats, survival at the end of the bioassay was lower in the dosed groups of both rats and mice than in the corresponding control groups. At week 90, at least 57% of the rats and 54% of the mice were still alive. In some male and female rats there was a dose-related increase in the incidence of hepatotoxic alterations characterized by fatty metamorphosis or necrosis. Also in mice, toxic hepatic lesions including liver degeneration, necrosis, fibrosis and/or cirrhosis were observed in slightly increased numbers in the dosed mice — particularly in the high-dose females. No tumors were induced in rats or mice of either gender at incidences that were significantly higher in the dosed groups than in the corresponding control groups. The authors concluded that unsubstituted dibenzo-p-dioxin exhibited very low toxicity and was noncarcinogenic in Osborne-Mendel rats and B6C3F1 mice, even when the maximum tolerated dose was approached (10,000 ppm in diet).

The Thomas et al. (1940) report consisted of two studies, a primary 200-day dibenzofuran feeding study and a follow-up 78-day study. In the primary study, groups of five female albino rats (strain not specified), approximately 30 days old, consumed 0, 250, 500, 1000, 2000, or 4000 ppm of dibenzofuran in their food for 200 days. In addition, two female rats consumed 8000 ppm of dibenzofuran in their diet for a shorter period (approximately 100 days). According to the authors, none of the animals exhibited any abnormal activity or behavior, nor was food intake appreciably altered by dibenzofuran administration, although it was noted that the rats receiving dibenzofuran tended to consume more water than controls. The authors also reported no effect on body weight gain at any dose during the exposure period; however, decreases in body length and absolute organ weights were observed in all dibenzofuran-exposed groups at necropsy. The authors also reported that the treated animals had unusually large amounts of abdominal fat, which they interpreted as accounting for the lack of effect on body weight gain. Quantitative data were not provided to support the assertions of no appreciable

changes in food intake or body weight gain, decreases in organ weight and overall length, and excess abdominal fat. In addition, the authors did not report whether a dose-response effect was observed for changes in body length or organ weight, or for excess abdominal fat.

Histological examination of the liver, kidney, spleen, heart, and adrenals was performed in rats exposed to dibenzofuran at 500 ppm and higher, and in the control animals (Thomas et al., 1940). The low dose group (250 ppm) apparently was not examined for histopathology. In the kidney, histological examination of rats exposed to concentrations of 500 ppm and higher revealed fine, brown-pigmented granules in the epithelial cells of proximal convoluted tubules in the deeper parts of the renal cortex. This effect was noted among all rats receiving dibenzofuran, and both the amount of pigmented material within cells and the frequency of occurrence among cells increased with dose of dibenzofuran. In addition, the two rats fed diet containing 8000 ppm dibenzofuran exhibited prominent, irregular dilatation of the collecting tubules with coagulated material resembling protein; other tubules in these two rats were slightly dilated and contained more granular and amorphous material than controls. These effects were reported as occurring without cellular degeneration or glomerular abnormalities. Some (frequency not specified) of the kidneys from rats receiving 4000 ppm showed similar, but less severe, changes. These lesions were not reported among rats fed the lower doses of dibenzofuran. However, quantitative data were not reported. In the spleen, slight hyperplasia of the Malpighian bodies was reported among several rats (frequency not given) in the 4000 and 8000 ppm groups. No alterations, other than reduced organ weight, were noted in the liver, heart, or adrenals of the treated rats.

In the follow-up study to determine whether dietary dibenzofuran affected water balance, an effect noted qualitatively (increased water consumption) in female rats receiving dibenzofuran in their food, Thomas et al. (1940) exposed groups of five male rats (average initial body weight 255 grams) to 0 or 5000 ppm of dibenzofuran in the diet for 78 days. Treated rats exhibited greater water consumption and urine output than controls, suggesting that dibenzofuran altered water balance. The excess in urine output was greater than the excess in water consumption in the treated group, suggesting a slight dehydration of tissues. The authors reported that no alterations in hematological parameters were observed (hemoglobin and erythrocyte, leukocyte, and reticulocyte counts). Tables 1 and 2 have summarized the hematological data reported in the 78-day study.

TABLE 1. Blood cell types in rats exposed to DBF in normal diet for 78 days					
Dose	Rats "N"	Hemoglobin	Erythrocytes	Reticulocytes	White cells
0	10	16.3%	8.12×10^6	3.0%	1.44×10^4
5000 ppm	5	16.6%	9.07×10^6	2.35%	1.65×10^4

TABLE 2. Average differential white blood cell counts in 78-day exposed rats vs. "normal rat blood"

Dose	Rats "N"	Lymphocytes	Polymorphonuclear neutrophils	Monocytes	Basophiles	Eosinophils
"Normal"	---	67.9%	27%	5.3%	0.77%	2.1%
5000 ppm	5	63.8%	33.5%	1.18%	0.64%	0.94%

In contrast to qualitative observations reported among the female rats exposed to similar concentrations in the 200-day primary study, the male rats treated for 78 days tended to consume less food than the controls and had a slightly lower rate of body weight gain than the control group. These data and water consumption data are summarized in Table 3. The authors noted that the odor and taste of dibenzofuran at 5000 ppm in the food was distinctly noticeable and may have contributed to this effect. Histological examination was not performed on tissues from these rats.

TABLE 3. Weight gain in male albino rats fed DBF for 78 days vs. controls

Dose	Rats "N"	Weight gain	Food ingestion	Water ingestion
0	5	321 g	6108 g	9652 cc
5000 ppm	5	243 g	5482 g	10,316 cc
Difference	-----	78 g (24%)	626 g (10%)	664 cc (6.9%)

The literature search revealed additional, peripheral data for dibenzofuran, including those for soil nitrification organisms (Sverdrup et al., 2002), drought resistance of certain insects (Sjursen et al., 2001), plant seedling growth (Sverdrup et al., 2003), fungi-specific enzyme systems (Kurihara et al., 2002), and a study of human intellectual effects of exposure (Schantz, 2001) that mistakenly referred to unhalogenated dibenzofuran. Abstracts for these studies reported the following conclusions.

- 75 mg DBF/kg (soil) NOEL for soil nitrification and no effects on soil bacterial diversity (Sverdrup et al., 2002)

- No dose-related decrease in drought tolerance in adult soil-dwelling insects, *Folsomia fimetaria* (Sjursen et al., 2001)
- 20% reduction in plant seedling weight when exposed to 43-93 mg DBF /kg soil (Sverdrup et al., 2003)
- No change in expression of NADH-ubiquinone oxidoreductase (NUO) among DBF-exposed fungus, *Phanerochaete chrysosporium* (Kurihara et al., 2002)

DERIVATION OF A PROVISIONAL SUBCHRONIC ORAL RfD VALUE FOR DIBENZOFURAN

The only subchronic or chronic toxicity data available for dibenzofuran were from the 200-day and 78-day feeding studies described by Thomas et al. (1940). These studies, though of apparently high quality for their era, had a number of major short comings, including the following:

- only qualitative data were reported for most endpoints
- only five organs were examined in the pathology
- the lowest dose group was not subjected to pathology examinations

No pertinent developmental or reproductive data were found for dibenzofuran. The LOAEL data from the Thomas et al. (1940) 200-day feeding study provided the POD for this derivation, because no NOAEL was reported. Data from the 78-day study were used to confirm food ingestion rates estimated using default rates in U.S. EPA, 1986b. Benchmark dose modeling was considered infeasible because adverse effects and the dose-response nature of the response were reported only qualitatively.

The lowest dose tested in the 200-day Thomas et al., 1940 study, 250 ppm in diet, was selected as the LOAEL POD for the aggregate critical effects of reduced length and organ weight, and excess abdominal fat. Ingestion data from the 78-day study was used to estimate the actual doses to the animals treated at the LOAEL, as follows. The 78-day feeding study was conducted under the same conditions as the 200-day primary study. This estimation made the following assumptions.

- Data from the 78-day study (Thomas et al., 1940) were more likely to represent actual food intakes than the default reference food factor from U.S. EPA, 1986b
- Rats in the 200-day study (Thomas et al., 1940) eating a diet treated with 250 ppm dibenzofuran consumed quantities of food closer to the control amounts (6108 g/diet/5 rats) than to the quantities of food treated with 5000 ppm dibenzofuran (5482 g/5 rats) in the 78-day study

- Growth of rats eating the 250 ppm diet in the 200-day study (Thomas et al., 1940) more closely approximated controls than those eating 5000 ppm, and that the 78-day weight provided a reasonable average weight for the 200 day study period.

In the 78-day study, Thomas et al. (1940) reported that a group of 5 control rats ingested a total of 6108 grams of food over the 78 days and grew from 1.273 kg to 1.594 kg/group, while experimental rats ingested 5482 g of food treated with 5000 ppm dibenzofuran and grew from 1.274 kg to 1.517 kg/group of 5 treated rats. The following calculations used food consumption data from the 78-day study to estimate dibenzofuran consumption in the 200-day study at the POD (250 ppm) for the critical effect of reduced length and organ weight, and excess abdominal fat among the exposed rats.

$$(6108 \text{ g diet}/5 \text{ rats}) / 78 \text{ days} = 78.3 \text{ g/diet}/5 \text{ rats/day}$$

$$(78.3 \text{ g}/5 \text{ rats/day}) \times (250/10^6) = 0.0196 \text{ g DBF}/5 \text{ rats/day} = 19.6 \text{ mg}/5 \text{ rats/day}$$

$$19.6 \text{ mg DBF}/5 \text{ rats/day} / (1.594 \text{ kg}/5 \text{ rats}) = 12.3 \text{ mg DBF}/\text{kg/day}$$

The estimated dibenzofuran dose of 12.3 g/kg/day was essentially the same as the dose of 12.5 g/kg/day calculated using the EPA default reference food factor (U.S. EPA, 1986b).

Based on the data available, the following uncertainty factors were applied to derive a subchronic oral p-RfD.

- 10 for variability in human susceptibility
- 10 for the uncertainty in animal-to-human extrapolation
- 1 for using data from a 200-day study (in rats) to derive a subchronic p-RfD
- 3 ($10^{0.5}$) for using a minimal LOAEL instead of a NOAEL
- 10 for deficiencies in the database, including the lack of reproductive and developmental data, and the minimal data details reported in the key study

The uncertainty factors noted above provide a composite UF of 3000 ($10^{3.5}$).

In the absence of a NOAEL, a LOAEL could be several orders of magnitude above the actual no adverse effect dose, since it merely represents the lowest dose tested. Nevertheless, the uncertainty factor for using a minimal LOAEL instead of a NOAEL was reduced from 10 to 3 ($10^{0.5}$) because the following findings suggested that the smaller uncertainty factor would be more appropriate in this case. While many of the dose levels tested and the organism effects considered in the following reports would be difficult to relate to humans, together they seem to

emphasize the relatively low toxicity and mild effects of dibenzofuran across a variety of species.

- The Thomas et. al (1940) study noted relatively minor effects in rats, even at very high doses, up to thirty times the LOAEL dose selected as the POD
- Peripheral data in other species indicated very minor effects or no effects among organisms exposed to dibenzofuran
 - 75 mg DBF/kg (soil) NOEL for soil nitrification and for soil bacterial diversity (Sverdrup et al., 2002)
 - No dose-related decrease in drought tolerance in the adult soil-dwelling insects, *Folsomia fimetaria* (Sjursen et al., 2001)
 - 20% reduction in plant seedling weight when exposed to 43-93 mg DBF /kg soil (Sverdrup et al., 2003)
 - No change in expression of NADH-ubiquinone oxidoreductase (NUO) among DBF-exposed *Phanerochaete chrysosporium* fungi (Kurihara et al., 2002)
- NCI (1979) reported no tumors and relatively low toxicity among rats and mice fed diets containing 5000 ppm and 10,000 ppm dibenzo-p-dioxin, a structural analog to dibenzofuran. Effects reported were hepatic lesions, slight reductions in weight gain and nephropathy (in male rats)

Applying the composite UF of $10^{3.5}$ (~3000) to the dietary LOAEL POD of 12.3 mg DBF/kg-day for the combined critical effects of reduced length and organ weight and excess abdominal fat observed in female albino rats allowed the following calculation of the subchronic p-RfD.

$$\begin{aligned}
 \text{Subchronic oral p-RfD} &= \text{LOAEL} / (\text{UF} \times \text{MF}) \\
 &= (12.3 \text{ mg/kg/day}) / (10^{3.5} \times 1) \\
 &= 4 \times 10^{-3} \text{ mg/kg-day} \\
 &= 4 \text{ } \mu\text{g dibenzofuran/kg-day}
 \end{aligned}$$

The data were insufficient to derive a chronic oral p-RfD value using an acceptable composite uncertainty. However, the Appendix of this document contains a Screening Value that may be useful in certain instances. Please see the attached Appendix for details.

DERIVATION OF PROVISIONAL INHALATION RfC VALUES FOR DIBENZOFURAN

Provisional inhalation RfC values were not derived for dibenzofuran because no useful inhalation exposure data were identified and data were insufficient to attempt inter-route extrapolation from the marginal ingestion data.

STATEMENT OF CONFIDENCE

Confidence in the principal study is low. Thomas et al. (1940) examined a number of endpoints, including histological examination of several major organs. The study had an adequate number of dose groups, but was limited by inclusion of only five rats in each group. Although only female rats were used for the 200-day portion of the study, male rats were used for the shorter water balance study (78 days). Thomas et al. (1940) did not report whether the critical effect selected displayed a dose-response relationship. However, the reductions in growth and organ weights, and the increase in abdominal fat were supported by histological changes noted in the kidney and impairment of water balance at higher doses. Because the critical effects were observed among rats receiving the lowest dose tested, one cannot be certain that the effects noted at 250 ppm (12.5 mg/kg-day), would not have been present at lower doses. Thus, it is uncertain whether 250 ppm is a true LOAEL. Confidence in the database and the resulting RfDs is low because of the limited toxicity data base for dibenzofuran, including lack of human studies and chronic, developmental, or reproductive oral animal studies. However, some confidence is gained from the relatively low toxicity and lack of tumors among rats and mice fed high doses of dibenzo-p-dioxin (NCI, 1979), the chemical identified by NCI (2000) as most structurally related to dibenzofuran. Nevertheless, risk managers are advised to consider any other available data before applying this p-RfD.

Suppliers and users of dibenzofuran should be encouraged to conduct toxicology studies, such as that initiated by EPA in 1978 (NCI, 2000) but then terminated because of lack of funding. The absence of inhalation, toxicokinetic, and metabolic data would justify especially encouraging studies to seek such information.

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APPENDIX

DERIVATION OF A SCREENING VALUE FOR DIBENZOFURAN

For reasons noted in the main PPRTV document, it is inappropriate to derive provisional toxicity values for Dibenzofuran, chronic RfD. However, information is available for this chemical which, although insufficient to support derivation of a provisional toxicity value, under current guidelines, may be of limited use to risk assessors. In such cases, the Superfund Health Risk Technical Support Center summarizes available information in an Appendix and develops a "Screening Value." Appendices receive the same level of internal and external scientific peer review as the PPRTV documents to ensure their appropriateness within the limitations detailed in the document. In the OSRTI hierarchy, Screening Values are considered to be below Tier 3, "Other (Peer-Reviewed) Toxicity Values."

Screening Values are intended for use in limited circumstances when no Tier 1, 2, or 3 values are available. Screening Values may be used, for example, to rank relative risks of individual chemicals present at a site to determine if the risk developed from the associated exposure at the specific site is likely to be a significant concern in the overall cleanup decision. Screening Values are not defensible as the primary drivers in making cleanup decisions because they are based on limited information. Questions or concerns about the appropriate use of Screening Values should be directed to the Superfund Health Risk Technical Support Center.

The Thomas et al. (1940) study provided insufficient data to derive a chronic oral p-RfD value with uncertainty in an acceptable range. The 200-day rat minimal LOAEL POD of 12.3 mg/kg-day was considered to derive a **screening chronic oral reference dose** by applying a composite uncertainty factor of 10,000 (10^4), including 10 for variability in human susceptibility, 10 for animal-to-human extrapolation, 3 ($10^{0.5}$) for extrapolating from 200-day rat data to a chronic screening value, 3 ($10^{0.5}$) for using a minimal LOAEL instead of a NOAEL, and 10 for deficiencies in the database, including the lack of developmental data and the minimal data details reported in the key study.

Applying the minimal LOAEL dietary POD of 12.3 mg DBF/kg-day and the composite uncertainty factor of 10,000 (10^4) allowed the following calculation:

$$\begin{aligned}
 \text{Screening chronic oral p-RfD} &= \text{LOAEL/UF} \\
 &= (12.3 \text{ mg/kg-day})/10^4 \\
 &= \underline{\underline{1 \times 10^{-3} \text{ mg/kg-day}}} \\
 &= 1 \text{ } \mu\text{g dibenzofuran/kg-day}
 \end{aligned}$$

Confidence in the key study was low, because of the lack of detail on the critical effects and other deficiencies noted in this document. Given the lack of additional studies, confidence in the database also was low, leading to low overall confidence in the screening toxicity value. Users are advised to consider any other available data and to consult with the STSC before using this screening p-RfD.

Provisional Peer Reviewed Toxicity Values for
Endosulfan
(CASRN 115-29-7)

Derivation of an Oral Slope Factor

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit

NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
ENDOSULFAN (CASRN 115-29-7)
Derivation of an Oral Slope Factor**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Endosulfan (CASRN 115-29-7) is a mixture of two stereoisomers, approximately 70% endosulfan I (endosulfan α ; CASRN 959-98-8) and 30% endosulfan II (endosulfan β ; CASRN 33213-65-9). An assessment of the carcinogenicity of endosulfan is not available on IRIS (U.S. EPA, 2002) or in the HEAST (U.S. EPA, 1997) or Drinking Water Standards and Health Advisories list (U.S. EPA, 2000). The CARA list (U.S. EPA, 1991a, 1994) includes a Health Effects Assessment for α - and β -endosulfan (U.S. EPA, 1987) that assigned endosulfan to cancer weight-of-evidence Group D, not classifiable as to human carcinogenicity, based on inconclusive animal data. A subsequent Health and Environmental Effects Document (U.S. EPA, 1991b) also assigned endosulfan to Group D. Based on more recent adequate negative studies, the Office of Pesticide Programs has classified endosulfan in Group E (U.S. EPA, 1999). IARC (2002) has not evaluated endosulfan for carcinogenicity. Review documents by ATSDR (2000) and WHO (1984), as well as the NTP (2002) status reports, were also consulted for relevant information. Literature searches were conducted from 1998 to December 2001 for studies relevant to the

derivation of an oral slope factor for endosulfan. The databases searched were: TOXLINE, MEDLINE, CANCERLIT, RTECS, GENETOX, HSDB, CCRIS, TSCATS, EMIC/EMICBACK and DART/ETICBACK.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

Case control studies of 261 patients with breast cancer (Ashengrau et al., 1998) and 30 patients with gall bladder carcinoma (Shukla et al., 2001) did not find associations between serum levels of endosulfan and cancer.

Animal Studies

The available carcinogenicity studies for endosulfan following oral exposure have been reviewed previously (U.S. EPA, 1987, 1991b, 1999; ATSDR, 2000; WHO, 1984). There was no evidence of carcinogenicity in male or female NMRI mice fed endosulfan in the diet at concentrations up to 18 ppm (2.5 mg/kg-day) for 2 years (Hoechst Celanese Corporation, 1988; Hack et al., 1995), in male or female Sprague-Dawley rats fed up to 75 ppm (3.25 mg/kg-day) for 2 years (Hoechst Celanese Corporation, 1989; Hack et al., 1995), or in male or female Wistar rats fed up to 100 ppm (8 mg/kg-day) for 2 years (Keller, 1959). Oral exposure (gavage followed by diet) of male and female B6C3F1 and B6AKF1 mice to 1.0 or 2.15 mg/kg-day of endosulfan for 73-76 weeks produced some suggestive findings (statistically significant elevations in total tumor incidence and pulmonary adenomas in all treatment groups combined), but these were not considered biologically relevant because no significant differences were apparent for individual endosulfan treatment groups, and because no pulmonary carcinomas were diagnosed in endosulfan-treated animals (Innes et al., 1969; NCI, 1968). Low survival in all treated B6C3F1 mice and high-dose B6AKF1 mice complicates interpretation of this study.

The results of a subsequent NCI (1978) study were inadequate for evaluation of carcinogenicity. No evidence of carcinogenicity was observed in male or female B6C3F1 mice fed up to 6.9 or 3.9 ppm (1.3 or 0.76 mg/kg-day), respectively, for 78 weeks, female Osborne-Mendel rats fed up to 445 ppm (39 mg/kg-day) for 71 weeks, or male Osborne-Mendel rats fed up to 952 ppm (75 mg/kg-day) for 72-82 weeks. The maximum tolerated dose was clearly exceeded, as evidenced by high mortality in male rats and mice and other serious non-neoplastic effects (weight loss, kidney and testicular damage) in all treated rat groups. A re-evaluation of the histology slides (Reuber, 1981) reported statistically significant increases in certain types of tumors grouped across tissues in female rats (total neoplasia, malignant tumors, sarcomas, lymphosarcomas and reproductive system tumors) and male rats (endocrine organ tumors). The incidence of parathyroid adenomas in male rats was also reported to be increased. In mice, the re-evaluation found a marginally significant increase in the incidence of liver carcinomas in low-dose females, but not in high-dose females or males. Reuber (1981) failed to report details

regarding definitions of neoplasia used, tissue occurrence of neoplasia observed, and how his data compare with data from the original study (NCI, 1978). The conclusions of the reevaluation have not been independently confirmed.

Other Studies

Evidence of hepatic tumor-promoting activity was observed in one of two studies in male Sprague-Dawley rats initiated by partial hepatectomy and nitrosodiethylamine treatment. Flodstrom et al. (1988) did not observe an increase in hepatic foci positive for γ -glutamyltranspeptidase in rats exposed to α -endosulfan, β -endosulfan, or technical endosulfan for 10 weeks at doses up to 5 mg/kg-day. In contrast, Fransson-Steen et al. (1992) observed statistically significant increases in the number and volume of hepatic foci positive for γ -glutamyltranspeptidase in larger test groups of male rats fed α -endosulfan, β -endosulfan, or technical endosulfan for 20 weeks up to 15 mg/kg-day. Based on observations that endosulfan has exhibited activity as an endocrine disruptor (U.S. EPA, 1999) and induced proliferation in hormone-responsive human (endometrial and breast) cancer cell lines (Coumoul et al., 2001; Soto et al., 1994; Vonier et al., 1996; others), a hypothesis has been suggested that endosulfan may promote cancer formation in humans through a mode-of-action involving endocrine disruption. However, other studies have produced conflicting results (e.g., Arcaro et al., 1998; Newbold et al., 2001) and insufficient data are available to evaluate this theory.

Reviews generally consider endosulfan to be genotoxic (U.S. EPA, 1991b, 1999; ATSDR, 2000; WHO, 1984). Extensive mutagenicity testing in *Salmonella typhimurium* and *Escherichia coli* strains reported both positive and negative results with and without metabolic activation. Conflicting positive and negative results were also seen in assays for mutation, gene conversion and chromosome aberrations in *Saccharomyces cerevisiae*, although no mutations were seen in *Schizosaccharomyces pombe*. Similarly, both positive and negative tests for gene mutation have been observed in cultured mouse lymphoma cells with and without metabolic activation. Endosulfan did not induce unscheduled DNA synthesis in primary rat hepatocytes. Endosulfan induced micronuclei in cultured sheep lymphocytes and sister chromatid exchange in both preimplantation embryos of hybrid mice and human lymphoid cells *in vitro*. Endosulfan also induced chromosome aberrations in bone marrow cells of Syrian hamsters. Both positive and negative results were seen in assays measuring the formation of micronucleated polychromatic erythrocytes in mice. Endosulfan induced sex-linked recessive lethal mutations and sex-chromosome loss in *Drosophila*. Both positive and negative results have been observed in dominant lethal mutation studies in male mice. A cluster of four women living near endosulfan-contaminated areas in Florida produced five children born with evidence of mitochondrial defects (global developmental delay and hypotonia, carnitine deficiency and β -hydroxy butyrate anomalies) suggestive of mitochondrial DNA damage (Thrasher, 2000). However, it is not clear that these effects can be attributed to endosulfan exposure.

FEASIBILITY OF DERIVING A PROVISIONAL ORAL SLOPE FACTOR FOR ENDOSULFAN

No evidence of carcinogenicity resulting from endosulfan exposure was observed in epidemiology or animal studies. On the basis of the available information, it is not possible to derive a provisional oral slope factor for endosulfan.

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Provisional Peer Reviewed Toxicity Values for
Hexachlorobutadiene
(CASRN 87-68-3)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
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Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR HEXACHLOROBUTADIENE (CASRN 87-68-3)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and

circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

No reference dose (RfD) or reference concentration (RfC) values are available for hexachlorobutadiene (HCBd) in the Integrated Risk Information System (IRIS) database (U.S. EPA, 2007). The Health Effects Assessment Summary Table (HEAST) lists a chronic oral RfD of 2E-4 mg/kg-day and no subchronic RfD (U.S. EPA, 1997). The source documents referenced for the RfD value in HEAST included a 2-year dietary study in rats (Kociba et al., 1977) and a 13-week dietary study in mice (NTP, 1991; Yang et al., 1989). The chronic oral RfD value cited in HEAST was derived from a LOAEL of 0.5 mg/kg-day, based on renal tubule regeneration observed in a 13-week dietary study in mice (NTP, 1991; Yang et al., 1989). The Drinking Water Standards and Health Advisories also includes an RfD of 2E-4 mg/kg-day for HCBd (U.S. EPA, 2004). The Chemical Assessments and Related Activities (CARA) list (U.S. EPA, 1991a, 1994) identifies a Health Effects Assessment (HEA) (U.S. EPA, 1984) and a Drinking Water Health Advisory report (U.S. EPA, 1987). No oral or inhalation RfD values were provided in the HEA (U.S. EPA, 1984). An RfD value of 0.002 mg/kg-day was calculated for use in the derivation of the drinking water equivalent level (DWEL) (U.S. EPA, 1987), based on kidney effects observed in the 2-year dietary study in rats (Kociba et al., 1977). A no-observed-adverse-effect level (NOAEL) of 0.2 mg/kg-day was identified from this study, based on functional and histopathological changes in the kidney, and a composite uncertainty factor (UF) of 100 was applied to account for interspecies and interindividual differences.

An Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for HCBd (ATSDR, 1994) derived an intermediate-duration oral Minimal Risk Level (MRL) of 0.0002 mg/kg-day, based on the presence of kidney damage in female mice from a 13-week

dietary study (NTP, 1991). A lowest-observed-adverse-effect level (LOAEL) value of 0.2 mg/kg-day was identified, based on tubular cell degeneration and regeneration in the renal cortex, and a composite uncertainty factor (UF) of 1000 was applied to derive the intermediate-duration oral MRL (factors of 10 each to account for the interindividual variation in the human population, the uncertainty in extrapolating animal data to the case of human and uncertainty in using LOAEL data rather than NOAEL data). Because renal tubular hyperplasia was observed at 2 mg/kg-day in a chronic dietary study in rats (Kociba et al., 1977) and no effect was seen at 0.2 mg/kg-day in this study (the LOAEL for kidney effects in the 13-week mouse study), the intermediate-duration MRL was considered protective for chronic exposures and a chronic MRL was not proposed. Inhalation MRL values were not derived by ATSDR for HCBd due to the lack of sufficient data to identify a target organ or reliable NOAEL values (ATSDR, 1994). Occupational exposure standards and guidelines for HCBd, based on skin irritation and kidney effects, include American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value–time-weighted average (TLV-TWA) and National Institute for Occupational Safety and Health (NIOSH) TWA values of 0.02 ppm (0.24 mg/m³) (ACGIH, 2005; NIOSH, 2005). An Occupational Safety and Health Administration (OSHA) Permissible Exposure Limit (PEL) value is not available for HCBd (OSHA, 2006).

A cancer assessment for HCBd is available on IRIS (U.S. EPA, 2007), in the HEAST (U.S. EPA, 1997), and on the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). HCBd is considered to be a possible human carcinogen (Group C) based on kidney tumors observed in male and female rats from one study. An oral slope factor of 0.078 (mg/kg-day)⁻¹ was derived, based on renal tubular adenomas and adenocarcinomas observed in rats given HCBd in the diet (Kociba et al., 1977). An inhalation unit risk value of 2.2×10⁻⁵ (µg/m³)⁻¹ was calculated based on route extrapolation from the oral data (U.S. EPA, 2007). The International Agency for Research on Cancer (IARC) assigned HCBd to Group 3 (not classifiable as to its carcinogenicity to humans), based on limited evidence for the carcinogenicity of HCBd in animals and inadequate evidence in humans (IARC, 1999). The World Health Organization (WHO) Environmental Health Criteria document (WHO, 1994) also indicated that there was limited evidence for carcinogenicity of HCBd in animals and insufficient evidence in humans. HCBd was not included in the NTP (2005) 11th Report on Carcinogens.

Literature searches were performed for the time period of 1965 to May, 2006 in TOXLINE, MEDLINE (plus PubMed cancer subset) and DART/ETICBACK. An update search of the TOXCENTER (BIOSIS) database was performed for the time period of 2000 to May, 2006. Databases searched without date limitations included TSCATS, RTECS, GENETOX, HSDB and CCRIS. Search of Current Contents encompassed November 2005 to May 2006.

REVIEW OF PERTINENT DATA

Human Studies

Oral Exposure. No data were located regarding the oral toxicity or carcinogenicity of HCBd in humans.

Inhalation Exposure. Very little information pertaining to effects of inhalation of HCBd in humans is available. Howse et al. (2001) investigated biomarkers of early renal dysfunction in a cohort of subjects exposed to HCBd. This study was presented as an abstract only and few details were provided regarding the subject cohort or the nature of the exposure to HCBd. Urinary markers of renal disease were evaluated in 70 subjects known to be environmentally exposed to HCBd. Twenty-five subjects were eventually eliminated from consideration due to age, preexisting renal disease, medication use or exposure to other nephrotoxic compounds. The parameters investigated for the remaining 45 subjects included urinary albumin, total protein, γ -glutamyl transpeptidase (GGT), N-acetyl- β -glucosaminidase (NAG), leucine aminopeptidase (LAP), α - and π -glutathione transferases (GST) and retinol binding protein (RBP). Results were compared to the laboratory reference range for healthy workers. Urinary abnormalities occurred in 21 subjects, with 11 subjects exhibiting 2 or more abnormal tests. The most common effects were seen with the tubular markers LAP, GGT and α - and π -GST. No further information was provided.

Driscoll et al. (1992) carried out a study investigating liver dysfunction in workers exposed to a variety of chlorinated solvents (mainly carbon tetrachloride and perchlorethylene) and HCBd at a solvent production plant. The study included all 53 members of the workforce, but a number of individuals were excluded from the analysis because their blood samples were inadequate (6 individuals), they had not fasted before the blood samples were taken (11 individuals) or were taking antibiotics (1 individual). This left 35 subjects who were included in the analysis. Workers were categorized in relation to both HCBd exposure and overall solvent exposure at the plant. The results of repeated environmental monitoring in the plant were used to assign each worker to one of four classes of exposure to HCBd (0.0, 0.005, 0.01 or 0.02 ppm). Overall solvent exposure for all workers was low (less than 1 ppm), but varied with task; routine monitoring data from the plant records were used to assign workers to either a “lower” or “higher” solvent exposure category. Workers assigned to the various categories were similar in age and duration of employment.

Blood samples were collected from each worker after an overnight fast (Driscoll et al., 1992). Serum bile acids were assayed by high performance liquid chromatography and compared for each group. Standard tests for liver function [serum protein, albumin and bilirubin concentrations and alkaline phosphatase (AP), alanine aminotransferase (ALT), aspartate aminotransferase (AST) and GGT activities] were also carried out. Total bile acids were not significantly increased in relation to HCBd exposure, but a positive exposure-effect relationship with HCBd concentration was found for three individual bile acids (deoxycholic acid, glycine deoxycholic acid, taurine chenodeoxycholic acid) and for total deoxycholate (this includes deoxycholic acid and glycine deoxycholic acid). Using multiple linear regression and controlling for age and overall solvent exposure, these parameters had significant positive log-linear relations with exposure to HCBd. With respect to overall solvent exposure, there was no significant positive relationship for total bile acids or any individual bile acids (the researchers suggested that significant negative relationships with glycine deoxycholic acid, taurine cholate and total cholate may have resulted from misclassification of exposure for some workers).

Although liver function tests did not show any significant relationship with exposure to either HCBd or solvents overall in the Driscoll et al. (1992) study, serum bile acid

concentrations may be a more sensitive indicator of liver damage than standard tests of hepatic function. Franco et al. (1986) compared liver function in workers occupationally exposed to a mixture of organic solvents and an unexposed control group. The results from conventional tests of hepatic function were compared with those of the serum bile acid test, and the researchers concluded that the serum bile acid test had a higher sensitivity for the detection of liver dysfunction for the solvent mixture tested. The bile acid test results of Driscoll et al. (1992) suggest that exposure to HCBd may affect liver function. However, there was no supporting evidence for hepatotoxicity from standard liver function tests, workers were exposed to multiple solvents, the study did not include a control group of individuals unexposed to any solvents and the study did not assess possible confounders, such as previous hepatic disease and alcohol intake.

The only other available study of effects of HCBd in humans is a study carried out in Russia (Krasniuk et al., 1969). Krasniuk et al. (1969) recorded multiple toxic effects in vineyard workers seasonally exposed to HCBd (0.8-30 mg/m³) and polychlorobutane-80 (0.12-6.7 mg/m³) in the air over fumigated areas. A total of 205 workers were examined medically; 153 workers had 4 years of exposure to HCBd and polychlorobutane-80, while 52 workers had worked under the same conditions without exposure to the chemicals. The study reported multiple toxic effects in exposed workers, including the development of hypotension, cardiac disease, chronic hepatitis and disturbance of nervous function. The effects, however, are not well documented and cannot be attributed solely to HCBd.

Animal Studies

Oral Exposure.

Jonker et al., 1993 — A 4-week dietary study was conducted in Wistar rats (five/sex/group, 10 controls/sex) fed HCBd (98% pure) at concentrations of 0, 25, 100 or 400 ppm (Jonker et al., 1993). Using reference values for body weight and food consumption in Wistar rats from a subchronic study (male body weight 0.217 kg, female body weight 0.156 kg, male food consumption 0.02 kg/day, female food consumption 0.016 kg/day) (U.S. EPA, 1988), daily dose estimates were calculated to be 0, 2.3, 9.2 or 37 mg/kg-day for male rats and 0, 2.6, 10.2 or 41 mg/kg-day for female rats. During the 4th week of the study, rats were deprived of water for 24 hours and food for 16 hours. Urine was collected during the last 16 hours of water deprivation and urine volume and density were measured. Urine samples were also visually inspected and analyzed for pH, protein, glucose, ketones, occult blood, urobilinogen and bilirubin. Urine samples were centrifuged and the sediment was examined microscopically. Hematology parameters, including hemoglobin, packed cell volume, red blood cells (RBCs) and total white blood cells (WBCs), were evaluated for tail vein blood samples that were obtained during the 4th week of the study. Blood samples obtained at necropsy were analyzed for serum AP, AST and ALT activities, protein, albumin, bilirubin, urea, creatinine, inorganic phosphorous, calcium, sodium and potassium. The organ weights of the kidneys, adrenals and liver were recorded at necropsy and kidney tissue was prepared for histopathological evaluation.

Growth retardation and decreased food and water consumption were observed in male and female rats exposed to 100 or 400 ppm. Mean body weights measured on day 28 were

reduced by 34% in both male and female rats given 400 ppm HCBP in the diet. At 100 ppm, 28-day body weights were decreased by 10% in male rats and 15% in female rats. Increased volume and decreased density of the urine were observed in male rats receiving 100 ppm only. An increase in urinary epithelial cells was seen at 100 and 400 ppm HCBP in male and female rats and urinary ketones were increased at 400 ppm HCBP in both males and females. Clinical chemistry findings demonstrated increased AST activity (400 ppm males and females, 46% and 22% increased respectively), decreased total protein and albumin (400 ppm males only, 5% decrease), decreased urea (all female treatment groups, maximal decrease of 34%; 400 ppm males, 25% decrease), decreased creatinine (400 ppm females, 12% decrease), increased total bilirubin (6.7-fold and 2.4-fold increase in 400 ppm males and females, respectively) and decreased calcium (400 ppm males, 8% decrease). HCBP treatment resulted in a 13% decrease in absolute kidney weight in high-dose (400 ppm) male rats. Absolute kidney weights were similar to controls for all other treatment groups. An increase in relative kidney weight (organ:body weight ratio) was seen in male and female rats given 100 or 400 ppm HCBP (12 and 31% increase for male rats; 21 and 40% increase for female rats). The absolute organ weight of the adrenals was decreased in female rats given 100 or 400 ppm, while the relative adrenal weight was increased in high-dose male rats. Absolute liver weight was decreased in male rats at 400 ppm and in female rats at 100 and 400 ppm. Relative liver weight was increased in male rats given 100 ppm HCBP only. Relative organ weight increases (kidney, adrenals, and liver) are likely due to the observed decreases in body weight in male and female rats exposed to 100 ppm or 400 ppm HCBP. Kidney histopathology evaluation showed diffuse tubular cytomegaly (females at 100 ppm, males and females at 400 ppm) and focal nephrosis (males at 400 ppm). Incidence data for these lesions were not provided. Histopathological changes in the kidney were further described for a separate group of male and female rats given 100 ppm HCBP in the diet for 4 weeks. In female rats, necrosis, karyomegaly, hypercellularity and variable nuclear size were observed in inner cortex (incidence of 5/5 treated rats, 0/10 controls). NOAEL and LOAEL values of 25 and 100 ppm (2.6 and 10.2 mg/kg-day, respectively) were derived from this study, based on the kidney histopathology data in female Wistar rats.

NTP, 1991; Yang et al., 1989 — Dietary studies with HCBP were conducted in B6C3F₁ mice (NTP, 1991; Yang et al., 1989). In a 2-week study, mice (five/sex/group) received diets containing 0, 30, 100, 300, 1000 or 3000 ppm for 15 days. Animals were observed twice daily and were weighed initially and on days 7 and 15. Food consumption was measured on day 3 and every 2 days thereafter. Necropsy was performed on all animals and histopathology was evaluated in bone marrow, kidneys and liver for animals in the control, 300, 1000 and 3000 ppm groups. Organ weights were measured for the liver thymus kidneys, heart, brain, lung and testis.

All mice that were fed 1000 or 3000 ppm HCBP died before the end of the study. Growth retardation was observed in all HCBP treatment groups. Terminal body weights were 10%, 17% and 20% lower than controls for the 30, 100, and 300 ppm treatment groups, respectively. Control mice gained an average of 2.2 g over the course of the study, while mice given 30 ppm HCBP did not gain weight and mice given 100 ppm and 300 ppm experienced an average weight loss of 1.7 g and 2.1 g, respectively. The study authors indicated that it was unclear whether the observed growth retardation was treatment-related, due to the variability in the measured food consumption caused by scattering of feed by mice in the treatment groups. Daily dose estimates were calculated by the study authors based on feed consumption and body

weight measurements. Dietary concentrations of 0, 3, 30 and 300 ppm resulted in dose estimates by the study authors of 0, 3, 12 and 40 mg/kg-day in male mice and 0, 5, 16 and 49 mg/kg-day in female mice. Lower dietary intakes were reported for the 1000 and 3000 ppm dose groups (19 and 24 mg/kg-day in males; 30 and 36 mg/kg-day in females) due to the decreased food consumption occurring in these dose groups.

Clinical signs of toxicity were seen in mice given dietary concentrations ≥ 300 ppm. Lethargy, rough hair coat, hunched position and incoordination were observed. Decreased organ weights were seen in male and female mice from the 300 ppm dose group (28-49% decrease in thymus weight, 69-75% decrease in heart weight). Although the report does not indicate whether organ weight decreases were absolute or relative to bodyweight, the study authors suggested that the reduced organ weights were the result of stress and growth retardation and may be only secondarily related to HCB treatment. Kidney lesions were observed in mice from each HCB treatment group examined (300, 1000 and 3000 ppm). Severe necrosis of the cortex and outer medulla of the kidney was seen in mice from the 1000 and 3000 ppm dose groups that died prior to the end of the study. The necrosis was less severe at 300 ppm and regeneration was evident, especially in the pars recta (outer stripe of the outer medulla). Other lesions were seen in mice from the two highest dose groups, including lymphoid necrosis and atrophy in the spleen, thymus and lymph nodes, atrophy and necrosis of the red pulp of the spleen, testicular degeneration, and vacuolization and necrosis of hepatocytes. Minimal to mild depletion of the bone marrow (decrease in hematopoietic cells) was observed in mice treated with dietary concentrations of ≥ 300 ppm HCB. NOAEL and LOAEL values were not identified from the 2-week study because histopathology evaluation was not performed for rats receiving 30 and 100 ppm HCB.

In the 13-week dietary study, concentrations of 0, 1, 3, 10, 30 or 100 ppm HCB (98% pure) were made available mixed in feed to 10 mice/sex. Body weights and food consumption rates were measured weekly. As reported by the authors, the average daily doses of HCB were estimated to be 0, 0.1, 0.4, 1.5, 4.9 or 16.8 mg/kg-day in males and 0, 0.2, 0.5, 1.8, 4.5 or 19.2 mg/kg/day in females, when food consumption and body weight data were taken into account. Mice were observed twice daily and necropsy and histopathological evaluation of the kidneys was performed for all animals. Complete histopathology evaluation of the full range of organs and tissues was conducted for control and high-dose mice (100 ppm), and for those animals dying before the end of the study. Organ weights were measured at necropsy and samples were taken for a sperm count and motility evaluation, and for an analysis of vaginal morphology and cytology.

Although no clinical signs were evident in any of the animals in the study, one male mouse (1 ppm) died before the end of the study. HCB treatment caused a decrease in the mean body weight in the two highest-dose groups of male mice and in the highest dose group in female mice throughout most of the study. Terminal mean body weights were 10 and 16% lower than controls for male mice in the 30 and 100 ppm dose groups, respectively, and 15% lower than controls for female mice exposed to 100 ppm HCB. No major differences in food consumption were noted among treatment groups, suggesting that growth retardation is a toxic effect of HCB. Absolute kidney weights were reduced (up to 24%) compared with controls in the three highest-dose male groups and the highest-dose female group (23%). Relative kidney weight was also decreased in these treatment groups (up to 19%) compared with controls. A 12% reduction

in absolute heart weight was also evident in the 100 ppm males (relative organ weight not reported). The principal histopathological finding was a compound-related increase in regeneration in the renal tubular epithelium that was most evident in the outer stripe of the outer medulla and extended into the medullary rays (pars recta) (see Table 1). Basophilic staining of the tubular cell cytoplasm and occasional mitoses were seen in regenerative cells. The necrosis that was evident at 300 ppm and above in the 2-week study was not seen after 13 weeks of exposure to 100 ppm or lower concentrations. Sperm motility was reduced in all dose groups, but the magnitude of this effect was not dose-related. No significant changes were seen in sperm count, incidence of abnormal sperm, estrous cyclicity or average length of the estrous cycle. NOAEL and LOAEL values of 0.2 and 0.5 mg/kg-day (1 and 3 ppm) were derived from this study, based on kidney lesions (renal tubule regeneration) in female rats exposed to HCBd in the diet for 13 weeks.

Table 1. The Incidence of Renal Tubule Regeneration in B6C3F₁ Mice Exposed to HCBd in the Diet for 13 Weeks (NTP, 1991; Yang et al., 1989)

	Incidence					
	0 ppm	1 ppm	3 ppm	10 ppm	30 ppm	100 ppm
Males	0/10	0/10	0/10	0/9	10/10 ^a	10/10 ^a
Females	0/10	1/10	9/10 ^a	10/10 ^a	10/10 ^a	10/10 ^a

^ap<0.05, Fisher's Exact test performed for this analysis

Field et al. (1990) — Field et al. (1990) fed pregnant female CD rats (8-9/group) diets containing HCBd (98% purity) at concentrations of 0, 100, 200, 400, 750, 1100 or 1500 ppm on gestation day (GD) 17 through postnatal day (PND) 10. Animals were observed twice daily for clinical signs and were weighed on GD 0, 6, 11, 16 and 17 through PND 10. The reproductive and developmental parameters evaluated included litter size, sex ratio, pup body weights and percentage survival. On PND 4, litters were culled to 10 with an equivalent sex ratio, if possible. Pups were counted and weighed on PND 4, 7 and 10. On PND 10, one pup of either sex from each litter was randomly selected for urine and blood collection. Urine and blood samples were tested for glucose, urea, creatinine or total protein and osmolality was measured in the urine collected immediately following removal from the dam. One additional rat of each sex from each of the five litters was selected on PND 10 to undergo a “hydropenic test” as an indicator of renal competence. In this test, urine samples collected 4 and 6 hours following isolation from the dams were tested for osmolality. Pups were euthanized on PND 10 and liver and kidneys were weighed and prepared for histopathology. In the dams, samples of milk were collected from three dams/group, and liver and kidney tissues were weighed and processed for histopathological examination.

All dams receiving chow containing 1500 ppm HCBd became moribund and had to be terminated prematurely. Similarly, all animals (and their pups) exposed to 1100 ppm HCBd had to be terminated between PND 1 and PND 3. Clinical signs of toxicity in the two highest dose groups included excessive urination, alopecia, nasal discharge, redness of paws, tremors, piloerection, urogenital discharge, hindlimb weakness, lethargy and rough coat. Maternal body weight was decreased in all treatment groups above 100 ppm. Dams given a dietary concentration of 100 ppm HDBD had body weights similar to controls. On PND 10, maternal

body weights were decreased by 11, 21 and 31% in the 200, 400 and 750 ppm treatment groups, respectively. These reductions were accompanied by decreased food consumption during the gestational exposure period, compared with controls (43% decrease at 200 and 400 ppm and 73% decrease at 750 ppm). The study authors calculated an estimate of the daily dose using the food consumption rates for GD 20. Dose estimates of 0, 12, 22.5, 35.3 or 52.2 mg/kg-day were associated with dietary concentrations of 0, 100, 200, 400 or 750 ppm HCB. The intake of HCB throughout the study was considered to be variable, due to the fluctuation in food consumption. The HCB content of maternal milk was shown to increase with increasing dietary concentration, when measured on PND 10. Relative kidney weight in dams was increased 25, 25, 44 and 78% above controls in rats from the 100, 200, 400 and 750 ppm HCB treatment groups, respectively. Absolute kidney weights were not reported. Histopathological findings in dams demonstrated tubular regeneration of the pars recta of the proximal tubules in all treatment groups, with severity of the lesions being dose-related. At the higher dose levels, tubules were occasionally distended, appearing either empty or full of cell debris.

Three out of nine dams receiving 750 ppm HCB delivered only dead pups, and, as a percentage, fewer pups from this group survived to PND 10 compared with controls (73% survival). In general, pups displayed dose-dependent reductions in body weight compared with controls, with those at the highest dose (750 ppm) displaying marked emaciation. Pup body weights on PND 10 were 94, 90, 59 and 51% of control pups for the 100, 200, 400 and 750 ppm HCB treatment groups, respectively (statistical analysis not reported). Clinical chemistry results for the treated pups were similar to control. Following fluid deprivation, urine osmolality was increased in all HCB-treated groups of dams and pups. Relative kidney weight in pups was increased by 8, 6, 12 and 21% above the control value for the 100, 200, 400 and 750 ppm HCB treatment groups, respectively (statistical analysis was not reported). Absolute kidney weight was not reported. Histopathology examination showed kidney lesions in pups from the high-dose group only. The primary morphological changes were reduced kidney size and retention of the subscapular metanephric blastemal zone, which was considered by the study authors to reflect a delay in the postnatal development of the kidneys and apparent dehydration. The daily intake of HCB in pups on PND 10 was calculated to range from 3 to 7% of the dose received by dams in the same dose group. The lowest dose tested (12 mg/kg-day, 100 ppm) is a LOAEL for maternal effects on the kidney (increased relative kidney weight, tubule regeneration). A NOAEL was not identified for maternal effects in this study. Effects in the offspring occurred at higher doses, with NOAEL and LOAEL values of 22.5 and 35.3 mg/kg-day (200 and 400 ppm), based on reduced pup body weight and increased relative kidney weight.

Stott et al., 1981 — Male Sprague-Dawley rats (4-6/group) were given 0, 0.2 or 20 mg/kg-day HCB by oral gavage in corn oil for 21 consecutive days. An osmotic pump loaded with ³H-thymidine was implanted 7 days prior to the end of the experiment and the rate of in vivo DNA synthesis was measured. Body weight gain was determined (frequency of measurement not indicated) and kidney weight was recorded at necropsy. Tissue samples were obtained from the central portion of the animal's left kidney and evaluated for histopathology. Rats were also given a single dose of ³H-HCB (20 mg/kg-day only) and were sacrificed 4 hours later for determination of in vivo renal DNA repair and DNA alkylation. In vitro studies conducted using HCB included reverse mutation in *Salmonella typhimurium* and unscheduled DNA synthesis in primary rat hepatocytes.

In rats given 20 mg/kg-day for 3 weeks, body weight was decreased by 44%, kidney to body weight ratio was increased 1.3-fold, and a 1.8-fold increase was observed in the rate of renal DNA synthesis in vivo (not statistically significant due to high variability between animals). Histopathological lesions were also observed in rats from this group, occurring in the tubular epithelial cells of the inner and middle cortex. Lesions were characterized as degenerative and regenerative changes and included loss of cytoplasm, nuclear pyknosis, increased basophilia, mitotic activity and increased cellular debris located within the tubular lumen. No changes were observed in rats given 0.2 mg/kg-day (NOAEL value). The LOAEL for this study was 20 mg/kg-day.

Renal DNA repair was increased 1.27-fold and 1.54-fold (two trials) in rats given in a single oral dose of 20 mg/kg-day HCBd, as compared to controls. DNA alkylation was also observed in these rats. HCBd did not cause mutagenicity in *Salmonella* or unscheduled DNA synthesis in isolated rat hepatocytes.

Harleman and Seinen (1979) — Harleman and Seinen (1979) conducted a 2-week dietary study, a dietary reproduction study and a 13-week oral gavage study to evaluate the potential toxicity of HCBd in Wistar rats. In the 2-week dietary study, rats (24/sex/group) were exposed to 0, 50, 150 or 450 ppm HCBd in the diet for 14 days. Using reference values for body weight and food consumption in weanling Wistar rats (male body weight 0.053 kg, female body weight 0.052 kg, food consumption of 0.008 kg/day for both males and females) (U.S. EPA, 1988), daily dose estimates were calculated to be 0, 8, 23 or 68 mg/kg-day for male rats and 0, 8, 23 or 69 mg/kg-day for female rats. Body weights were measured at the beginning and end of the study. Liver and kidney weights were recorded at necropsy and histopathology of these organs was evaluated. Body weight was decreased in all HCBd treatment groups of female rats (10-33% decrease) and in the two highest dose groups of male rats (21 and 31% decrease at 150 or 450 ppm, respectively). Relative kidney weights were significantly increased in the two highest dose groups of male and female rats exposed to HCBd (21-28% increase in males, 7-22% increase in females). Absolute kidney weights were not reported. Histopathology findings demonstrated dose-related kidney lesions occurring in all exposed animals. These lesions were described as degeneration of the tubule epithelial cells, especially in the straight limbs of the proximal tubules located in the outer zone of the medulla. The LOAEL for the 2-week dietary study was 8 mg/kg-day (50 ppm). A NOAEL value was not identified.

In the reproduction study, female Wistar rats (six females/group) were exposed to 0, 150 or 1500 ppm for 18 weeks (4 weeks prior to mating and a 3-week mating period with untreated males). The number of pups/litter and the pup body weights were measured at parturition and the pups were culled to eight per litter. Offspring body weights were measured at PND 10 and 20 and necropsy of the adult females was conducted at 18 weeks. Organ weights were recorded for the heart, liver, kidneys, spleen, brain, adrenals, thymus and thyroid. Histopathology was performed for these organs and the lungs, pancreas, digestive tract (six segments), urinary bladder, axillary and mesenteric lymph nodes, trachea, spinal cord, and femoral nerve. No conception occurred for female rats exposed to a dietary concentration of 1500 ppm HCBd. Progressive weight loss was seen in rats from this group and an unsteady gait, hind limb weakness and ataxia occurred by 6 weeks of exposure to HCBd. Necropsies were performed during week 10, due to the moribund condition of the animals. Gross examination revealed large

pale kidneys and extensive tubule degeneration was seen by histopathology. Proliferation of bile duct epithelial cells in the liver and fragmentation and demyelination of single fibers of the femoral nerve were also seen.

Five out of six rats in the 150 ppm dose group were fertile with a mean litter size similar to control rats. The birth weight of pups from this treatment group was lower than controls (16% decrease) and a decreased pup body weight was also observed at weaning (19% decrease). The resorption quotient was low for both control and treated rats and no gross malformations of offspring were observed. At 18 weeks, the body weight of treated dams was 15% lower than control dams (231 g for controls, 196 g for 150 ppm group). The average daily dose for the 150 ppm group was estimated to be 11 mg/kg-day, assuming a body weight of 0.196 g and a food consumption rate of 0.015 kg/day (calculated using equations in U.S. EPA, 1988). Relative kidney weight was increased by 22%, as compared with controls. Absolute kidney weights were not reported. HCBBD treatment caused histopathological changes in the kidney of dams, including hypercellularity of tubule epithelial cells and hydropic necrosis of cells in the straight limbs of the proximal tubules. Treatment related effects were not observed in other organs or tissues. A LOAEL of 11 mg/kg-day (150 ppm) was derived for the reproduction study, based on maternal effects (decreased weight gain, increased kidney weights and altered kidney histopathology) and decreased fetal body weight.

In the 13-week subchronic study, 60 rats/sex/group received 0, 0.4, 1.0, 2.5, 6.3 or 15.6 mg/kg-day HCBBD in arachid oil for 13 weeks. Blood samples were collected at 8 weeks (six rats/sex/group) and analyzed for hemoglobin, hematocrit, RBC count, and total and differential leukocytes. Blood samples were also obtained at study termination and tested for total protein, albumin, globulin, BUN, AST, AP and γ -glutamyl transferase activities. At 10 weeks, urine samples were collected from six rats/sex/group during the 2nd-6th and 7th-21st hour deprivation period of food and water. Urine samples were analyzed for glucose, protein, hemoglobin, ketones and pH. Urine volume and osmolarity were used as measures of the concentrating ability of the kidney. At termination, organs were weighed (heart, liver, kidney, spleen, brain, adrenals, thymus, thyroid and gonads) and a gross pathological examination was carried out on all animals. Key organs and tissues from the control and high-dose groups were processed for histopathological examination (heart, liver, kidneys, spleen, brain, adrenals, thymus, thyroid, lungs, pancreas, six sections of digestive tract, urinary bladder, axillary and mesenteric lymph nodes, trachea, spinal cord, femoral nerve, prostate, skeletal muscle, aorta, Harder's gland, skin, sternum and bone marrow). The HCBBD content of kidney, liver and fat samples from high-dose female rats was measured by gas chromatography (GC) analysis.

Body weight gain and food consumption were significantly reduced at the two highest doses in male and female rats, as compared to controls (13-30% decrease in body weight at 6.3 mg/kg-day; >40% reduction in body weight at 15.6 mg/kg-day). No clinical chemistry changes were observed at any dose levels. Following a 21-hour deprivation period, a dose-related decrease in urine osmolarity was observed in female rats that were given HCBBD at doses greater than 2.5 mg/kg-day. An increase in urine volume was also observed in the two highest dose groups of female rats (6.3 and 15.6 mg/kg-day), indicating an impairment in the urine concentrating ability of the kidney. No change in urine volume was observed in treated male rats, and urine osmolarity was increased only in high-dose males (15.6 mg/kg-day). Relative

kidney weight was significantly increased in all dose groups of male rats (7-31% increase) and in the two highest dose groups of female rats (19 and 32% increase for 6.3 and 15.6 mg/kg-day respectively). The relative liver weight was increased 8-24% in male rats at doses greater than 1 mg/kg-day, but was only increased in the high-dose group in female rats by 11%. In male rats, the relative weights of the brain and the spleen were increased in the 15.6 mg/kg-day dose group by 45 and 18%, respectively. Increases in the relative weight of the brain and spleen were seen at the two highest doses in female rats (21-29% increase for brain, 14-21% increase for spleen). The relative weight of the gonads was increased in male rats (12 and 40% increase at 6.3 and 15.6 mg/kg-day respectively). Absolute organ weights were not reported in the study.

Though no changes in organ appearance were seen on gross pathological examination, marked histopathological lesions were evident in the kidney, most notably in the proximal tubule, where increases in hypercellularity, necrosis and the incidence of hyperchromatic nuclei were evident. Epithelial cells in treated rats were described as small, basophilic and finely vacuolated, with large hyperchromatic nuclei. At the highest dose in female rats, changes were seen in both the straight and convoluted portions of the tubules with focal necrosis and a thin or absent epithelial brush border. The changes were similar, but less severe, in females given 6.3 mg/kg-day and were limited to the straight portion of the proximal tubule. The brush border was generally unchanged and few necrotic cells were present in the tubule lumen. Only minor effects were seen in female rats given 2.5 mg/kg-day, although tubule epithelial cells were observed to contain enlarged hyperchromatic nuclei. Kidney effects were less pronounced in male rats, as compared to females. Kidney lesions in male rats given 15.6 mg/kg-day were similar in severity to those seen in female rats given 6.3 mg/kg-day. Liver effects were seen in male rats only at doses greater than 6.3 mg/kg-day and consisted of a basophilic granulation of hepatocytes. The GC analysis of kidney, liver and adipose tissue from high-dose female rats revealed no HCBd accumulation in the liver or kidney and only slight accumulation in the fat. NOAEL and LOAEL values of 1.0 and 2.5 mg/kg-day, respectively, were derived from this study based on kidney toxicity in female rats.

Kociba et al. (1977) — Kociba et al. (1977) administered HCBd (99% purity) mixed in feed to 39-40 Sprague-Dawley rats/sex/group for 2 years. Ninety rats of each sex were used as controls. Rats were observed frequently (not quantified) for clinical signs of toxicity. Feed consumption and body weights were monitored in 15 rats/sex/group weekly for the first 3 months of the study, and then for 1 week out of each month until study termination. The average doses for either sex were calculated by the authors to be 0, 0.2, 2 or 20 mg/kg-day. Subsets of animals (5-6/sex/group) were sampled for blood and urine after approximately 12, 22 (males only) or 24 (females only) months. The hematological parameters evaluated included packed cell volume (PCV), RBC count, hemoglobin concentration, total WBC count and differential WBC count. The urinary parameters evaluated were specific gravity, pH and the presence or absence of glucose, protein, ketones, bilirubin and occult blood. Urinary creatinine, coproporphyrin and uroporphyrin were also determined from a urine sample collected over 24 hours. After 1 year, blood samples were collected from an additional subset of animals (five/sex from the high-dose and control groups only) for clinical chemistry determinations. Serum samples were also collected from all rats necropsied at the end of the study. Serum chemistry parameters included blood urea nitrogen (BUN), and AP and ALT activity. All rats (moribund and terminal sacrifice) were necropsied and pieces of all major organs and lesions were excised

and preserved. For the rats that were killed during the course of the study, histopathological evaluation was performed for the liver, kidney, stomach and all tumors or gross lesions. For those sacrificed at term, a fully comprehensive list of organs and tissues was examined microscopically for 10 females at each dose level, 10 males from the 0 and 2 mg/kg-day dose levels and 3 males at the 20 mg/kg-day dose level that survived to term. Histopathology evaluation for the remaining rats that were killed at study termination (including all male rats from the 0.2 mg/kg-day group) was limited to the kidneys, liver, stomach and any gross lesions observed during necropsy.

There was a reduction in body weight gain at the high dose level in both male and female Sprague-Dawley rats that appeared not to be associated with the sporadic changes in food consumption. This decrease in body weight was evident by 27 days in female rats and 69 days in male rats and body weight remained low throughout most of the study. A significant increase in mortality (approximately 20%, estimated from graph) occurred during the last 2 months of the study in male rats that ingested 20 mg/kg-day HCBd. Survival was not reduced in any other HCBd treatment group.

Compound-related changes in hematological parameters were limited to a 20% decrease in RBCs after 22 months in high dose male rats. Routine urinalysis parameters were not affected by HCBd treatment; however, the excretion of coproporphyrins was increased in high-dose male rats at 1 year, mid-dose female rats at 14 months and high-dose female rats at 2 years. No dose response or temporal trend was apparent from these data (see Table 2). A 57% decrease in the excretion of uroporphyrin was also seen in high-dose female rats after 2 years. Clinical chemistry parameters were generally not altered by HCBd treatment for 12 months or 2 years, with the exception of a decrease in ALT activity in high-dose (20 mg/kg-day) males at 12 months and low- and high-dose females (0.2 and 20 mg/kg-day, respectively) at 2 years. This finding in female rats was considered to result from an abnormally increased ALT activity in female control rats and was not considered to be treatment-related.

Table 2. Average Amounts of Coproporphyrins in Urine of Sprague-Dawley Rats in Response to HCBd in Feed ($\mu\text{g}/24$ hours) (Kociba et al., 1977)						
HCBd in feed (mg/kg-day)	1 Year		14 Months		2 Years	
	Male	Female	Male	Female	Male	Female
0	10.2 \pm 8.5	5.0 \pm 1.3	13.1 \pm 3.0	5.6 \pm 2.4	6.8 \pm 1.8	4.5 \pm 2.4
0.2	14.2 \pm 2.6	4.7 \pm 2.1	13.0 \pm 3.8	6.2 \pm 3.3	7.1 \pm 2.3	5.4 \pm 0.8
2.0	18.8 \pm 2.4	8.9 \pm 5.2	18.3 \pm 4.0	10.6 ^a \pm 2.4	10.7 \pm 2.4	5.8 \pm 1.3
20.0	23.1 ^a \pm 11.8	9.4 \pm 3.5	17.7 \pm 12.5	8.4 \pm 2.5	14.0 \pm 9.5	12.3 ^a \pm 2.9

Values are means \pm SD (n=5).

^ap<0.05 as determined by ANOVA and Dunnett's test.

An increase in the absolute and relative weight of the kidneys was observed in male rats given 20 mg/kg-day HCBd for 22 months. An increase was also observed in relative, but not

absolute, testes weight; however, this may have been due to the observed decrease in body weight. A decrease in the absolute weight of the heart and liver and an increase in the relative weight of the brain and kidney were seen in high-dose female rats. Organ weights in the low- and mid-dose groups of male and female rats were similar to control. Histopathological examination revealed treatment-related kidney lesions in male and female rats consisting of tubular epithelial hyperplasia and proliferation, observed in the mid- and high-dose groups (2 and 20 mg/kg-day) (incidence data not provided) and tubular adenomas and adenocarcinomas in high-dose rats only. The histopathology findings in low-dose rats were similar to controls. The incidence of combined adenomas and carcinomas in kidney was 1/90, 0/40, 0/40 and 9/39 in males and 0/90, 0/40, 0/40 and 6/40 in females, for the control, low-, mid- and high-dose groups, respectively. Metastasis to the lung was noted in two cases. NOAEL and LOAEL values of 0.2 and 2 mg/kg-day, respectively, were derived from this study based on kidney lesions (tubular epithelial hyperplasia and proliferation) observed in male and female rats that ingested HCB in the diet for 2 years.

Schwetz et al., 1977 — The same research group carried out a combined subchronic and reproductive study (Schwetz et al., 1977) in parallel to that of Kociba et al. (1977). Male and female Sprague-Dawley rats (10-12 males/treatment group, 17 male controls, 20-24 females/treatment group, 34 female controls) received 0, 0.2, 2.0 or 20 mg/kg-day HCB (99% purity) in feed for 90 days prior to mating, throughout a 15-day mating period, and then through gestation and lactation. Blood and urine samples were collected from control and high-dose rats prior to the end of the study. At study termination, blood samples were taken from the dams prior to necropsy to measure levels of BUN, serum creatinine and ALT activity. The brain, heart, liver, kidneys and testes (males) were obtained from 10 adult rats/sex/group and organ weights were determined. For the controls and high-dose groups, many organs and tissues were excised, weighed and processed for histopathological examination (brain, heart, liver, kidneys, testes, eye, pituitary, thyroid gland, parathyroid gland, trachea, esophagus, lungs, aorta, stomach, pancreas, small intestine, colon, mesenteric lymph nodes, muscle, sciatic nerve, spinal cord, sternum, sternal bone marrow and adrenal gland). Histopathology was also carried out on kidney tissue excised from five animals from each exposure group. Standard indices of reproductive performance were evaluated, and weanling skeletons were examined after alcohol fixation and appropriate extraction and staining. Bone marrow was taken from four adults and four weanlings/sex/group for cytological examination.

No clinical signs were evident in any of the adults receiving HCB. A decrease in food consumption was noted in high-dose male and female rats. Female rats from this group weighed significantly less than controls throughout the study (22% decrease in final body weight), while male body weights were sometimes, but not always, lower than controls (10% decrease in final body weight). There were no differences among the groups in any reproductive or survival parameters for the dams and neonates (percent pregnant, litter size, gestation survival index, sex ratio, duration of gestation); however, the mean weight of high-dose neonates was significantly reduced (13% decrease) in the 20 mg/kg-day group compared with controls at weaning (21 days of age). No gross abnormalities were observed in neonates at necropsy. Skeletal alterations were not evident in neonates at any dose level.

Among clinical chemistry parameters, BUN was decreased in male rats by 17 and 13% in the 0.2 and 2 mg/kg-day dose groups, respectively, but was similar to controls in the 20 mg/kg-day dose groups. Serum levels of creatinine and ALT did not differ from those of controls. Hematology and urinalysis results were not presented or discussed. A significant increase in the relative weights of the liver (male only, 26% increase) and kidney (27 and 19% increase in males and females, respectively) was observed at the highest dose of HCB. Absolute liver and kidney weights were not different from control values in any treatment group. An increase in relative brain weight (31% increase) and a decrease in relative heart weight (24% decrease) were also observed in female rats from the 20 mg/kg-day dose group. No changes in absolute brain or heart weight were observed. Kidneys from male rats ingesting 2 or 20 mg/kg-day HCB were described as roughened with a mottled cortex. No gross abnormalities were noted for female rat kidneys. Histopathological examination revealed renal tubular dilation and hypertrophy with foci of tubular epithelial degeneration and regeneration. The incidence of these kidney lesions in rats ingesting 0, 0.2, 2 or 20 mg/kg-day was 1/5, 0/5, 0/5 and 3/5 for male rats and 0/5, 0/5, 1/5 and 5/5 for female rats. No histopathological kidney lesions were evident in weanling rats. Although these findings are limited by the small number of animals examined for histopathological evaluation, NOAEL and LOAEL values of 0.2 and 2 mg/kg-day, respectively, were derived from this study, based on gross and microscopic kidney lesions in adult rats exposed to HCB for 90 days prior to mating, 15 days during mating, and throughout gestation and lactation.

Kociba et al., 1971 - HCB (99% pure) was administered to female Sprague-Dawley rats (4/group) in the diet for 30 days at doses of 0, 1, 3, 10, 30, 65, 100 mg/kg-day (Kociba et al., 1971). Rats were observed daily and feed consumption and body weight gain were recorded weekly throughout the study. Blood samples obtained during necropsy were analyzed for hematology parameters and ALT activity. Organ weights of heart, liver, kidney, spleen and brain were recorded and several organs and tissues were prepared for histopathology evaluation (heart, liver, kidney, spleen, brain, pituitary, thyroid, parathyroid, lung, adrenal, mesenteric lymph node, ovary uterus, stomach and intestinal tract). Clinical signs of toxicity were not observed during the study. The food consumption rate was significantly decreased in rats receiving HCB doses greater than 30 mg/kg-day. The mean body weight values measured at 28 days were 4, 10, 22 and 28% lower than controls for the 10, 30, 65 and 100 mg/kg-day groups, respectively. A decrease in absolute organ weight was seen in the liver, heart and spleen of rats in the 65 and 100 mg/kg-day dose groups. Absolute kidney weight was increased at 3 mg/kg-day, but was similar to controls for all other treatment groups. An increase in relative organ weight (organ:body weight ratio) was seen in the brain, liver and kidneys of rats given 30, 65 or 100 mg/kg-day HCB. Hematology results were considered to be within a normal range. No change in AST activity was observed. Gross findings revealed a depletion of abdominal fat deposits in rats given 65 or 100 mg/kg-day HCB. Histopathology results showed hepatocellular swelling in rats given 100 mg/kg-day HCB only (4/4 rats). Kidney lesions included tubular epithelial cell degeneration, single cell necrosis and regeneration in all rats (4/4) from the 30, 65 and 100 mg/kg-day dose groups. Liver and kidney lesions were not observed in control rats or in rats given 1 or 3 mg/kg-day (0/4 per group). NOAEL and LOAEL values of 10 and 30 mg/kg-day, respectively, were derived from this study based on renal lesions observed in female Sprague-Dawley rats.

Inhalation Exposure. Few studies were located regarding the toxicity of HCBd by inhalation exposure in animals. Saillenfait et al. (1989) conducted a developmental toxicity study in which groups of 24-25 pregnant Sprague-Dawley rats inhaled 0, 2, 5, 10 or 15 ppm of HCBd for 6 hours/day on days 6-20 of gestation. The pregnant rats were weighed prior to exposure on days 0 and 6 of gestation, and again prior to sacrifice on day 21 of gestation. After sacrifice, the uterus was removed from each female and examined for numbers of implantation and resorption sites and live and dead fetuses. Live fetuses were sexed, weighed and examined for external malformations and cleft palate. Half of the viable fetuses from each litter were examined for soft tissue alterations and the other half were examined for skeletal alterations. No deaths or changes in general behavior were noted for exposed females. There was a concentration-related reduction in maternal weight gain in animals exposed to HCBd. Weight gain was reduced by 8% at 2 ppm, 15% at 5 ppm, 12% at 10 ppm and 39% at 15 ppm. The difference from controls was statistically significant in the 5 and 15 ppm groups.

Mean numbers of implantations, total fetal loss, resorptions and live fetuses were similar in treated and control animals (Saillenfait et al., 1989). Incidence of pregnancy and fetal sex ratio were also unchanged by HCBd exposure. However, body weight of both male and female fetuses was significantly reduced in the 15 ppm group (decreased by 9.5 and 12.5% in males and females, respectively). External examination of fetuses did not find any abnormalities, and no major anomalies were found after skeletal and soft tissue examination. The only minor anomalies were a non-significant incidence of hydronephrosis at 15 ppm and a non-significant increase in the incidence of extra 14th ribs at 10 ppm. Although there was a significant reduction in fetal weight at the greatest exposure to HCBd, there was no significant retardation of development (e.g., delayed ossification) and the change was accompanied by a reduction in maternal weight gain. This study identified a NOAEL of 2 ppm and a LOAEL of 5 ppm for maternal toxicity (decreased weight gain), and a NOAEL of 10 ppm and a LOAEL of 15 ppm for developmental effects (decreased fetal body weight).

Dow Chemical Company conducted a subchronic inhalation study of HCBd that was described by Torkelson and Rowe (1982), as follows: "small groups of rats, rabbits and guinea pigs exposed 7 hours/day, 100 times to 3 ppm in a 143 day period were adversely affected, but those exposed 129 times in 184 days to 1 ppm were not. The livers and kidneys of the animals exposed to 3 ppm were the organs most affected." No further details of this study were located. Representatives from Dow have stated that a more detailed report of this study, which was conducted in the 1950s, is no longer available (Dow, 1992).

Respiratory irritation and renal effects were observed in short-term, repeated inhalation studies of HCBd in rats. Alderley Park SPF rats (four rats of each sex for each treatment) were exposed to concentrations of HCBd ranging from 5 to 250 ppm for durations up to 3 weeks (Gage, 1970). A day after exposure was terminated, animals were sacrificed and necropsied. The following organs were routinely examined microscopically for damage: lungs, liver, kidneys, spleen and adrenals. Blood and urine tests were normal for all treatments. At 250 ppm of HCBd (2 x 4 hours), irritation and breathing difficulties were observed (more pronounced in females). Necropsy showed degeneration of the middle renal proximal tubules and the adrenal cortex. At 100 ppm (6 hours/day, 5 days/week, 12 days) irritation and respiratory difficulties were observed; animals had poor condition and weight loss. Females had slight anemia and two

died. Necropsy showed enlarged adrenal glands and pale, enlarged kidneys with degeneration of the renal cortical tubules and epithelial regeneration. At 25 ppm (6 hours/day, 5 days/week, 3 weeks) respiratory difficulties and poor condition were observed. Females had diminished weight gain. At necropsy, kidneys were pale and enlarged with damage to the renal proximal tubules. Ten ppm (6 hours/day, 5 days/week, 3 weeks) produced diminished weight gain in females, but no organ damage. Exposure to 5 ppm (6 hours/day, 5 days/week, 3 weeks) caused no symptoms of toxicity or organ damage.

DeCaurriz et al. (1988) assessed respiratory irritation and kidney damage after acute exposure of male Swiss OF₁ mice to HCBd. The respiratory rates of mice (six mice per treatment group) were measured during a 15-minute oronasal exposure to HCBd (83, 143, 155, 210 or 246 ppm) using individual body plethysmographs. The decreases in respiratory rate recorded for each concentration were used to calculate the concentration associated with a 50% decrease in respiratory rate (RD₅₀). The RD₅₀ for HCBd was 211 ppm. In a previous study, DeCaurriz et al. (1981) calculated the RD₅₀ for a number of different chemicals. The RD₅₀ for hexachlorobutadiene places it among the more potent irritants. For instance, the RD₅₀ for phenol was 166 ppm and that of formaldehyde was 5.3 ppm, while the RD₅₀ of toluene was 3373 ppm and that of xylene was 1467 ppm (DeCaurriz et al., 1981).

Mice were also exposed to various concentrations of HCBd (2.75, 5, 10 and 25 ppm) or clean filtered air for 4 hours (DeCaurriz et al., 1988). After a recovery period of 24 hours, the animals were sacrificed and their kidneys were examined microscopically for damaged tubules and alkaline phosphatase staining. There was a significant, concentration-related increase in nephrotoxicity associated with HCBd exposure. The percentage of altered renal tubular cross-sections increased from 4% in the 2.75 ppm group to 92% in the 25 ppm group (versus 0.2-1.5% in the corresponding control groups). The researchers estimated an EC₅₀ of 7.2 ppm for kidney histopathology produced by HCBd. On the basis of these findings, the researchers concluded that the kidney is a more sensitive target for HCBd than the respiratory tract following acute inhalation exposure in the mouse. However, DeCaurriz et al. (1988) did not perform a histopathological examination of the upper respiratory tract. A more recent evaluation of the applicability of sensory irritation tests (Bos et al., 1992) has described a number of compounds for which histopathological damage was observed at exposure levels more than 10 times lower than the RD₅₀.

Although respiratory tract effects of HCBd have not been studied following chronic exposure, it is reasonable to suspect that such effects may be important for this chemical. Nasal toxicity was a prominent finding in chronic bioassays of the structurally related chemical 2-chloro-1,3-butadiene (chloroprene) in rats and mice (NTP, 1998).

Other Studies

The mode of action for the kidney toxicity of HCBd has been described (reviewed in Green et al., 2003; NTP, 1991; Dekant et al., 1990). HCBd is metabolized in the liver to a glutathione conjugate, which is further transformed by γ -glutamyl transpeptidase and dipeptidase enzymes to yield a cysteine conjugate. The cysteine conjugate may be cleaved by the renal β -lyase enzymes to give toxic thiol intermediates that cause localized kidney damage. The

cysteine conjugate may also be metabolized by N-acetyl transferase to form a N-acetyl cysteine conjugate that can be excreted in the urine or converted back to the cysteine conjugate by acylase enzymes. The nephrotoxicity of HCBd is linked to the relative activity of the renal β -lyase enzyme and the amount of cysteine conjugate available to be metabolized to toxic intermediates.

Green et al. (2003) compared the key metabolic steps for HCBd in both rat and human tissues. Human liver and kidney samples were obtained as excess tissue during organ transplantation. In vitro studies were used to evaluate glutathione conjugation of HCBd (in liver microsomes), the metabolism of cysteine conjugates by renal β -lyase (in kidney cytosol and mitochondria) or N-acetyl transferase (kidney microsomes) and the metabolism of the N-acetyl cysteine conjugate by acylase enzymes (kidney cytosol). The metabolic rates (V_{max}) for each of these steps were lower in humans as compared to rats (5-fold lower for glutathione conjugation, 3-fold lower for β -lyase activity and 3.5-fold lower for N-acetyl transferase activity). Acylase enzyme activity was not detected in human kidney cytosol. The metabolic rate constants obtained for rats and humans were used in a physiologically-based pharmacokinetic (PBPK) model to quantify metabolism through the β -lyase pathway to form reactive intermediates. The uptake and distribution of HCBd was estimated in the PBPK model using measured partition coefficients and standard values for physiological parameters. The PBPK model predicted that metabolism by the β -lyase pathway is approximately 20-fold lower in humans than in rats exposed to the same inhalation concentration. The predicted decrease in the formation of β -lyase metabolites was related to decreased uptake of HCBd, lower glutathione transferase and β -lyase activities and the absence of acylase activity in human kidney. Comparable model predictions for the oral exposure route were not provided in this study.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfDs FOR HCBd

No data were located regarding the oral toxicity of HCBd in humans. Kidney toxicity was the primary effect of oral HCBd exposure in laboratory animals. Short-term studies (2-4 weeks in duration) demonstrated necrosis and degeneration of kidney tubules at high doses (>10 mg/kg-day) (Jonker et al., 1993; NTP, 1991; Yang et al., 1989; Harleman and Seinan, 1979; Kociba et al., 1971). Necrosis was less severe at lower doses and regeneration of kidney tubules was observed. In subchronic and chronic studies, the primary histopathological change observed was renal tubule regeneration, also characterized as hyperplasia and proliferation, and necrosis was not generally seen (NTP, 1991; Yang et al., 1989; Harleman and Seinen, 1979; Kociba et al., 1977; Schwetz et al., 1977). Kidney effects were most prevalent in the straight limbs of the proximal tubule in the outer zone of the medulla; however, the convoluted portions of the proximal tubule were also involved at high doses (Harleman and Seinen, 1979). The regenerative hyperplasia observed in tubule epithelial cells (NTP, 1991; Yang et al., 1989; Harleman and Seinen, 1979; Kociba et al., 1971) may be a response to HCBd-induced cell injury and/or may be a precursor to the renal neoplasms that were observed following chronic exposure (Kociba et al., 1977). The chronic and subchronic oral toxicity studies for HCBd are summarized in Table 3.

Table 3. Chronic and Subchronic Oral Toxicity Studies for HCB

Species	Dose/Duration	NOAEL (mg/kg-day)	LOAEL (mg/kg-day)	Effect	Reference
B6C3F ₁ mice	13-week dietary study; 0, 0.1, 0.4, 1.5, 4.9 or 16.8 mg/kg-day in males; 0, 0.2, 0.5, 1.8, 4.5 or 19.2 mg/kg/day in females	0.2	0.5	Renal tubule regeneration	NTP (1991); Yang et al. (1989)
Wistar rats	13-week oral gavage study; 0, 0.4, 1.0, 2.5, 6.3 or 15.6 mg/kg-day HCB in arachid oil	1.0	2.5	Tubule epithelial cell with enlarged hyperchromatic nuclei; focal necrosis at higher doses	Harleman and Seinen (1979)
Sprague-Dawley rats	2-year dietary study; 0, 0.2, 2 or 20 mg/kg-day	0.2	2	Renal tubule hyperplasia and proliferation	Kociba et al. (1977)
Sprague-Dawley rats	Dietary study, 13 weeks pre-mating, 15 day mating period and throughout gestation and lactation; 0, 0.2, 2 or 20 mg/kg-day	0.2	2	Renal tubule degeneration and regeneration	Schwetz et al. (1977)

The quantal Benchmark Dose (BMD) models in the BMD software package (U.S. EPA, 2007; Version 1.3.2) were fit to the female mouse renal tubule regeneration data in Table 1 (NTP, 1991; Yang et al., 1989). The gamma, log-probit, Weibull and log-logistic give virtually the same fit with indistinguishable Akaike Information Criterion (AIC). The BMD and BMDL₁₀ values were all the same at 0.2 and 0.1 mg/kg-day, respectively. The Weibull had the best fit in the region of the BMR (lowest absolute scaled residual at 0.2 mg/kg-day, although the differences among the models are minimal (see Appendix 1). The 1st-order multistage fit adequately ($p = 0.37$) but had a much higher AIC than the aforementioned model fits. Therefore, the point of departure (POD) is set equal to the common BMDL₁₀ of 0.1 mg/kg-day.

The **subchronic p-RfD of 1E-3 mg/kg-day** is based on the BMDL₁₀ of 0.1 mg/kg-day for renal tubule regeneration observed in a 13-week dietary study in mice (NTP, 1991; Yang et al., 1989). Kidney toxicity was also seen in a 13-week gavage study in rats at higher doses (Harleman and Seinen, 1979).

The subchronic p-RFD is derived by dividing the BMDL₁₀ of 0.1 mg/kg-day by a composite UF of 100, as follows:

$$\begin{aligned}
 \text{Subchronic p-RfD} &= \text{NOAEL/ UF} \\
 &= 0.1 \text{ mg/kg-day} / 100 \\
 &= \mathbf{0.001 \text{ or } 1\text{E-3 mg/kg-day}}
 \end{aligned}$$

The composite UF of 100 includes factors of 3 ($10^{0.5}$) each for animal-to-human extrapolation and database deficiencies, and a factor of 10 for interindividual variability.

The interspecies UF of 3 was used to account for pharmacodynamic differences across species. The role of metabolism in HCBd-induced kidney toxicity is well established. Pharmacokinetic differences between the rat and the human were investigated by Green et al. (2003). In vitro studies were used to evaluate key steps in the metabolism of HCBd (glutathione conjugation in the liver, β -lyase, N-acetyl transferase and acylase enzyme activity in the kidney). The metabolic rate for each of these steps was lower in humans as compared to rats, suggesting that humans may be less sensitive to the kidney toxicity of HCBd (no pharmacokinetic adjustment was necessary). Although comparable in vitro metabolism data are not available for mice, the dose response data suggest that rats and mice are similarly sensitive to the kidney toxicity caused by HCBd. Given the large difference (20-fold) in predicted toxic metabolite formation in the PBPK model (Green et al., 2003), there is marginal justification for reducing UF_A to unity, despite the lack of information on toxicodynamic differences between rats (or mice) and humans. However, a somewhat limiting assumption is already made about the similarity of mice and rats for the metabolism of HCBd and there is no information on the relative *in vivo* abundance of key enzymes across species, only on specific activities. These limitations preclude further reduction of UF_A . The interspecies UF of 3 was therefore considered appropriate for both rats and mice.

The interindividual variability UF of 10 is used to account for variation in sensitivity within human populations because there is limited information on the degree to which humans of varying gender, age, health status or genetic makeup might vary in the disposition of, or response to, HCBd. A partial UF of 3 for database deficiencies is selected due to the lack of a multigeneration reproductive toxicity study. Prenatal and postnatal developmental toxicity studies are available for HCBd using the oral and inhalation exposure route (Field et al., 1990; Harleman and Seinen, 1979; Saillenfait et al., 1989). There was little assessment of the immune and nervous systems in the literature. With respect to the latter, Harleman and Seinen (1979) demonstrated neuropathy in rats at 1500 ppm (150 mg/kg-day) after 18 weeks of exposure. As this LOAEL is more than 3 orders-of-magnitude greater than the BMDL of 0.1 mg/kg-day, it is probably not much of a concern, either for the subchronic p-RfD or for longer periods of exposure relative to the chronic p-RfD.

Confidence in the critical study is medium. NTP (1991)/Yang et al. (1989) was a well-conducted, 13-week dietary study with relatively small number of animals (10/group). The critical effect (kidney lesions) was well studied. Limitations include lack of hematology and clinical chemistry and lack of histopathology on organs other than the kidney. NOAEL and LOAEL values were derived from the study based on kidney toxicity. Confidence in the database is medium. An additional subchronic oral gavage study showed similar effects at doses that were approximately 5-fold higher (Harleman and Seinen, 1979). Limitations of the database

include the lack of multigeneration reproductive toxicity data. Prenatal and postnatal developmental toxicity studies have been performed (Field et al., 1990; Harleman and Seinen, 1979; Saillenfait et al., 1989). Overall, confidence in the subchronic p-RfD is medium.

The **chronic p-RfD of 1E-3 mg/kg-day** is also based on renal tubule regeneration in the 13-week dietary study in mice (NTP, 1991; Yang et al., 1989), as the BMDL of 0.1 mg/kg-day is lower than the NOAEL of 0.2 mg/kg-day for renal effects in the 2-year rat study (Kociba et al., 1977). The incidence of kidney lesions was not reported for each dose group in the 2-year study. Therefore, benchmark dose modeling could not be used to derive a point of departure. Therefore, the chronic p-RfD is derived by dividing the subchronic BMDL₁₀ of 0.1 mg/kg-day by a composite UF of 100, as follows:

$$\begin{aligned}\text{Chronic p-RfD} &= \text{NOAEL} / \text{UF} \\ &= 0.1 \text{ mg/kg-day} / 100 \\ &= \mathbf{0.001 \text{ or } 1\text{E-3 mg/kg-day}}\end{aligned}$$

The composite UF of 100 includes factors of 3 ($10^{0.5}$) each for animal-to-human extrapolation and database deficiencies, and a factor of 10 for interindividual variability. A subchronic-to-chronic uncertainty factor is not required because the chronic 2-year rat study indicates that prolonged exposure does not result in toxicity at lower doses than for subchronic exposure. The interspecies UF of 3 was used to account for pharmacodynamic differences across species as described previously for the subchronic p-RfD. The interindividual variability UF of 10 is used to account for variation in sensitivity within human populations because there is limited information on the degree to which humans of varying gender, age, health status or genetic makeup might vary in the disposition of, or response to, HCB. A partial UF of 3 for database deficiencies is selected due to the lack of a multigeneration reproductive toxicity study. Prenatal and postnatal developmental toxicity studies are available for HCB using the oral and inhalation exposure route (Field et al., 1990; Harleman and Seinen, 1979; Saillenfait et al., 1989).

Overall confidence in the chronic p-RfD is medium for the same reasons as for the subchronic p-RfD.

FEASIBILITY OF DERIVING PROVISIONAL CHRONIC AND SUBCHRONIC RfCs FOR HCB

The available data are inadequate to support derivation of a provisional inhalation RfC for HCB. Reduced body weight gain was observed in dams following inhalation exposure in the Saillenfait et al. (1989) rat developmental toxicity study (NOAEL of 2 ppm). However, this study included limited evaluation of non-developmental endpoints, no examination of the respiratory tract and no assessment of kidney toxicity, the critical effort for oral exposure. The only other inhalation study of appropriate duration to consider for RfC derivation is the Dow Chemical study briefly described by Torkelson and Rowe (1982). However, the existing description of this study provides insufficient information to assess the study, and attempts to obtain more detailed information about the study were unsuccessful.

The database for oral toxicity of HCBd is more extensive than that for inhalation toxicity (ATSDR, 1994). The kidney appears to be the most sensitive target of HCBd by oral exposure. However, due to overt signs of respiratory irritation and uncertainty regarding the relative sensitivity of the respiratory tract as compared to the kidney with long-term inhalation exposure, an RfC is not derived.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR HEXACHLOROBUTADIENE

The carcinogenicity assessment, which includes an oral slope factor and inhalation unit risk, is on IRIS (U.S. EPA, 1991b).

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APPENDIX

BENCHMARK DOSE MODELING RESULTS (BMDS, VERSION 1.3.2): FEMALE MOUSE RENAL TUBULE REGENERATION DATA (NTP, 1991; Yang et al., 1989; see Table 1 in main document).

BMDS MODEL RUN: Weibull

The form of the probability function is:

$$P[\text{response}] = \text{background} + (1 - \text{background}) * [1 - \text{EXP}(-\text{slope} * \text{dose}^{\text{power}})]$$

Power parameter is restricted as power ≥ 1

Total number of observations = 6

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

Default Initial (and Specified) Parameter Values

Background = 0.0454545

Slope = 0.158569

Power = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -Background
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

	Slope	Power
Slope	1	0.93
Power	0.93	1

Parameter Estimates

Variable	Estimate	Std. Err.
Background	0	NA
Slope	23.743	24.862
Power	3.36618	1.18082

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-6.50166			
Fitted model	-6.50166	1.54667e-011	4	1
Reduced model	-38.1909	63.3784	5	<.0001

AIC: 17.0033

Goodness of Fit

Dose	Est._Prob.	Expected	Scaled		Residual
			Observed	Size	
0.0000	0.0000	0.000	0	10	0
0.2000	0.1000	1.000	1	10	-3.148e-006
0.5000	0.9000	9.000	9	10	2.358e-006
1.8000	1.0000	10.000	10	10	0
4.5000	1.0000	10.000	10	10	0
19.2000	1.0000	10.000	10	10	0

Chi-square = 0.00 DF = 4 P-value = 1.0000

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.2

BMDL = 0.0992532

BMDS MODEL RUN: gamma

The form of the probability function is:

$P[\text{response}] = \text{background} + (1 - \text{background}) * \text{CumGamma}[\text{slope} * \text{dose}, \text{power}]$,
 where CumGamma(.) is the cumulative Gamma distribution function

Power parameter is restricted as power ≥ 1

Total number of observations = 6

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

Default Initial (and Specified) Parameter Values

Background = 0.0454545

Slope = 2.65597

Power = 1.3

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -Background
 have been estimated at a boundary point, or have been specified by the user,
 and do not appear in the correlation matrix)

	Slope	Power
Slope	1	0.98
Power	0.98	1

Parameter Estimates

Variable	Estimate	Std. Err.
Background	0	NA
Slope	24.0026	14.4638
Power	8.1874	4.77621

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-6.50166			
Fitted model	-6.50166	1.40514e-009	4	1
Reduced model	-38.1909	63.3784	5	<.0001

AIC: 17.0033

Goodness of Fit

Dose	Est._Prob.	Expected	Scaled		Residual
			Observed	Size	
0.0000	0.0000	0.000	0	10	0
0.2000	0.1000	1.000	1	10	3.123e-005
0.5000	0.9000	9.000	9	10	1.059e-005
1.8000	1.0000	10.000	10	10	1.26e-005
4.5000	1.0000	10.000	10	10	0
19.2000	1.0000	10.000	10	10	0

Chi-square = 0.00 DF = 4 P-value = 1.0000

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.200001

BMDL = 0.110583

BMDS MODEL RUN: log-logistic

The form of the probability function is:

$$P[\text{response}] = \text{background} + (1 - \text{background}) / [1 + \text{EXP}(-\text{intercept} - \text{slope} * \text{Log}(\text{dose}))]$$

Slope parameter is restricted as slope ≥ 1

Total number of observations = 6

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

User has chosen the log transformed model

Default Initial Parameter Values

background = 0

intercept = 1.20641

slope = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -background
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

	intercept	slope
intercept	1	0.93
slope	0.93	1

Parameter Estimates

Variable	Estimate	Std. Err.
background	0	NA
intercept	5.53548	2.00046
slope	4.8069	1.61467

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-6.50166			
Fitted model	-6.50405	0.00478203	4	1
Reduced model	-38.1909	63.3784	5	<.0001

AIC: 17.0081

Goodness of Fit

Dose	Est._Prob.	Expected	Scaled		Residual
			Observed	Size	
0.0000	0.0000	0.000	0	10	0
0.2000	0.0997	0.997	1	10	0.003517
0.5000	0.9006	9.006	9	10	-0.006035
1.8000	0.9998	9.998	10	10	0.04836
4.5000	1.0000	10.000	10	10	0.005346
19.2000	1.0000	10.000	10	10	0.0001635

Chi-square = 0.00 DF = 4 P-value = 1.0000

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.200154

BMDL = 0.122758

BMDS MODEL RUN: log-probit

The form of the probability function is:

$$P[\text{response}] = \text{Background} + (1 - \text{Background}) * \text{CumNorm}(\text{Intercept} + \text{Slope} * \text{Log}(\text{Dose})),$$

where CumNorm(.) is the cumulative normal distribution function

Slope parameter is restricted as slope ≥ 1

Total number of observations = 6

Total number of records with missing values = 0

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

User has chosen the log transformed model

Default Initial (and Specified) Parameter Values

background = 0
 intercept = -0.328418
 slope = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -background
 have been estimated at a boundary point, or have been specified by the user,
 and do not appear in the correlation matrix)

	intercept	slope
intercept	1	0.93
slope	0.93	1

Parameter Estimates

Variable	Estimate	Std. Err.
background	0	NA
intercept	3.22052	1.03364
slope	2.7973	0.834194

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-6.50166			
Fitted model	-6.50167	1.14638e-005	4	1
Reduced model	-38.1909	63.3784	5	<.0001

AIC: 17.0033

Goodness of Fit

Dose	Est._Prob.	Expected	Scaled		Residual
			Observed	Size	
0.0000	0.0000	0.000	0	10	0
0.2000	0.1000	1.000	1	10	2.746e-005
0.5000	0.9000	9.000	9	10	-4.763e-005
1.8000	1.0000	10.000	10	10	0.002394
4.5000	1.0000	10.000	10	10	7.437e-007
19.2000	1.0000	10.000	10	10	0

Chi-square = 0.00 DF = 4 P-value = 1.0000

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.200001

BMDL = 0.125663

BMDS MODEL RUN: Multistage (1st-order)

The form of the probability function is:

$$P[\text{response}] = \text{background} + (1 - \text{background}) * [1 - \text{EXP}(-\text{beta1} * \text{dose}^1)]$$

The parameter betas are restricted to be positive

Total number of observations = 6

Total number of records with missing values = 0

Total number of parameters in model = 2

Total number of specified parameters = 0

Degree of polynomial = 1

Maximum number of iterations = 250

Relative Function Convergence has been set to: 1e-008

Parameter Convergence has been set to: 1e-008

Default Initial Parameter Values

Background = 1

Beta(1) = 4.46022e+018

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -Background
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

Beta(1)

Beta(1) 1

Parameter Estimates

Variable	Estimate	Std. Err.
Background	0	NA
Beta(1)	2.51013	0.870953

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-6.50166			
Fitted model	-9.83272	6.66212	5	0.247
Reduced model	-38.1909	63.3784	5	<.0001

AIC: 21.6654

Goodness of Fit

	Dose	Est._Prob.	Expected	Observed	Size	Chi^2 Res.
i: 1	0.0000	0.0000	0.000	0	10	0.000
i: 2	0.2000	0.3947	3.947	1	10	-1.234
i: 3	0.5000	0.7149	7.149	9	10	0.908
i: 4	1.8000	0.9891	9.891	10	10	1.011
i: 5	4.5000	1.0000	10.000	10	10	1.000
i: 6	19.2000	1.0000	10.000	10	10	0.000

Chi-square = 5.43 DF = 5 P-value = 0.3661

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.0419741

BMDL = 0.0265053

Provisional Peer Reviewed Toxicity Values for
Iron and Compounds
(CASRN 7439-89-6)

Derivation of a Chronic Inhalation RfC

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
IRON (CASRN 7439-89-6) AND COMPOUNDS
Derivation of an Inhalation RfC**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

An RfC for iron is not listed on IRIS (U.S. EPA, 2001) and was not considered by the RfD/RfC Work Group (U.S. EPA, 1995). The HEAST (U.S. EPA, 1997) reported that data regarding iron were inadequate for quantitative risk assessment. The CARA list (1991, 1994a) includes a Health Effects Assessment for Iron and Compounds (U.S. EPA, 1984) that reported negative epidemiological studies (no association between excess mortality or respiratory diseases and occupational exposure to iron oxide dusts) and no available subchronic or chronic inhalation studies in animals. In March, 2004, a literature search was also conducted using TOXLINE, MEDLINE, Chemical Abstracts and Biological Abstracts data bases.

Occupational exposure limits have been established for soluble iron salts and iron oxide, as well as for organic iron compounds not covered in this issue paper. The ACGIH (1991a,

2001) has adopted a TLV-TWA, NIOSH (2001a) has established a REL-TWA, and OSHA (2001a, 2001b) has adopted a construction industry PEL-TWA of 1 mg/m³, as Fe, to reduce the likelihood of irritation to eyes, skin, and respiratory tract from exposure to aerosols or mists of soluble iron salts (ferrous and ferric sulfates and chlorides, and ferric nitrate). The ACGIH (1991b, 2001) has adopted a TLV-TWA and NIOSH (2001b) has established a REL-TWA of 5 mg/m³, as Fe, for dust and fume of ferric oxide (Fe₂O₃) to protect against siderosis, a benign pneumoconiosis. OSHA (2001c) has adopted a PEL-TWA of 10 mg/m³ for ferric oxide fume, to protect against accumulation of iron dust in the lungs.

Iron has not been the subject of a toxicological profile by ATSDR (2001) or the WHO (2001). Monographs by IARC (1972, 1984, 1987), a toxicity review on iron (Grimsley, 2001), and the NTP (2001a, 2001b) management status report and chemical repository summary were consulted for information relevant to inhalation toxicity of iron and inorganic iron compounds. The following computer searches, performed in April, 1993, were screened to identify additional pertinent studies not discussed in review documents: TOXLINE (1983-April, 1993), CANCERLIT (1990 - April, 1993), MEDLINE (1991 - April, 1993), TSCATS, RTECS, and HSDB. Update literature searches were conducted in September, 2001 in TOXLINE (1992-September, 2001), CANCERLIT (1992-September, 2001), MEDLINE (1992-September, 2001), TSCATS, RTECS, DART/ETICBACK, EMIC/EMICBACK, HSDB, GENETOX, and CCRIS.

REVIEW OF PERTINENT LITERATURE

Human Studies

A number of studies have examined the relationship between respiratory disease and inhalation exposure to iron compounds for workers employed in hematite mining or other iron-related occupations, such as welding or steel-making (U.S. EPA, 1984; IARC, 1972, 1984; Grimsley, 2001). However, since these studies involved concurrent exposure to silica and other metals, they are not suitable for the health risk assessment of iron or iron compounds. The literature search did not discover any studies that examined subchronic or chronic inhalation exposures of humans to quantified levels of iron or iron compounds alone.

In a case-control study of cancer incidence, a Swedish male worker population (1958-1971) was reported to have had a high exposure to iron oxides from the production of sulfuric acid from pyrite (FeS₂) (Axelson and Sjöberg, 1979). The workers were exposed to iron oxide (Fe₂O₃) along with 1-2% copper, 0.01-0.1% arsenic, nickel and cobalt as impurities. Exposure in the workroom was estimated as approximately 50-100 mg/m³, and the particle size as 25% below 10 µm and 5-10% below 5 µm. However, there were no measurements of exposure levels or particle size, and exposure durations were not reported. No cases of siderosis were known from the plant.

Animal Studies

Inhalation studies for iron compounds in animals include a chronic study of hamsters exposed to ferric oxide (Fe_2O_3) dust (Nettesheim et al., 1975) and a 2-month study in rabbits exposed to aerosols of ferric chloride (Johansson et al., 1992).

In a cancer study, groups of male Syrian hamsters (132 per group) were exposed to filtered air or Fe_2O_3 (analytic grade) dust at a concentration of 40 mg/m^3 , 6 hours/day, 5 days/week for life (Nettesheim et al., 1975). The particle size had a geometric mean diameter of $0.11 \text{ }\mu\text{m}$. In addition, two satellite groups (15 hamsters per treatment) were sacrificed, three animals at a time, at 2, 4, 8, 12, and 104 weeks, so that the accumulation of iron in the lung from inhaled Fe_2O_3 could be compared to background iron concentrations in heme. The animals were examined daily, before and after each exposure, for clinical signs, and body weights were recorded monthly. All animals except those cannibalized (<2%) were necropsied. Histological analyses were performed on the major organs, including heart, trachea, lungs, and nasal cavities. Examination of the satellite groups demonstrated the gradual increase in iron accumulation in the lung, reaching a total of 10 mg per lung at 104 weeks. Histological examination revealed iron deposits in the lungs and tracheal and bronchial lymph nodes of all exposed animals. Diffuse and focal alveolar fibrosis was also frequently observed in the lungs of treated animals. Results for the histological endpoints were not reported quantitatively. In this study, 40 mg/m^3 is a LOAEL for respiratory effects (alveolar fibrosis) in hamsters exposed to Fe_2O_3 dust.

Groups of 8 male rabbits (strain not reported) were exposed to aerosols of 0, 1.4, or 3.1 mg/m^3 of iron as FeCl_3 6 hours/day, 5 days/week for 2 months (Johansson et al., 1992). At termination, the upper left lung lobe was examined by light microscopy, pieces of the lower left lung were analyzed by electron microscopy or used for phospholipid analysis, and the right lung was lavaged to obtain macrophages for morphological and functional analyses. The mass median aerodynamic diameter of the aerosols was $\sim 1 \text{ }\mu\text{m}$ as measured with an impactor. Treatment had no effect on survival. Lungs were spotted with black in 7/8 high-iron rabbits, in 2/8 low-iron rabbits, and in 0/8 controls. The absolute weight of the left lower lobe of the lung was significantly elevated compared to controls in the high-iron group. Exposure-related histopathology was observed in the lungs. In the high-exposure group, the lungs contained naked granulomas [large nodules ($\geq 1 \text{ mm}$) of densely packed granular macrophages], accumulations of granular macrophages in terminal bronchioles, and foci of interstitial lymphocytic inflammatory reaction. Small granulomas were observed in one low-iron and one control rabbit. Accumulations of normal and granular macrophages were observed in the alveoli of exposed rabbits. In the control group, normal lung tissue contained some small accumulations of macrophages with occasional small inflammatory reaction. The high exposure group had a significantly higher density of alveolar type II cells than the controls. Ultrastructural analysis of macrophages showed a significantly higher number of abnormal cells, cells with enlarged lysosomes, and black inclusions in cells in both exposed groups; the high-iron group had higher

percentages of cells with laminar inclusions or with smooth cell surfaces. In functional tests, macrophages from the high-exposure group showed significantly elevated phagocytic activity, but no significant increase in oxidative metabolic activity (superoxide generation). Total phospholipids were elevated in the high-exposure group, but, as indicated by the lack of increase in phosphatidyl cholines or the percentage of 1,2-dipalmitoylphosphatidylcholine, the amount of surfactant was unchanged. In this study, the low concentration of 1.4 mg/m^3 is a NOAEL and the high concentration of 3.1 mg/m^3 is a LOAEL for adverse lung effects (nodular granulomas $\geq 1 \text{ mm}$ in diameter, abnormal macrophages) in rabbits exposed to ferric chloride aerosols. Because of its focus on alveolar macrophage effects, this study provided no information regarding clinical signs of toxicity, body weight changes, clinical biochemistry, nasopharyngeal effects or histology of any other tissue besides the lung.

Other Studies

In a cancer study, groups of Syrian golden hamsters (24 per sex per group) received intratracheal instillations of 0 or “a maximum dose”¹ of 3 mg of Fe_2O_3 dust in 0.2 ml of saline once a week for 15 weeks, and then were observed up to week 120 (Stenbäck et al., 1976). Analysis by the sedimentation method demonstrated that 98% of the particles were less than $10 \mu\text{m}$ in diameter. Animals were weighed weekly and autopsied. Organs with gross lesions and the larynx, trachea, bronchi, and lungs were examined histologically. Treatment with ferric oxide had no effect on survival and no effect on body weight except during the final weeks of survival (data not shown). Deposited iron oxide was grossly visible as dark patches on the lung surface. Histologically, dust accumulations surrounded by cellular infiltrates were observed in the peribronchial region. Interstitial fibrosis was observed occasionally, but distinct inflammatory changes were rare. Results for the nonneoplastic endpoints were not reported quantitatively.

FEASIBILITY OF DERIVING A PROVISIONAL RfC FOR IRON

No adequate human or animal inhalation data are available for exposure to iron or inorganic iron compounds. The epidemiological study of Axelson and Sjöberg (1979) did not provide quantitative measures of exposure and did not characterize noncancer endpoints. Although Nettesheim et al. (1975) reported diffuse and focal alveolar fibrosis in the lungs of hamsters chronically exposed to iron oxide by inhalation at a concentration of 40 mg/m^3 , the lack of incidence data prevents an evaluation of the significance of these findings. The subchronic study of Johansson et al. (1992), in which rabbits were exposed to aerosols of ferric chloride for

¹The authors provided no further information regarding dosage. It is not clear whether animals were given amounts lower than 3 mg on some occasions.

2 months, demonstrated a NOAEL of 1.4 mg/m³ and a LOAEL of 3.1 mg/m³ for respiratory effects (granuloma nodules greater than 1 mm diameter in the lungs). However, this study does not meet the minimum standards for an inhalation bioassay as stipulated by the U.S. EPA (1994b) guidelines for derivation of an inhalation reference concentration. Inadequacies of the study include relatively small group sizes, relatively short study duration, and the failure to examine a sufficient array of endpoints. Thus this study is inadequate for the purposes of deriving a p-RfC for iron. Consequently, the available data are insufficient for derivation of a p-RfC.

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Provisional Peer Reviewed Toxicity Values for
Iron and Compounds
(CASRN 7439-89-6)

Derivation of a Carcinogenicity Assessment

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
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MRL	minimal risk level

MTD	maximum tolerated dose
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NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
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NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
IRON (CASRN 7439-89-6) AND COMPOUNDS
Derivation of a Carcinogenicity Assessment**

Background

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2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
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It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A cancer assessment for iron is not listed on IRIS (U.S. EPA, 2005a), the HEAST (U.S. EPA, 1997), or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2000), and was not considered by the CRAVE Work Group (U.S. EPA, 1995). The CARA list (1991, 1994) includes a Health Effects Assessment for Iron and Compounds (U.S. EPA, 1984) that assigned iron and its compounds to weight-of-evidence Group C, possible human carcinogen. This assessment was based on conflicting evidence of lung tumors following occupational inhalation exposure to ferric oxide (mixed exposure), and injection-site tumors in one patient and in mice treated with iron-dextran. IARC (1972, 1987) assigned ferric oxide to Group 3, not classifiable as to its carcinogenicity to humans based on inadequate data in humans (increased incidence of lung cancer following occupational exposure to iron dusts in mixtures) and apparently negative

evidence for carcinogenicity in mice, hamsters and guinea pigs exposed by inhalation or intratracheal instillation. For ferric oxide dust and fume, the ACGIH (1991, 2001) lists an A4 notation, not classifiable as a human carcinogen; this is based on mixed exposure studies in humans and primarily negative studies in animals. In March, 2004, a literature search was also conducted using TOXLINE, MEDLINE, Chemical Abstracts and Biological Abstracts data bases.

Iron has not been the subject of a toxicological review by ATSDR (2001) or the WHO (2001). Monographs by IARC (1972, 1984, 1987), a toxicity review on iron (Grimsley, 2001), and the NTP (2001a, 2001b) management status report and chemical repository summary were consulted for information relevant to the carcinogenicity of iron and inorganic iron compounds. The following computer searches, performed in April, 1993, were screened to identify additional pertinent studies not discussed in review documents: TOXLINE (1983-April, 1993), CANCERLIT (1990 - April, 1993), MEDLINE (1991 - April, 1993), TSCATS, RTECS, and HSDB. Update literature searches were conducted in September, 2001 in TOXLINE (1992-September, 2001), CANCERLIT (1992- September, 2001), MEDLINE (1992-September, 2001), TSCATS, RTECS, DART/ETICBACK, EMIC/EMICBACK, HSDB, GENETOX, and CCRIS.

REVIEW OF PERTINENT LITERATURE

Human Studies

Oral Exposure

Because iron is an essential element, the NAS (2001) has established guidelines for daily dietary intakes, based on gender, age, and physiological status, that are designed to avoid adverse effects of deficiency and excess. Individuals of northern European descent who are affected by hereditary hemochromatosis, an autosomal, recessive disorder, are not protected by these guidelines. These individuals exhibit excessive absorption of dietary iron, which results in abnormally high accumulations of iron in liver and brain tissues. When the liver consequently develops cirrhosis, the risk of developing primary hepatocellular carcinoma increases significantly. It is not clear whether these findings are relevant to excess iron intake by the general population.

Bird et al. (1996) investigated the association between plasma ferritin and iron intake and the development of adenomatous polyps, which are intermediate markers for colorectal cancer. The study population consisted of men and women between the ages of 50 and 75 years old who underwent routine screening by flexible sigmoidoscopy at one of two medical centers during 1991-1993. Individuals with cancer, inflammatory bowel disease, or familial polyposis were excluded. Cases (300 men and 167 women) were subjects diagnosed for the first time with one

or more histologically confirmed adenomatous polyps. Controls (331 men and 167 women) had no history of polyps and none discovered at sigmoidoscopy. Cases and controls were matched by sex, age (± 5 years), date of sigmoidoscopy (± 3 months), and medical center. Plasma ferritin levels, hematocrit, and certain nutritional indicators (carotenoids, ascorbate, folate) were measured in blood samples drawn 6 months after examination. Iron intakes for the year preceding sigmoidoscopy were estimated by means of a semiquantitative food frequency questionnaire. After controlling for possible confounding factors, subjects with high plasma ferritin levels ($>289 \mu\text{g/L}$) had a multivariate-adjusted odds ratio for colorectal polyps of 1.5 (95% confidence interval (C.I.) = 1.0-2.3) compared to subjects with low/normal levels ($73\text{-}141 \mu\text{g/L}$). The pattern for iron intake was U-shaped. Compared with subjects consuming an adequate amount of iron ($11.6\text{-}13.6 \text{ mg/day}$), multivariate-adjusted odds ratios for colorectal polyps in men were 1.6 (95% C.I. = 1.1-2.4) for intakes below 11.6 mg/day and 1.4 (95% C.I. = 0.9-2.0) for intakes above 27.3 mg/day . The highest odds ratio of 2.1 (95% C.I. = 1.3-3.5) was found after further adjustment for smoking for men at the lowest level of iron intake. The association between iron intake and colorectal polyps disappeared when exposure group class of reaction was based on dietary intake alone (i.e., high iron supplementation ignored). The authors concluded that there was a weak positive association between iron exposure and colorectal polyps that may increase the risk of colorectal cancer but note that some factor in supplementation may have been responsible for the effect.

Inhalation Exposure

Most studies of cancer incidence following occupational exposure to iron dust are excluded from consideration because of confounding exposures to silica, radon daughters, soot, asbestos, or other types of metals in the study populations (U.S. EPA, 1984; IARC, 1972, 1984, 1987).

A case-control study examined cancer incidence in a Swedish male worker population (1958-1971) with a high exposure to iron oxides from the production of sulfuric acid from pyrite (FeS_2) (Axelson and Sjöberg, 1979). The workers were exposed to iron oxide (Fe_2O_3) along with 1-2% copper, 0.01-0.1% arsenic, nickel and cobalt as impurities. Exposure in the workroom was estimated as approximately $50\text{-}100 \text{ mg/m}^3$, and the particle size as 25% below $10 \mu\text{m}$ and 5-10% below $5 \mu\text{m}$. No cases of siderosis were known from the plant. The Swedish National Cancer Register was consulted for locating cases of cancer that could have been caused by environmental exposure; the study examined cancers of the stomach, liver, lung, kidney, and bladder, and hematological malignancies. Each cancer case was matched with two controls from the local population register by matching for sex, age, and residency in the same or adjacent neighborhood block. Company files were searched to determine the length of exposure; those with less than 5 months of exposure were considered to be nonexposed. The study found no association between exposure to iron oxides and any of the selected types of cancer.

Animal Studies

Oral Exposure

Groups of F344 rats (50 per sex per group) were given ferric chloride ($\text{FeCl}_3 \cdot 6\text{H}_2\text{O}$) in drinking water at concentrations of 0, 0.25, or 0.5% (weight/volume) for 104 weeks, and then given distilled water for an 8 week recovery period (Sato et al., 1992). The intake of ferric chloride was reported to be 0, 169.7, or 319.7 mg/kg-day for males and 0, 187.9, or 336.0 mg/kg-day for females. The iron intakes were 0, 58.4, or 110 mg/kg-day in males and 0, 64.6, or 115.6 mg/kg-day in females. Rats were observed daily for clinical signs and mortality. Body weights were measured once a week for 13 weeks and every fourth week thereafter. All rats dying prematurely and survivors at week 112 were examined for gross and microscopic neoplastic and non-neoplastic lesions. There were dose-related decreases in drinking water intake and terminal body weight in both sexes. These may have been related to reduced palatability. Survival in both sexes was not significantly affected by exposure to ferric chloride. No increases in tumor incidence were observed in rats exposed to ferric chloride for two years.

Inhalation Exposure

Groups of male Syrian hamsters (132 per group) were exposed to filtered air or Fe_2O_3 (analytic grade) dust at a concentration of 40 mg/m³, 6 hours/day, 5 days/week for life (Nettesheim et al., 1975). The particle size had a geometric mean diameter of 0.11 μm . In addition, two satellite groups (15 hamsters per treatment) were sacrificed, three animals at a time, at 2, 4, 8, 12, and 104 weeks, so that the accumulation of iron in the lung from inhaled Fe_2O_3 could be compared to background iron concentrations in heme. The animals were examined daily, before and after each exposure, for clinical signs; body weights were recorded monthly. All animals except those cannibalized (<2%) were necropsied. Histological analyses were performed for the major organs, including heart, trachea, lungs, and nasal cavities. Examination of the satellite groups demonstrated a gradual increase in iron accumulation in the lung, reaching a total of 10 mg per lung at 104 weeks. Exposure to Fe_2O_3 had no effect on survival or body weight gain and did not increase the incidence of tumors. The authors concluded that inhalation of Fe_2O_3 was not carcinogenic to hamsters.

Groups of Syrian golden hamsters (24 per sex per group) received intratracheal instillations of 0 or 3 mg¹ of Fe_2O_3 dust in 0.2 ml of saline once a week for 15 weeks, and then were observed up to week 120 (Stenbäck et al., 1976). Analysis by the sedimentation method demonstrated that 98% of the particles were less than 10 μm in diameter. Animals were weighed

¹The authors characterized the treatment as a 'maximum dose of 3 mg'. It is not clear whether the hamsters received lower doses on some occasions.

weekly and autopsied. Organs with gross lesions and the larynx, trachea, bronchi, and lungs were examined histologically. Treatment with ferric oxide had no effect on survival and did not affect body weight except during the final weeks of survival (data not shown). Treatment did not induce tumors of the respiratory tract and the incidence of forestomach papillomas in the treatment group was less than in the control group.

Other Studies

Genotoxicity

Genotoxicity assays of inorganic iron salts were primarily negative in bacteria, but were more often positive in mammalian systems. Iron did not induce reverse mutations in *Salmonella typhimurium* strains TA98, TA102, TA1535, or TA1537, with or without activation (Wong, 1988). Ferric chloride and ferrous sulfate tested negative in strains TA98, TA100, TA1535, TA1537, and TA1538 with or without metabolic activation (Shimizu et al., 1985; Dunkel et al., 1999). Ferrous sulfate also tested negative in strains TA97 and TA102, with or without activation (Fujita et al., 1994), but positive in TA1537 and TA1538 (U.S. EPA, 1984). Ferrous and ferric chloride did not induce DNA repair in *Bacillus subtilis* (rec assay) (Leifer et al., 1981). Ferrous sulfate increased the frequency of mutations at the TK locus of mouse L5178Y lymphoma cells, with or without metabolic activation, but only at high concentrations that were likely to be cytotoxic; ferric chloride only increased the frequency of TK mutations when tested with metabolic activation (Dunkel et al., 1999). Ferrous sulfate did not induce sister chromatid exchanges *in vitro* (Ohno et al., 1982). DNA-protein cross-links were generated in mammalian cells cultured in the presence of ferrous iron (Altman et al., 1995). Single- and double-strand DNA breaks were produced in supercoiled plasmid DNA (Toyokuni and Sagripanti, 1992) and in isolated rat liver nuclei (U.S. EPA, 1984) treated with ferrous or ferric chloride. No breakage was detected electrophoretically in Chinese hamster ovary cell DNA treated with ferrous chloride (U.S. EPA, 1984). In a model of oxidative damage within cells, ferrous sulfate, in the presence of hydrogen peroxide, was demonstrated to induce double-strand breaks and intra-strand cross-links in DNA *in vitro* (Lloyd and Phillips, 1999).

Cell transformation

Iron compounds have yielded variable results in studies of cell transformation *in vitro*. Particles of magnetite (Fe_3O_4) induced transformation of cultured a Chinese hamster lung cell line (V_{79}), but only at cytotoxic concentrations (Elias et al., 1995). Ferrous chloride and ferrous sulfate induced cell transformation in viral-enhanced Syrian hamster embryo (SA7/SHE) cells (U.S. EPA, 1984).

Mechanistic Studies

Adverse effects of iron are thought to be related to the formation of reactive oxygen species via the Fenton reaction (Henle and Linn, 1997). Hydrogen peroxide can react with ferrous ion, resulting in the conversion to ferric ion and the production of hydroxyl radicals. Ferric ion can also react with hydrogen peroxide, producing superoxide radical. Reactive oxygen species may react with DNA. However, because of the complex homeostatic mechanisms involved in iron transport and metabolism, unbound ferrous iron is not likely to be present except in conditions of excessive iron intake.

PROVISIONAL WEIGHT-OF-EVIDENCE CLASSIFICATION

U.S. EPA (1984) classified iron and its compounds, including ferric dextran, as possible human carcinogens (Group C). This assessment was based on reports associating an increased incidence of lung cancer with exposure to hematite dust (confounded by coincident exposures to tobacco, alcohol, silica, soot, and fumes of other metals), inconsistent reports of lung tumors in animals exposed by inhalation or tracheal instillation to ferric oxide, and reports of injection site tumors in one patient injected with iron dextran and in mice injected with iron dextran or saccharated iron oxide. The current PPRTV assessment excludes organic forms of iron and studies in which the levels of impurities are significant.

Results of the case-control study by Bird et al. (1996) provide evidence of a weak association between elevated iron intake or high plasma ferritin (a measure of body stores) and the prevalence of adenomatous colorectal polyps, a possible precursor to colorectal cancer. Weaknesses of this study include the 6-month period between examination and ferritin measurements, and the possible recall errors affecting the dietary questionnaire for the previous year. In addition, the association between iron intake and colorectal polyps was stronger at low iron intake and not related to dietary (i.e., environmental) intake. Although the association between cirrhotic hereditary hemochromatosis and hepatocellular carcinoma is well established, the evidence for dietary iron intake and hepatic cancer in the general population was characterized by the NAS (2001) as inconclusive. In a chronic rat assay, Sato et al. (1992) found no evidence of carcinogenicity of ferric chloride ingested in drinking water at concentrations up to 0.5%. In summary, the evidence for carcinogenicity of ingested inorganic iron compounds in humans and animals is inadequate.

Evidence from the case-control study of Axelson and Sjöberg (1979) suggests that inhaled iron oxide may not be carcinogenic to humans. However, uncertainty remains because levels of exposure were not measured, the durations of exposure were not reported, and individuals exposed for up to 5 months were categorized as 'nonexposed.' In addition, the lack of reported cases of siderosis in the workplace suggests that the exposure levels may have been

lower than estimated. Thus, the evidence for carcinogenicity of inhaled iron oxide in humans is considered inadequate. Results of the study of Nettesheim et al. (1975) indicate that chronic inhalation exposure to iron oxide at a concentration of 40 mg/m³ is not carcinogenic to hamsters. This finding is supported by the negative results for carcinogenicity of iron oxide administered by intratracheal instillation to hamsters for 15 weeks (Stenbäck et al., 1976). However, as both hamster studies used single exposure concentrations, the possibility of carcinogenicity at higher exposure levels cannot be disregarded.

Following the U.S. EPA (2005b) guidelines for carcinogen risk assessment, the available data are inadequate for an assessment of the human carcinogenic potential of inhaled iron oxide or ingested iron chloride.

QUANTITATIVE ESTIMATES OF CARCINOGENIC RISK

Derivation of quantitative estimates of cancer risk for ingested or inhaled iron or iron oxide is precluded by the absence of adequate data demonstrating carcinogenicity.

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9-11-2006

Provisional Peer Reviewed Toxicity Values for
Iron and Compounds
(CASRN 7439-89-6)

Derivation of Subchronic and Chronic Oral RfDs

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY INFORMATION FOR
IRON (CASRN 7439-89-6) AND COMPOUNDS
Derivation of Subchronic and Chronic Oral RfDs**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

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Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

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Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI

INTRODUCTION

A reference dose (RfD) for iron is not available on the Integrated Risk Information System (IRIS) (U.S. EPA, 2006) or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2005). The Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997) reported that data regarding iron were inadequate for quantitative risk assessment. The Chemical Assessment and Related Activities (CARA) list (1991, 1994) includes a Health Effects Assessment (HEA) for Iron and Compounds (U.S. EPA, 1984) that found no reliable quantitative oral toxicity data. Iron has not been the subject of a toxicological review by the Agency for Toxic Substances Disease Registry (ATSDR) (2005) or the World Health Organization (WHO) (2005). Monographs by the International Agency for Research on Cancer (IARC) (1972, 1987), toxicity reviews by Jacobs (1977), Bothwell et al. (1979), Lauffer (1991) and Grimsley (2001), a review on dietary iron by the National Academy of Sciences (NAS) (2001), and the National Toxicology Program (NTP) (2001, 2005) management status report and chemical repository summary were consulted for relevant information. The NAS (2001) derived a Tolerable Upper Intake (TUI) level of 45 mg iron/day. The TUI is based on a minimal LOAEL of 70 mg/day (60 mg iron as ferrous fumarate plus 11 mg/day of dietary iron) identified by Frykman et al. (1994) for gastrointestinal effects and an uncertainty factor of 1.5 for use of a minimal LOAEL; a higher

uncertainty factor was not used since the nature of the observed gastrointestinal effects was considered to be self-limiting. The U.S. Food and Drug Administration (FDA) promulgated a Rule in 1997 for labeling of iron-containing dietary supplements for the prevention of accidental poisoning in children (U.S. FDA, 1997). The Rule, as modified in 2003, does not contain specific exposure limits (U.S. FDA, 2003). In general, the FDA follows the NAS guidance on exposure limits for toxicity of essential elements, such as iron. Previous literature searches were conducted through September, 2001 as follows: TOXLINE (oral and inhalation toxicity and cancer from 1983 - September, 2001); CANCERLIT (1990 - September, 2001); MEDLINE (1991 - September, 2001); TSCATS, RTECS, DART/ETICBACK, EMIC/EMICBACK, HSDB, GENETOX, and CCRIS. Update literature searches were performed in October, 2005 in MEDLINE, TOXLINE (NTIS subfile), TOXCENTER, TSCATS, CCRIS, DART/ETIC, GENETOX, HSDB, RTECS and Current Contents.

REVIEW OF PERTINENT LITERATURE

Iron is an essential element and deriving a risk assessment value for such chemicals poses a special problem in that the dose-adversity curve is "U-shaped". Thus, the risk value must be protective against deficiency as well as toxicity. The NAS (2001) has established guidelines for iron intake that take into account physiological differences during different life stages. For non-breast-fed infants aged 0-6 months, the NAS (2001) established a daily adequate intake (AI) for iron of 0.27 mg/day (0.04 mg/kg-day for infants 2-6 months old) based on the daily amount of iron secreted in human milk; breast-fed infants typically receive only 0.15 to 0.3 mg Fe/day. The NAS (2001) Dietary Reference Intakes (DRIs) for children are as follows: 11 mg/day (1.2 mg/kg-day) for infants between the ages of 7 and 12 months, 7 mg/day (0.54 mg/kg-day) for children aged 1-3 years, 10 mg/day (0.45 mg/kg-day) for ages 4-8 years, 8 mg/day (0.2 mg/kg-day) for ages 9-13 years and 11 mg/day (0.17 mg/kg-day) for boys and 15 mg/day (0.26 mg/kg-day) for girls aged 14-18 years. The DRI for men aged 19 years and above is 8 mg/day (0.11 mg/kg-day). The DRI for non-pregnant women is 18 mg/day (0.29 mg/kg-day) for ages between 19 and 50 years and 8 mg/day (0.13 mg/kg-day) for ages 51 years and older. The DRI for pregnant women is 27 mg/day (0.37 mg/kg-day for those aged 14-18 years and 0.35 mg/kg-day for those aged 19-50 years). The DRI during lactation is 10 mg/day (0.18 mg/kg-day) for women aged 14-18 years and 9 mg/day (0.15 mg/kg-day) for women aged 19-50 years.

According to the Centers for Disease Control and Prevention (CDC, 1998; CDC, 2005), iron deficiency is one of the most common known forms of nutritional deficiency. Its prevalence is highest among young children and women of childbearing age, particularly pregnant women. In children, iron deficiency causes developmental delays and behavioral disturbances, and in pregnant women, it increases the risk for a preterm delivery and delivering a low-birthweight baby. Young children are at great risk of iron deficiency because of rapid growth and increased iron requirements. Iron deficiency can occur due to lack of iron in the diet. If this continues, anemia results. Anemia is a manifestation of iron deficiency when it is relatively severe. Iron deficiency anemia significantly impairs mental and psychomotor development in infants and children. Although iron deficiency can be reversed with treatment, the reversibility of the mental and psychomotor impairment is not yet clearly understood. Thus, prevention and treatment need to be emphasized more than detection. In addition, iron deficiency increases a child's

susceptibility to lead toxicity. Lead replaces iron in the absorptive pathway when iron is unavailable.

In humans and other animals, levels in the body are regulated primarily through changes in the amount of iron absorbed by the gastrointestinal mucosa. The absorption of dietary iron is influenced by body stores, by the amount and chemical nature of iron in ingested food and by a variety of dietary factors that increase or decrease the availability of iron for absorption (Hillman, 2001; Santi and Masters, 2001). Iron contained in meat protein (hemoglobin and myoglobin) is absorbed intact without first being broken down to elemental iron. Non-heme iron must first be reduced to ferrous iron (Fe^{2+}) before it can be absorbed. Ferrous iron is transported across intestinal mucosal cells by active transport with the rate of transport inversely related to body iron stores. Depending upon the iron status of the body, iron is stored bound to ferritin within mucosal cells and macrophages in the liver, spleen and bone, or is transported in the plasma bound to transferrin. Serum levels of ferritin and transferrin, along with several red blood cell parameters, can be used clinically to evaluate iron balance. Although iron absorption is regulated, excessive accumulation of iron in the body resulting from chronic ingestion of high levels of iron cannot be prevented by intestinal regulation and humans do not have a mechanism to increase excretion of absorbed iron in response to elevated body levels (NAS, 1989, 2001).

Human Studies

Acute Exposure

Information on acute oral toxic doses of iron in humans is available from numerous case reports of ingestion by children, but values vary because it is difficult to obtain accurate estimates of the amount taken in most overdose situations. Reviews of these case reports indicate that doses in the range of 200-300 mg iron/kg are generally considered lethal (Arena, 1970; Krenzelok and Hoff, 1979; NRC, 1979; Engle et al., 1987; Mann et al., 1989; Klein-Schwartz et al., 1990).

Therapeutic Studies

Ferrous salts are administered orally for the therapeutic treatment of iron deficiency. The oral absorption of ferrous iron supplements is considered to be essentially the same for all ferrous salts (e.g., sulfate, fumarate, succinate and gluconate) and is approximately three times greater than that of ferric (Fe^{3+}) salts (Hillman, 2001); thus, ferric iron is not used therapeutically. Constipation and other gastrointestinal effects, including nausea, vomiting, diarrhea and gastrointestinal pain are commonly associated with administration of oral ferrous salt supplements (Hillman, 2001; Santi and Masters, 2001). Severity of effects is variable, ranging from mild to severe, and depends upon dose and individual susceptibility. The onset of symptoms typically occurs at the initiation of treatment and continues throughout the duration of treatment. Although there is no indication that the severity of gastrointestinal effects varies over the course of treatment, severity is decreased in some patients when iron supplements are administered with food (Hillman, 2001; Santi and Masters, 2001). For most patients, iron deficiency is reversed within six months of treatment, thus limiting the duration of exposure.

The mechanism of iron-induced gastrointestinal toxicity is not established, although it is postulated that adverse effects are due to irritant effects of the free iron ion on the gastric mucosa (Liguori, 1993). The role of absorbed iron in the development of gastrointestinal adverse effects is unknown. The adverse effects of exposure to oral iron supplements has been investigated in several studies (Blot et al., 1981; Brock et al., 1985; Coplin et al., 1991; Frykman et al., 1994; Hallberg et al., 1966; Liguori, 1993).

Frykman et al. (1994) evaluated the adverse effects of daily oral therapy with iron fumarate in a double-blind, crossover, placebo-controlled study in Swedish male [n=25; mean age 45 years (range 40-52)] and female [n=23; mean age 41 years (range 34-45)] adult blood donors. Study subjects were administered 60 mg elemental iron as a daily dose of iron fumarate for one month, with each study subject serving as their own placebo control. Compared to the placebo treatment period, the percentage of subjects reporting constipation (placebo 20%, ferrous fumarate 35%, $p<0.05$) and total gastrointestinal symptoms (nausea, obstipation, gastric pain and diarrhea (placebo 14%, ferrous fumarate 25%, $p<0.01$) was significantly increased during ferrous fumarate treatment. Although the severity of gastrointestinal effects was graded as minor in most study subjects, four subjects withdrew from the study due to severe gastrointestinal symptoms associated with iron fumarate. In a matched group of 49 adults taking a daily combination supplement of porcine-derived heme-iron and iron fumarate containing a total daily supplement of 18 mg iron/per day, the frequency of gastrointestinal symptoms was not increased compared to placebo. No differences in therapeutic efficacy, as measured by serum ferritin and hemoglobin levels, were observed between the non-heme iron and heme-iron treatment groups.

Adverse effects of four oral iron preparations were evaluated in 1496 male and female adult blood donors in a series of double-blind, placebo controlled trials (Hallberg et al., 1966). The following treatment groups were compared: (1) placebo (195 subjects) and ferrous sulfate (198 subjects; 222 mg elemental iron/day); (2) placebo (199 subjects), ferrous sulfate (120 subjects; 222 mg elemental iron/day), ferrous fumarate (118 subjects, 222 mg elemental iron/day), and ferrous gluconate (120 subjects; 222 mg elemental iron/day); and (3) placebo (200 subjects), ferrous sulfate (195 subjects; 180 mg elemental iron/day), ferrous glycine sulfate (200 subjects; 180 mg elemental iron/day), and ferrous gluconate (196 subjects; 180 mg elemental iron/day). Treatments were administered for two weeks. For all iron treatments, the frequency of adverse gastrointestinal effects was significantly increased compared to the matched placebo group ($p<0.05$). Adverse effects reported include constipation, diarrhea, heartburn, nausea and epigastric pain. No statistically significant differences in the frequency of adverse effects were observed between iron treatments for subjects receiving 222 mg elemental iron/day or between iron treatments for subjects receiving 180 mg elemental iron/day. In the seven iron treatment groups, the percentage of subjects reporting gastrointestinal effects ranged from 22.9% in the 222 mg ferrous sulfate group to 31.5% in the 222 mg ferrous gluconate group. In the three placebo treatment groups, the percentage of subjects reporting gastrointestinal effects ranged from 12.4 to 13.6%. Although statistical comparisons were not made between the 180 and 222 mg iron/day treatments, the frequency of adverse effects was similar for all iron treatment groups.

Gastrointestinal symptoms were reported in pregnant women treated daily with oral iron supplements containing 105 mg elemental iron and 500 mg ascorbic acid (55 women) or 105 mg

elemental iron, 500 mg ascorbic acid and 350 mg folic acid (54 women) during the third trimester of pregnancy (Blot et al., 1981). The form of iron was not reported. No placebo control group was included. Gastrointestinal adverse effects reported include nausea, diarrhea, constipation and epigastric pain. Approximately 16% of all patients reported minor gastrointestinal symptoms, 14% reported severe effects and 6% stopped treatment due to adverse effects. Adverse effects occurred with approximately the same frequency in the two treatment group, although data were not reported.

The tolerability of iron protein succinylate and ferrous sulfate were compared in a double-blind clinical trial in 1095 patients with iron deficiency (Liguori, 1993). Patients received daily treatment with a controlled-release formulation of ferrous sulfate containing 105 mg elemental iron (64 males and 485 females) or iron protein succinylate containing 120 mg elemental iron (55 males and 491 females) for 60 days. No placebo control group was included. In the ferrous sulfate group, 26.3% of patients reported adverse gastrointestinal effects (heartburn, epigastric pain, constipation and abdominal pain), compared to 11.5% of patients treated with iron protein succinylate ($p < 0.05$).

The adverse effects of oral treatment with a conventional ferrous sulfate tablet were compared to a ferrous sulfate wax-matrix tablet in a single-blind, parallel group study in 543 subjects (Brock et al., 1985). No placebo control group was included. Subjects were administered a conventional ferrous sulfate tablet containing 50 mg elemental iron/day (272 subjects) or a sulfate wax-matrix tablet containing 50 mg elemental iron/day (271 subjects) for 56 days. Approximately 45% of subjects treated with conventional ferrous sulfate reported moderate-to-severe gastrointestinal effects, including abdominal discomfort, nausea, vomiting, constipation and diarrhea, compared to approximately 17% of subjects treated with the ferrous sulfate wax-matrix preparation, a statistically significant difference ($p < 0.001$).

The tolerability of ferrous sulfate (50 mg elemental iron/day) and bis-glycino iron II (50 mg elemental iron/day) was compared in a double-blind, crossover trial in 42 women (Coplin et al., 1991). The treatment period for each iron supplement was two weeks. No placebo treatment period was included. The frequency of adverse gastrointestinal effects (abdominal pain, bloating, constipation, diarrhea and nausea) was similar for the two treatments, with 54% and 59% of subjects reporting gastrointestinal symptoms during treatment with bis-glycino iron II and ferrous sulfate, respectively. The difference between treatments was not statistically significant.

Effects of iron therapy on the upper gastrointestinal tract were evaluated in 14 healthy volunteers [13 women, 1 man; mean age 29 years (range: 24-48 years)] who were instructed to ingest 325 mg tablets of ferrous sulfate (119.5 mg elemental iron) three times/day before meals (358.5 mg elemental iron/day) for 2 weeks (Laine et al., 1988). Evaluation consisted of a gastrointestinal symptom survey, qualitative (Hemoccult) and quantitative (HemoQuant; mg mercury/g stool) testing for fecal blood loss, endoscopy of the upper gastrointestinal tract and histological examination of pinch biopsies of the gastric body, antrum and duodenum. Based on actual average ingestion of 2.5 tablets/day (2-week study) and 2.6 tablets/day (1-week study) and a reference human body weight of 70 kg (U.S. EPA, 1987), the estimated doses consumed by the subjects were 4.3 and 4.4 mg iron/kg-day, respectively, in addition to dietary iron. Compared to

baseline measurements in the two weeks prior to treatment, all subjects had significantly increased ($p < 0.05$) dark brown-black stools and symptoms of nausea and vomiting during the treatment period, but not abdominal pain. Hemoglobin levels in stool did not change significantly after iron treatment. Endoscopic examination showed a significant ($p = 0.003$) increase in abnormalities in the stomach, but not duodenum, after therapy. These changes consisted of erythema, small areas of subepithelial hemorrhage and solitary antral erosions in nine, six and two subjects, respectively, and were considered only minimally abnormal. No treatment-related histological changes were observed. Although it was speculated that the changes in the stomach could represent a mild form of iron poisoning, the investigators concluded that the treatment caused mild endoscopic abnormalities of uncertain clinical significance in the stomach. Evidence for iron overload (tissue biopsies or hematologic iron status indices) was not examined. Considering additional dietary exposure, an exposure level of about 4.3 mg/kg-day represents, at worst, a minimal LOAEL.

Adverse developmental effects in humans have not been associated with the ingestion of supplemental iron during pregnancy. As indicated above, NAS (2001) recommended that pregnant women supplement their diets with 27 mg iron/day (0.35 mg/kg-day). McElhatton et al. (1991) reported on 49 women who took an overdose of a simple iron preparation (53%) or iron with folate preparation (47%). In 48 of the women, the amount of iron ingested was known; 28 took > 1.2 g and the remainder took 1.2 g. There were 25 women who received chelation treatment with desferrioxamine (DFO) and 12 who received an emetic. Maternal toxicity, consisting of nausea, vomiting, hematoemesis, abdominal pain and diarrhea, was observed in 35 of the women. Two spontaneous abortions occurred and there were three premature deliveries. One of the spontaneous abortions and the premature deliveries were not related to the iron overdose. It is not known if the other spontaneous abortion occurring at 22 weeks (3 weeks after the overdose) was caused by the iron overdose. No conclusions on the developmental toxicity of iron can be made.

Chronic Exposure

While chronic iron toxicity occurs in people with genetic metabolic disorders resulting in excessive iron absorption or abnormal hemoglobin synthesis, or who receive frequent blood transfusions (Jacobs, 1977; Bothwell et al., 1979), there is a long-standing controversy as to whether a chronic overload due to oral intake is possible in individuals with a normal ability to control iron absorption (Hillman and Finch, 1985). Nevertheless, "the cumulative experience in human subjects suffering from iron overload of various etiologies strongly suggests that iron is noxious to tissues [when]...present in parenchymal cells...for a sufficiently long period of time" (Bothwell et al., 1979).

Looker et al. (1988) made comparisons of dietary iron intake and biochemical indices of iron status based on values taken from the second National Health and Nutrition Examination Survey (NHANES II) data base¹. NHANES II was a probability sample of the noninstitutionalized U.S. population aged 6 months to 74 years, conducted between 1976 and

¹ The latest version of this data base, NHANES III (1984-1988) evaluated 30,000 subjects aged 2 months and above (NAS, 2001). Despite minor differences in the data sets, the conclusions drawn by Looker et al. (1988) based on NHANES II appear to be valid for the NHANES III data.

1980 by the National Center for Health Statistics. These data suggest that normal intake of iron by men 16-74 years old exceeds the DRI, and that iron intake is somewhat lower than the DRI for women younger than 51 years. Concomitant with the study of dietary intake, the NHANES II measured the iron status of these populations. The percent serum transferrin saturation, a measure of the residual capacity of the iron transport system to process potential variations in iron from dietary intake or catabolized body stores, ranged from 24% saturation for pre- and post-menopausal women not using iron supplements to 29% saturation for adult male supplement users. These values are within the normal range (20-40%). The Looker et al. (1988) evaluation of the NHANES II iron status data concerned iron deficiencies, only, and did not address iron overload directly. However, iron overload conditions would likely be evidenced by increased saturation of serum transferrin and increased serum ferritin concentrations, which were also within the normal range. Therefore, the corresponding dietary intakes are presumed to represent chronic NOAELs. Looker et al. (1988) estimated daily iron intakes ranging from 10.0 for elderly women to 18.7 mg/day for young adult men in the study population. These daily intakes correspond to a range of about 0.15 to 0.27 mg/kg-day, depending on assumptions of average body weight. Taking the highest intake level of 18.7 mg/day and a body weight of 70 kg, a NOAEL of 0.27 is established for chronic iron toxicity.

Hemosiderosis (or siderosis) and iron overload are increases in tissue iron or a general increase in iron stores without associated tissue damage (Bothwell et al., 1979; Jacobs, 1977). Hemochromatosis describes massive iron overload (15 g of body iron stores or greater) together with cirrhosis and/or other tissue damage attributable to iron. Although focal deposits of iron may occur in any part of the body where red cells are extravasated, the clinical syndrome of hemochromatosis typically involves damage to the hepatic parenchyma (particularly fibrosis), heart (cardiac dysfunction including failure) and endocrine glands (particularly hypogonadism). Pancreatic iron deposition is common and massive deposits may be associated with fibrosis and diabetes. A number of studies involving chronic oral administration of iron to animals have been designed in an attempt to identify an animal model for hemochromatosis. Most of these studies have been negative (Bothwell et al., 1979; NRC, 1979). Animal studies involving parenteral administration of iron have been generally negative as well, even though parenteral routes bypass the mechanisms that regulate absorption of iron from the gastrointestinal tract.

Chronic iron toxicity has been observed in people with idiopathic hemochromatosis (a genetic metabolic disorder resulting in excessive iron absorption), abnormalities of hemoglobin synthesis (e.g., thalassemia) or various anemic states (e.g., sideroblastic anemia), frequent blood transfusions or a combination of these conditions (Jacobs, 1977; Bothwell et al., 1979). Chronic hemochromatosis has also occurred among the South African Bantu population from an excessive intake of absorbable iron in an alcoholic beverage.

Habitual excessive intake of iron by the Bantus is attributed to consumption of home-brewed Kaffir beer, which was contaminated by iron vessels during brewing (Bothwell and Bradlow, 1960; Bothwell et al., 1964). The beer's high acidity (pH 3-3.5) enhanced iron leaching from the vessels. The iron in the beer is readily assimilable (i.e., ionizable) due to the acidity and presence of iron-complexing ligands such as fructose, and is absorbed to approximately the same degree as ferric chloride. The alcohol content of the beer is also believed to contribute to the bioavailability of the iron (Jacobs, 1977; Finch and Monsen, 1972). Based

primarily on drinking habits and analyses of beer samples, the estimated average dietary iron intake of the Bantu men ranged from 50-100 mg/day from beer alone (Bothwell et al., 1964). Using a reference body weight of 70 kg (U.S. EPA, 1987), this range corresponds to 0.7-1.4 mg/kg-day. Histological examinations of the liver of 147 Bantus (129 male, 18 female) ranging in age from 11-70 years (most were between 20 and 50 years old) that died from acute traumatic causes were performed (Bothwell and Bradlow, 1960). Varying degrees of hepatic siderosis were observed in 89% of the cases; the degree tended to increase with age 40-50 years or less. The siderosis was mild in 59% and severe in 19% of the cases, respectively. There was a close correlation between hepatic iron concentration and portal fibrosis and cirrhosis. Although the overall prevalence was low (15.6% fibrosis and 1.4% cirrhosis), all 11 subjects with the highest iron concentrations (>2.0% dry weight of liver) showed either fibrosis or cirrhosis. Histological examination of the spleen (50 subjects) also showed siderosis and unspecified histological changes. Malnutrition and alcoholism could have played a role in the etiology of the hepatic and splenic siderosis in the Bantus. A NOAEL in the range of 0.7 - 1.4 mg/kg-day is indicated but may be low given the likely higher bioavailability of iron in the beer than for normal dietary exposure. Given the generally poor nutritional health status of this population, the relevance of this study for application to the U.S. population is questionable.

Ethiopia reportedly has the highest per capita iron intake in the world, with an average daily intake of 471 mg iron/day (range 98-1418 mg/day; 1.4-20.3 mg iron/kg-day assuming 70 kg body weight) (Roe, 1966; Hofvander, 1968). Increased stored iron in the liver and adverse health effects have not been observed due to low bioavailability of the iron in Ethiopian food.

A few studies have suggested that high iron intake may be a risk factor for myocardial infarction (Salonen et al., 1992; Lauffer, 1991; Sullivan, 1992). Five other large studies found no association between serum ferritin levels and coronary heart disease (NAS, 2001). Various other measures of iron status (serum transferrin saturation, serum iron concentration and total iron-binding capacity) have been examined for a possible link to cardiovascular disease in prospective cohort studies, but results overall have been characterized as contradictory (Meyers, 1996; NAS, 2001). The NAS (2001) concluded that the available evidence “does not provide convincing support for a causal relationship” between the level of dietary iron intake and the risk for coronary heart disease, although iron cannot be definitively excluded as a risk factor.

Animal Studies

Repeated-dose oral studies in experimental animals found no significant effect of treatment with inorganic iron compounds. No treatment-related adverse changes in clinical signs, body or organ weights, food consumption or histopathology were observed in male Sprague-Dawley rats that had daily dietary intakes of 35, 70 or 140 mg of iron (as FeSO₄ or FeEDTA) per kg for up to 61 days (Appel et al., 2001). In male and female F344 rats that were exposed to drinking water containing 0.25 or 0.5% ferric chloride (FeCl₃ • 6H₂O) for 104 weeks, there were no dose-related effects other than reduced water intake (possibly affected by palatability) and body weight gain (Sato et al., 1992). In the latter study, the iron intakes were 58 or 110 mg/kg-day in males and 65 or 116 mg/kg-day in females.

No treatment-related teratogenic or embryotoxic effects were observed in rats given 2.7 mg iron/kg-day as ferric chloride on gestational days 6-15 (Nolen et al., 1972), or in rats and mice given 24-76 mg iron/kg-day as ferrous sulfate for 6 days during gestation (days unspecified) (Tadokoro et al., 1979). Some embryonic mortality (numbers and species not reported) occurred in the latter study at 240 mg iron/kg-day.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfDs FOR IRON

Iron is an essential element, as such, the RfD must be protective against both toxicity and deficiency. Using the values for dietary intake and iron status indices taken from the second National Health and Nutrition Examination Survey (NHANES II) data base, it is possible to establish a NOAEL for chronic toxicity. Looker et al. (1988) made comparisons of dietary iron intake and biochemical indices of iron status using data from NHANES II. The average intakes of iron ranged from 0.15 to 0.27 mg/kg-day. The serum ferritin levels and percent serum transferrin saturation were within the normal range. Thus, intake levels of 0.15-0.27 mg/kg-day are sufficient to protect against iron deficiency. However, the NHANES II data do not provide information to identify daily dietary iron intakes associated with toxicity. Therefore, daily dietary iron intakes were not considered as the basis for the p-RfD.

Most of the quantitative chronic oral toxicity data for iron have been obtained from studies of the Bantu population of South Africa. These data indicate that intakes in the range of 0.7-1.4 mg iron/kg-day in home-brewed beer are associated with hemosiderosis and liver cirrhosis (Bothwell and Bradlow, 1960; Bothwell et al., 1964). However, confounding factors such as malnutrition and unusually high iron bioavailability due to the high acidity and ethanol in the beer preclude use of these data for risk assessment. Much higher dietary intakes (average 6.7 mg/kg-day) of less soluble forms of iron are tolerated in non-western diets as indicated by studies of populations in Ethiopia. Thus, although toxicity associated with iron overload due to chronic oral intake can be demonstrated qualitatively or even semiquantitatively, assignment of a precise LOAEL for normal individuals consuming western diets is compromised by studies containing confounding factors.

Gastrointestinal toxicity, which is commonly associated with the therapeutic use of iron supplements, was identified as the critical effect for the basis of the provisional subchronic and chronic RfDs. The most frequently reported symptoms include epigastric pain, nausea, vomiting, constipation and diarrhea. Several prospective clinical trials in healthy subjects and iron-deficient patients identify a LOAEL for gastrointestinal toxicity of 50 to 180 mg elemental iron/day; NOAELs were not established (Blot et al., 1981; Brock et al., 1985; Coplin et al., 1991; Frykman et al., 1994; Hallberg et al., 1966; Liguori, 1993). The treatment durations in these studies range from 2 weeks to approximately 3 months. Although no chronic exposure studies reporting gastrointestinal toxicity were identified, clinical experience with iron supplements indicates that gastrointestinal effects are associated with oral iron therapy, regardless of the duration of treatment and that symptom intensity does not change over the course of treatment (Hillman, 2001; Santi and Masters, 2001). This observation suggests that the response is related to the concentration of iron in the intestinal tract and not to the time-integrated dose. Therefore,

gastrointestinal toxicity is considered as the critical effect for both the subchronic and chronic p-RfDs.

The lowest LOAEL of 50 mg elemental iron/day for gastrointestinal toxicity associated with iron supplements was reported in two studies that did not use a placebo-controlled design (Brock et al., 1985; Coplin et al., 1991); therefore, data were not considered suitable for derivation of the p-RfD. The placebo-controlled, cross-over design study by Frykman et al. (1994) reporting a LOAEL of 60 mg/day in Swedish men and women was identified as the critical study. Results of this study show that daily treatment with ferrous fumarate (60 mg elemental iron/day) for one month produced a statistically significant increase in gastrointestinal effects compared to placebo. To determine the LOAEL for total daily iron intake, the LOAEL for daily supplementation with ferrous fumarate of 60 mg elemental iron/day was added to the estimated mean dietary intake for six European countries of 11 mg elemental iron/day (NAS, 2001) for a total daily iron intake of 71 mg elemental iron/day. Based on a reference body weight of 70 kg (U.S. EPA, 1987), the LOAEL for gastrointestinal effects for total daily iron intake is 1 mg elemental iron/kg-day. This LOAEL is considered to be a minimal LOAEL because gastrointestinal effects were characterized by most study participants as minor in severity.

The provisional subchronic and chronic RfD for iron was derived from the LOAEL of 1 mg/kg-day for total daily iron intake for adverse gastrointestinal effects as follows:

$$\begin{aligned}\text{p-RfD (subchronic and chronic)} &= \text{LOAEL} \div \text{UF} \\ &= 1 \text{ mg/kg-day} \div 1.5 \\ &= 0.7 \text{ mg/kg-day}\end{aligned}$$

Dividing the LOAEL of 1 mg/kg-day by an uncertainty factor of 1.5 yields a subchronic and chronic p-RfD of 0.7 mg/kg-day. The uncertainty factor of 1.5 includes the individual uncertainty factors of 1.5 for use of a minimal LOAEL, 1 for sensitive individuals, 1 for less than lifetime exposure, and 1 for an adequate data base. An uncertainty factor of 1.5 was applied to account for extrapolation from a minimal LOAEL to a NOAEL for a non-serious effect. A higher uncertainty factor for use of a minimal LOAEL was not used since the observed gastrointestinal effects are not considered serious and are reversible when exposure is discontinued. Furthermore, gastrointestinal symptoms are not associated with dietary intake of similar levels of iron (NAS, 2001). Because individuals sensitive to gastrointestinal symptoms are considered to be included in the studies investigating effects of therapeutic iron; an uncertainty factor of 1 for sensitive individuals results. An uncertainty factor of 1 was used to account for less than lifetime exposure. Although exposure duration in the Frykman et al. (1994) study was only one month, there is no evidence to suggest that symptoms increase with longer exposure periods. An uncertainty factor of 1 was used to reflect an adequate database in humans, due to the extensive use of therapeutic iron.

Except for individuals with disorders of iron metabolism, little information is available on the long-term systemic toxicity of orally ingested iron. This assessment, therefore, focuses more on what is known to be a safe oral intake of iron for the general human population (i.e., apparently healthy normal individuals). The provisional reference dose is estimated to be an

intake for the general population that is adequately protective from adverse health effects. Further, it is also important to note that individual requirements for, as well as adverse reactions to, iron may be highly variable. Some individuals may, in fact, consume a diet that contributes more than the provisional reference dose, without any cause for concern. In addition, specific population subgroups may have higher nutritional requirements than the provisional RfD would provide. The p-RfD may not be protective of individuals with inherited disorders of iron metabolism or other conditions which affect iron homeostasis.

This assessment is essentially the same as that proposed by Stifelman et al. (2005).

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May 29, 2008

Michael Sivak
U.S. EPA, Region 2

ASSISTANCE REQUESTED: PPRTVs for p-Isopropyltoluene, Pyrene and sec-Butylbenzene (*Onondaga Lake*)

ENCLOSED INFORMATION:

- Attachment 1: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR *p*-ISOPROPYLTOLUENE (CASRN 99-87-6) Derivation of a Chronic Oral RfD**
- Attachment 2: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR *p*-ISOPROPYLTOLUENE (CASRN 99-87-6) Derivation of a Chronic Inhalation RfC**
- Attachment 3: **PROVISIONAL PEER REVIEWED TOXICITY VALUE FOR *p*-ISOPROPYLTOLUENE (CASRN 99-87-6) Derivation of a Carcinogenicity Assessment**
- Attachment 4: **PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR PYRENE (CASRN 129-00-0)**

Attachment 5: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR n-BUTYLBENZENE (CASRN 104-51-
8), sec-BUTYLBENZENE (CASRN 135-98-8) AND tert-
BUTYLBENZENE (CASRN 98-06-6) Derivation of
Subchronic and Chronic Oral RfDs**

Attachment 6: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR n-BUTYLBENZENE (CASRN 104-51-
8), sec-BUTYLBENZENE (CASRN 135-98-8) AND tert-
BUTYLBENZENE (CASRN 98-06-6) Derivation of
Subchronic and Chronic Inhalation RfCs**

Attachment 7: **PROVISIONAL PEER REVIEWED TOXICITY
VALUES FOR n-BUTYLBENZENE (CASRN 104-51-
8), sec-BUTYLBENZENE (CASRN 135-98-8) AND tert-
BUTYLBENZENE (CASRN 98-06-6) Derivation of a
Carcinogenicity Assessment**

BE ADVISED: Unless specifically indicated to have been peer reviewed, it is to be noted that the attached Provisional Toxicity Value Paper(s) have not been through the U.S. EPA's formal review process; therefore, they do not represent a U.S. EPA verified assessment.

If you have any questions regarding this transmission, please contact the STSC at (513) 569-7300.

Attachments (7)

cc: STSC Files

11-23-2004

Provisional Peer Reviewed Toxicity Values for

p-Isopropyltoluene
(CASRN 99-87-6)

Derivation of a Chronic Oral RfD

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
p-ISOPROPYLTOLUENE (CASRN 99-87-6)
Derivation of a Chronic Oral RfD**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A subchronic or chronic RfD for *p*-isopropyltoluene (also known as *p*-cymene or *p*-methyl isopropyl benzene) is not available on IRIS (U.S. EPA, 2003), the HEAST (U.S. EPA, 1997), or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2002). One relevant document, the Drinking Water Health Advisory for *p*-Cymene (U.S. EPA, 1987), was located in the CARA list (U.S. EPA, 1991, 1994), but no RfD was derived due to insufficient data. ATSDR (2003), NTP (2003), IARC (2003), and WHO (2003) have not produced documents for this chemical. Literature searches of the following databases were conducted for the period between 1965 through June 2003 to locate relevant studies on *p*-isopropyltoluene: TOXLINE (supplemented with BIOSIS and NTIS updates), CANCERLIT, MEDLINE, CCRIS, GENETOX, HSDB, DART/ETICBACK, EMIC/EMICBACK, RTECS and TSCATS.

Additional literature searches from June 2003 through January 2004 were conducted by NCEA-Cincinnati using MEDLINE, TOXLINE, Chemical and Biological Abstracts databases.

p-Isopropyltoluene is used as an intermediate in the manufacture of *p*-cresol and thymol (HSDB, 2003). It also serves as a component in commercial terpene solvent mixtures and as a component of paint thinners, lacquers, and varnishes. It is found to occur naturally in many arboreous plants and as a constituent of many essential oils, including oils of eucalyptus, lemon, sage, thyme, coriander, star anise, and cinnamon (Browning, 1965; Opdyke, 1974; HSDB, 2003). The FDA (2003) has approved the use of *p*-isopropyltoluene as a synthetic flavoring substance and food additive.

REVIEW OF PERTINENT DATA

Human Studies

No data regarding the toxicity of *p*-isopropyltoluene to humans following chronic or subchronic oral exposure were located. Limited information is available on the acute oral toxicity of *p*-isopropyltoluene in humans. Reports from the early literature indicate that headache, nausea and vomiting occurred in humans following ingestion of *p*-isopropyltoluene, but information on ingested dose levels was not available (Browning, 1965).

p-Isopropyltoluene appears to be a primary skin irritant. Cases of irritation of the skin and mucous membranes have been reported in workers exposed to *p*-isopropyltoluene (Browning, 1965; Carlson, 1946). Contact with the undiluted liquid has been reported to produce dermal erythema, dryness and defatting in humans (Gerarde, 1960). However, 4% *p*-isopropyltoluene in (petroleum jelly) produced no dermal irritation or sensitization in a 48-hour closed-patch maximization test with 25 human volunteers (Kligman, 1972) and no dermal irritation following daily occluded application in diethylphthalate (petroleum jelly) on the backs of 10 human subjects for 10 days (Kligman and Wooding, 1967; Kligman, 1973).

Animal Studies

No data regarding the toxicity of *p*-isopropyltoluene to animals following chronic or subchronic oral exposure were located. However, several brief reports of the effects following acute exposures to *p*-isopropyltoluene were located.

An oral LD₅₀ value of 4.75 g/kg (3.72-6.06 g/kg, 95% C.I.) was reported for Osborne-Mendel rats administered single doses of undiluted chemical via intubation and observed for 14 days (Jenner et al., 1964). Clinical signs observed included CNS depression soon after dosing, coma, bloody lacrimation, and diarrhea. The exposed animals appeared irritable and scrawny

through the observation period; the time to death ranged from 4 hours to 12 days. Additional study details were not provided. An oral LD₅₀ value of 3 g/kg was reported for white rats administered single doses of *p*-isopropyltoluene via gavage (Du Pont & Co., 1949). Post mortem findings revealed gross and microscopic evidence of gastritis and liver damage. Smyth et al. (1951) reported an oral LD₅₀ value of 2.46 g/kg in rats administered single doses of *p*-isopropyltoluene (additional details were not reported). No gross or micropathological findings were reported for six rats given oral doses of 510 mg/kg-day of *p*-isopropyltoluene, 5 days a week, for a total of 10 treatments (Du Pont & Co., 1949). All six rats survived treatment and were sacrificed 11 days after the last treatment. No other effects or study details were provided. A study from the very early literature reported that dogs tolerated daily oral doses of 2 g of *p*-isopropyltoluene, with diarrhea as the only adverse effect (Ziegler, 1873). No other study details were provided.

Intraperitoneal administration of 2.162 g/kg of *p*-isopropyltoluene reportedly induced lethality in the guinea pig (Chassevant and Garnier, 1903). Subcutaneous injection of *p*-isopropyltoluene in rabbits produced hematological changes similar to those produced by xylene that resulted in an increase in immature white cells similar in appearance to human myeloid leukemic cells (Miyamoto, 1938; Woronow, 1929). An acute dermal LD₅₀ of >5 g/kg was reported for *p*-isopropyltoluene in rabbits (Moreno, 1973). Application of undiluted *p*-isopropyltoluene to intact or abraded skin of rabbits for 24 hours under occlusion was moderately irritating (Moreno, 1973).

No developmental or reproductive toxicity studies of *p*-isopropyltoluene by any route of exposure were located.

Other Studies

p-Isopropyltoluene appears to be well absorbed by the oral route. Eighty and 71% of a single 100 mg/kg gavage dose of *p*-isopropyltoluene in propylene glycol was recovered as urinary metabolites within 48 hours in 3 rats and 3 guinea pigs, respectively (Walde et al., 1983). The remainder reportedly consisted of metabolites excreted in the feces and unextracted urinary material, indicating that at least 70-80% of the administered dose was absorbed.

Studies that have examined the metabolism of *p*-isopropylbenzene in rats and guinea pigs have presented a unified picture of a compound that is subject to considerable metabolic rearrangement. Thus, Walde et al. (1983) identified 18 total urinary metabolites in the oral- and inhalation-exposed rats and guinea pigs, and found that their excretion was nearly complete within 48 hours which amounted to 60-80% of the administered dose. The principal metabolites resulting from oxidation of the isopropyl and/or methyl group in the Walde et al. (1983) study included *p*-isopropyl benzoic acid (cumic acid, 19% of administered dose), 2-*p*-carboxyphenylpropan-1-ol (11%) and 2-*p*-carboxyphenylpropionic acid (16%) in oral-exposed

rats; *p*-isopropylbenzoyl glycine (31%) and 2-*p*-tolylpropan-2-ol (14%) in oral-exposed guinea pigs; 2-*p*-carboxyphenylpropionic acid (15%) in inhalation-exposed rats; and *p*-isopropylbenzoyl glycine (31%) and 2-*p*-tolylpropionic acid (15%) in inhalation-exposed guinea pigs. The remaining metabolites identified by Walde et al. (1983) ranged from trace amounts to 6-9% of the administered dose. In general, both the methyl and isopropyl side groups have been shown to be extensively oxidized in all species tested (Bakke and Scheline, 1970; Ishida et al., 1981; Walde et al., 1983; Matsumoto et al., 1992). The numerous monohydric alcohols, diols, mono- and dicarboxylic acids and hydroxyacids identified in the urine established that oxidation to alcohol with and without further oxidation to the corresponding acid occurred at all possible aliphatic sites (i.e., the three isopropyl carbons and the methyl carbon).

Comparative studies of the *in vivo* metabolism of *p*-isopropyltoluene in Eucalyptus - eating marsupials (the brushtail possum and the koala) and the rat (Boyle et al., 1999, 2000; Southwell et al., 1980) identified a species-specific pattern with a higher level of oxidation of *p*-isopropyltoluene in the marsupials compared with the rat, and a higher level of conjugation of metabolites in the rats compared to the marsupials. *In vitro* studies of *p*-isopropyltoluene metabolism by liver microsomes of the possum, koala, and rat showed that the major metabolite in each species was cuminyl alcohol and that the possum and koala microsomes oxidized cuminyl alcohol to cumic acid (Pass et al., 2002). The rank order of the liver microsomes to metabolize *p*-isopropyltoluene, measured by intrinsic clearance (V_{\max}/K_m), was terpene-pretreated possum > possum with standard diet > koala with standard diet > rat with standard diet.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC ORAL RfDs FOR *p*-ISOPROPYLTOLUENE

The available oral toxicity data for *p*-isopropyltoluene indicate that the compound has a low order of acute lethality (rat LD₅₀ >2000-4000 mg/kg), with symptoms of CNS depression and gastrointestinal tract irritation at lower dose levels. These findings are supported by limited acute dermal exposure data that show local irritative effects of *p*-isopropyltoluene, and are consistent with the known acute effects of alkyl benzenes in general. No adequate information is available on the subchronic or chronic toxicity of *p*-isopropyltoluene, thereby precluding derivation of a subchronic or chronic p-RfD from the oral data or from inhalation data by route-to-route extrapolation.

There are no data indicating that *p*-isopropyltoluene is metabolized to any compounds that have toxicity data sufficient for risk assessment or into the same metabolites as other alkyl benzenes that have adequate toxicity data. Therefore, in summary, derivation of a p-RfD for *p*-isopropyltoluene is precluded by insufficient toxicity data on either the subject compound or on any of its projected primary metabolites.

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11-23-2004

Provisional Peer Reviewed Toxicity Values for

p-Isopropyltoluene
(CASRN 99-87-6)

Derivation of a Chronic Inhalation RfC

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
p-ISOPROPYLTOLUENE (CASRN 99-87-6)
Derivation of a Chronic Inhalation RfC**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A subchronic or chronic RfC for *p*-isopropyltoluene (also known as *p*-cymene or *p*-methyl isopropyl benzene) is not available on IRIS (U.S. EPA, 2003) or in the HEAST (U.S. EPA, 1997). One relevant document, the Drinking Water Health Advisory for *p*-Cymene (U.S. EPA, 1987), was located in the CARA list (U.S. EPA, 1991, 1994). ATSDR (2003), NTP (2003), IARC (2003), and WHO (2003) have not produced documents for this chemical. ACGIH (2003), NIOSH (2003), and OSHA (2003) have not recommended occupational exposure limits for *p*-isopropyltoluene. Literature searches of the following databases were conducted from 1965 through June 2003 to locate relevant studies on *p*-isopropyltoluene: TOXLINE (supplemented with BIOSIS and NTIS updates), CANCERLIT, MEDLINE, CCRIS, GENETOX, HSDB, DART/ETICBACK, EMIC/EMICBACK, RTECS and TSCATS. Additional literature searches

from June 2003 through January 2004 were conducted by NCEA-Cincinnati using MEDLINE, TOXLINE, Chemical and Biological Abstracts databases.

p-Isopropyltoluene is used as an intermediate in the manufacture of *p*-cresol and thymol (HSDB, 2003). It also serves as a component in commercial terpene solvent mixtures and as a component of paint thinners, lacquers, and varnishes. It is found to occur naturally in many arboreal plants and as a constituent of many essential oils, including oils of eucalyptus, lemon, sage, thyme, coriander, star anise, and cinnamon (Browning, 1965; Opdyke, 1974; HSDB, 2003). The FDA (2003) has approved the use of *p*-isopropyltoluene as a synthetic flavoring substance and food additive.

REVIEW OF THE PERTINENT DATA

Human Studies

Few data are available on the inhalation toxicity of *p*-isopropyltoluene in humans. The primary effects of *p*-isopropyltoluene in industrial environments were skin and mucous membrane irritation; however, information on exposure levels was unavailable (Browning, 1965). Systemic effects, including ecchymosis, severe anemia, leucopenia, thrombocytopenia and histopathologic alterations in bone marrow, were reported in a man occupationally exposed to *p*-isopropyltoluene in the paper pulp industry (Carlson, 1946). These changes were possibly indicative of aplastic anemia, but the evidence for a direct association with exposure to *p*-isopropyltoluene was inconclusive (Browning, 1965). No other cases of a "benzene-like" action on the bone marrow following exposure to *p*-isopropyltoluene were reported by Browning (1965) or located in the available literature.

Animal Studies

No toxicity studies of animals chronically exposed by inhalation to *p*-isopropyltoluene were located. Available inhalation toxicity studies are restricted to a study of dogs repeatedly exposed by inhalation and two acute inhalation studies of rats in which neurological endpoints were examined.

In repeated-exposure studies, four dogs (of unspecified breed) were exposed to vapors of *p*-isopropyltoluene at a reported concentration range of 50 to 110 ppm (average 75 ppm) for 6 hours/day, 5 days/week, for one month (Du Pont & Co., 1949). One of the dogs was withdrawn from the experiment after the 12th exposure period due to illness not related to compound exposure. After one month of exposure, the surviving 3 dogs were exposed to concentrations ranging from 75 to 155 ppm (average 100 ppm) for 6 weeks, after which the animals were sacrificed for pathological examination. Another group of four dogs was subsequently exposed

by the same protocol for a total period of 18 weeks to average concentrations of *p*-isopropyltoluene of 50 ppm for the first 40 exposures, 75 ppm for the next 24 exposures, and 160 ppm for the last 28 exposures, after which dogs were sacrificed for pathological examination. The report of the study only referred to the pathological examinations as “gross” and “micropathology,” and did not further specify tissues examined or techniques employed. Blood pressure, respiration rate, and body weight were measured at unspecified intervals during exposure. Blood and urine samples were collected at unspecified intervals during exposure; hematologic and chemical parameters that were measured were not specified in the available report, other than red blood cell counts and hemoglobin concentrations. Results were reported only in a summary fashion (the magnitude of exposure-related changes was not reported). Body weights were reportedly unaffected by exposure. Blood pressure depression (compared with pre-exposure patterns) was observed in both groups of dogs during exposure, with some inter-dog variance in whether the reduction was in systolic, diastolic, or pulse pressure. Respiration rates were increased (compared with pre-exposure values) during exposure in the dogs exposed during the first experiment, but not in those exposed in the second experiment. Red blood cell counts during the exposure period were reported as normal in both groups of dogs, but hemoglobin concentrations were reported as “decreased somewhat” in the first dog experiment, but not in the second dog experiment. “No gross or micropathology” attributable to exposure was found in the three dogs that survived the first experiment or in 3 of the 4 dogs that were exposed in the second experiment. The fourth dog in the second experiment had a “filarial infestation” associated with fibrosis and slight congestion of the lungs and zones of fibrosis in the kidney.

Lam et al. (1996) exposed groups of 7, 11, or 12 male Long-Evans rats to *p*-isopropyltoluene vapor at 0, 50, or 250 ppm (0, 275 or 1370 mg/m³), respectively, 6 hours/day, 5 days/week for 4 weeks, followed by an 8-week recovery period prior to sacrifice. However, the authors largely restricted their observations to the neurochemistry of synaptosomes isolated from whole brain (minus cerebellum), measuring specific amounts of neurotransmitters, such as noradrenaline, dopamine, and 5-hydroxytryptamine, and specific activities of enzymes, such as acetylcholinesterase, butyrylcholinesterase, and lactate dehydrogenase, as a model for *in situ* conditions at the level of the presynaptic nerve terminal. There were no overt clinical signs of toxicity in any of the animals involved in the experiment and no differences in mean body weight between the groups. Similarly, there were no treatment-induced effects on brain weight or protein concentration in whole brain minus cerebellum, cerebellum alone, or whole brain. Though the concentrations of neurotransmitters/g wet weight were the same for each brain fraction irrespective of treatment, the relative yield of synaptosomal protein/unit wet weight of brain was reduced dose-dependently and statistically significant as the concentration of *p*-isopropyltoluene increased. Similarly, potential dose-dependent fluctuations in synaptosomal enzyme activities were observed, though a constant ratio relative to the activity of LDH was maintained across all dose groups. The relative amounts of synaptosomal neurotransmitters compared to enzyme units of LDH in synaptosomal preparations appeared to be unaffected by treatment, although increases in noradrenaline and dopamine were observed relative to

synaptosomal protein. Fluctuations in the relative and absolute amounts of 5-hydroxytryptamine were also observed in relation to treatment, although, as with all these results, their relationship to the neurotoxicity of the compound is unclear. The authors interpreted their results in terms of *p*-isopropyltoluene having the capacity to reduce the density and total number of synapses *in situ*, with functional compensation by concurrent increases in the release of noradrenaline and dopamine from noradrenergic and dopaminergic neurons. However, the authors were unable to link their observed changes to manifest neurotoxicity in the whole animal, beyond the speculation that the changes in synaptosomal neurochemistry might be a reflection of “first stage affective syndrome,” a condition of uncertain pathophysiology, but with possible clinical consequences marked by depression, irritability, and a general loss of interest in daily activities.

An earlier neurotoxicity evaluation was performed on male Long-Evans rats that were exposed acutely to high concentrations of *p*-isopropyltoluene vapor (Furnas and Hine, 1958). In an initial experiment, a pair of rats were exposed to 10,000 ppm for 45 minutes, i.e., until they became apneic. Clinical signs preceding the apnea were reported to be similar to those of benzene (e.g., dyspnea, ataxia, twitching and profuse salivation), but more rapid. Another experiment involved 8 rats that were successively exposed as a group to 5000-10,000 ppm of *p*-isopropyltoluene (concentrations not otherwise specified) for 50, 45, 20 and 30 minutes. The rats were removed from the exposure chamber when they showed convulsions or respiratory arrest and returned for another exposure as soon as they recovered (duration between exposures not reported). Exposure was terminated after several deaths occurred (i.e., after 4 exposures), and survivors were sacrificed 24 hours later for gross pathologic observations and histological examination of brain, spinal cord and sciatic nerve. The exposed rats showed clinical signs of respiratory irritation, CNS depression and quivering or twitching, but no convulsions, and 4 of the 8 died. No gross lesions or CNS histopathologic changes were found other than gross changes to the respiratory tract attributable to local irritation.

Subcutaneous injection of *p*-isopropyltoluene in rabbits produced hematological changes similar to those produced by xylene that resulted in an increase in immature white cells similar in appearance to human myeloid leukemic cells (Miyamoto, 1938; Woronow, 1929). The lethal dose via intraperitoneal administration is 2.162 g/kg in the guinea pig (Chassevant and Garnier, 1903).

No developmental or reproductive studies by any route of exposure to *p*-isopropyltoluene were located.

Other Studies

p-Isopropyltoluene appears to be readily absorbed following inhalation. For example, at least 70% and 60% of the dose inhaled in 24 hours (reportedly 100 mg/kg) was recovered as

urinary metabolites within 48 hours in two rats and two guinea pigs, respectively (Walde et al., 1983).

Studies that have examined the metabolism of *p*-isopropylbenzene in rats and guinea pigs have presented a unified picture of a compound that is subject to considerable metabolic rearrangement. Thus, Walde et al. (1983) identified 18 total urinary metabolites in the oral- and inhalation-exposed rats and guinea pigs, and found that their excretion was nearly complete within 48 hours and amounted to 60-80% of the administered dose. The principal metabolites resulting from oxidation of the isopropyl and/or methyl group in the Walde et al. (1983) study included *p*-isopropyl benzoic acid (cumic acid, 19% of administered dose), 2-*p*-carboxyphenylpropan-1-ol (11%) and 2-*p*-carboxyphenylpropionic acid (16%) in oral-exposed rats; *p*-isopropylbenzoyl glycine (31%) and 2-*p*-tolylpropan-2-ol (14%) in oral-exposed guinea pigs; 2-*p*-carboxyphenylpropionic acid (15%) in inhalation-exposed rats; and *p*-isopropylbenzoyl glycine (31%) and 2-*p*-tolylpropionic acid (15%) in inhalation-exposed guinea pigs. The remaining metabolites identified by Walde et al. (1983) ranged from trace amounts to 6-9% of the administered dose. In general, both the methyl and isopropyl side groups have been shown to be extensively oxidized in all species tested (Walde et al., 1983; Bakke and Scheline, 1970; Ishida et al., 1981; Matsumoto et al., 1992). The numerous monohydric alcohols, diols, mono- and dicarboxylic acids and hydroxyacids identified in the urine established that oxidation to alcohol with and without further oxidation to the corresponding acid occurred at all possible aliphatic sites (i.e., the three isopropyl carbons and the methyl carbon).

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfCs FOR *p*-ISOPROPYLTOLUENE

The available inhalation toxicity data for *p*-isopropyltoluene indicate that acute exposure causes CNS depression and mucous membrane irritation. These findings are consistent with known acute effects of alkyl benzenes in general. However, there are no studies of the chronic-duration toxicity of *p*-isopropyltoluene in humans or animals, and there are only a few studies examining toxic endpoints in animals following repeated inhalation exposure. For example, the study by Lam et al. (1996) exposed Long-Evans rats to *p*-isopropyltoluene for a longer period of time than that of a typical acute dosing regimen (4 weeks with an 8-week recovery period). However, this remained less than a typical subchronic dosing regimen ($\geq 10\%$ of the average life span of the animals being tested). Furthermore, it remains unclear whether the *ex situ* observations in synaptosomes reported by these workers represent the onset of neurotoxicological consequences of exposure to the compound (Lam et al., 1996). This points to the difficulty of assigning a NOAEL or LOAEL for neurotoxicity to either of the dose levels employed in the subject study, irrespective of its duration. A report of toxicity studies of dogs exposed for up to 18 weeks (6 hours/day, 5 days/week) in 1948 to varying concentrations of *p*-isopropyltoluene between 50 and 160 ppm specified that exposure was associated with small

decreases in hemoglobin levels, but not with any other marked pathological changes (Du Pont & Co., 1949). However, reporting of methodological details and results was not sufficient to allow an independent evaluation of the study. Because of this deficiency, as well as the small number of animals studied and the lack of a control group, the study does not provide a suitable basis for deriving a p-RfC. In summary, no adequate information is available on the subchronic or chronic toxicity of *p*-isopropyltoluene associated with any route of exposure. This precludes the derivation of a p-RfC for the compound from inhalation data or from oral data by route-to-route extrapolation.

There are no data indicating that *p*-isopropyltoluene is metabolized to any compounds that have toxicity data sufficient for risk assessment or to the same metabolites as other alkyl benzenes that have adequate toxicity data. For example, metabolism that removes the methyl group or part or all of the isopropyl group from *p*-isopropyltoluene does not appear to occur under realistic exposure conditions (e.g., in species not adapted to diets high in *p*-isopropyltoluene), thereby preventing the use of adequately studied mono- or bifunctional alkyl benzene compounds, such as cumene (isopropylbenzene), *p*-xylene and toluene, which already have toxicity values, as surrogates. Other bifunctional alkyl benzenes, particularly compounds such as *p*-ethyltoluene and *p*-*tert*-butyltoluene, appear to have very limited toxicity data and have not been assessed by U.S. EPA (1997, 2003). In summary, derivation of a p-RfC for *p*-isopropyltoluene is precluded by insufficient toxicity data, as well as metabolism data that provide no basis for using a surrogate alkyl benzene for derivation of a toxicity value for the subject compound.

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11-23-2004

Provisional Peer Reviewed Toxicity Values for

p-Isopropyltoluene
(CASRN 99-87-6)

Derivation of a Carcinogenicity Assessment

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUE FOR
p-ISOPROPYLTOLUENE (CASRN 99-87-6)
Derivation of a Carcinogenicity Assessment**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A carcinogenicity assessment for *p*-isopropyltoluene (also known as *p*-cymene or *p*-methyl isopropyl benzene) is not available on IRIS (U.S. EPA, 2003), the HEAST (U.S. EPA, 1997), or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2002). One relevant document, the Drinking Water Health Advisory for *p*-Cymene (U.S. EPA, 1987), was located in the CARA list (U.S. EPA, 1991, 1994). ATSDR (2003), NTP (2003), IARC (2003), and WHO (2003) have not produced documents for this chemical. Literature searches of the following databases were conducted from 1965 through June 2003 in order to locate relevant studies: TOXLINE (supplemented with BIOSIS and NTIS updates), CANCERLIT, MEDLINE, CCRIS, GENETOX, HSDB, DART/ETICBACK, EMIC/EMICBACK, RTECS and TSCATS. Additional literature searches from June 2003 through January 2004 were conducted by NCEA-Cincinnati using MEDLINE, TOXLINE, Chemical and Biological Abstracts databases.

p-Isopropyltoluene is used as an intermediate in the manufacture of *p*-cresol and thymol (HSDB, 2003). It also serves as a component in commercial terpene solvent mixtures and as a component of paint thinners, lacquers, and varnishes. It is found to occur naturally in many arboreous plants and as a constituent of many essential oils, including oils of eucalyptus, lemon, sage, thyme, coriander, star anise, and cinnamon (Browning, 1965; Opdyke, 1974; HSDB, 2003). The FDA (2003) has approved the use of *p*-isopropyltoluene as a synthetic flavoring substance and food additive.

REVIEW OF THE PERTINENT DATA

Human Studies

No data regarding the possible carcinogenicity of *p*-isopropyltoluene in humans were located.

Animal Studies

No reports of animal studies examining the carcinogenicity of *p*-isopropyltoluene by any route of exposure were located.

Other Studies

p-Isopropyltoluene was not mutagenic in *Salmonella typhimurium* strains TA98 or TA100 with metabolic activation in assays of the chemical or fractions of urine collected from rats following administration of *p*-isopropyltoluene via gavage (Rockwell and Raw, 1979). No increased frequencies of streptomycin-independent mutant colonies were found in Sd-4-73 *Escherichia coli* exposed to *p*-isopropyltoluene using the paper disk method (Iyer and Syzbalski, 1958; Syzbalski, 1958).

The metabolism of *p*-isopropyltoluene has been investigated in several studies, including oral studies in rats (Walde et al., 1983; Bakke and Scheline, 1970), guinea pigs (Walde et al., 1983) and rabbits (Ishida et al., 1981; Matsumoto et al., 1992), and inhalation studies in rats and guinea pigs (Walde et al., 1983). In general, the results suggest that the compound is readily absorbed and rapidly metabolized to a range of intermediates. For example, the compound's methyl and isopropyl side groups appeared to be extensively oxidized in all species tested. The numerous monohydric alcohols, diols, mono- and dicarboxylic acids and hydroxyacids identified in the urine established that oxidation to alcohol with and without further oxidation to the corresponding acid occurred at all possible aliphatic sites (i.e., the three isopropyl carbons and the methyl carbon).

PROVISIONAL WEIGHT-OF-EVIDENCE CLASSIFICATION

No studies examining the carcinogenic potential of *p*-isopropyltoluene in humans or animals were located. Genotoxicity data are limited to two negative mutagenicity assays in bacteria. The available data are insufficient to assess carcinogenic potential in animals or humans as specified by the proposed U.S. EPA (1999) Guidelines for Carcinogen Risk Assessment.

QUANTITATIVE ESTIMATES OF CARCINOGENIC RISK

Derivation of quantitative estimates of cancer risk for *p*-isopropyltoluene is precluded by the lack of data to assess carcinogenicity associated with *p*-isopropyltoluene exposure.

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9-5-2007

Provisional Peer Reviewed Toxicity Values for
Pyrene
(CASRN 129-00-0)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose

PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR PYRENE (CASRN 129-00-0)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
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3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

IRIS (U.S. EPA, 1997a) provides a chronic RfD of 3E-2 mg/kg-day developed from a NOAEL of 75 mg/kg-day with a combined uncertainty factor of 3000 (10 each for intra- and interspecies variability, 10 for the use of a subchronic study for chronic RfD derivation, and an additional 3 to account for the lack of both toxicity studies in a second species and developmental/reproductive studies) using a 13 week gavage study in mice, conducted by Toxicity Research Laboratories, Muskegon, MI (U.S. EPA, 1989) for the Office of Solid Waste, Washington, DC. The critical effects were renal tubular pathology and decreased kidney weights. IRIS (U.S. EPA, 2007) did not develop an RfC. IRIS (U.S. EPA, 2007) provided a classification of D, not classifiable as to human carcinogenicity based on no human data and inadequate data from animal experiments. A subchronic RfD is currently listed on HEAST of 3E-1 mg/kg-day which will be removed when this PPRTV is activated (U.S. EPA, 1997b). It was based on the same study as this PPRTV.

A cancer classification for pyrene of Group D is listed in the Drinking water Standard and Health Advisory lists (U.S. EPA, 2000) based on an assessment of pyrene from the Drinking Water Criteria Document for polycyclic aromatic hydrocarbons (U.S. EPA, 1990). The CARA lists (U.S. EPA, 1991, 1994) report a Health Effects Assessment (U.S. EPA 1984) and a Health and Environmental Effects Profile (HEEP) U.S. EPA 1987) for pyrene. ATSDR (2001) has not published a toxicological profile for pyrene, but it is included in the profile for polycyclic aromatic hydrocarbons (ATSDR, 1995). IARC has assigned pyrene to Group 3, not classifiable as to its carcinogenicity to humans, based on no human data and limited animal data (IARC, 1983, 1987). A multimedia document for polycyclic aromatic hydrocarbons (U.S. EPA, 1992) and the NTP status reports (NTP, 2001) were also searched to identify relevant data. Literature searches for all exposure routes and effects were conducted from 1989 to December 2000 and updated to 2007. The databases searched were: TOXLINE, TSCATS, CANCERLIT,

MEDLINE, GENETOX, HSDB, EMIC/EMIC/EMICBACK, DART/ETICBACK, CCRIS AND RTECS.

This document has passed the STSC quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

REVIEW OF THE PERTINENT LITERATURE

Human Studies

No human studies were located regarding exposure of humans to pyrene.

Animal Studies

A U.S. EPA (1989) study conducted by Toxicity Research Laboratories, Muskegon MI for the Office of Solid Waste, Washington DC was the basis of IRIS's chronic RfD of $3\text{E-}2$ mg/kg-day. Male and female CD-1 mice (20/sex/group) were gavaged with 0, 75, 125, or 250 mg/kg/day pyrene in corn oil for 13 weeks. The toxicological parameters examined in this study included body weight changes, food consumption, mortality, clinical pathological evaluations of major organs and tissues, and hematology and serum chemistry. Nephropathy, characterized by the presence of multiple foci of renal tubular regeneration, often accompanied by interstitial lymphocytic infiltrates and/or foci of interstitial fibrosis, was present in 4, 1, 1, and 9 male mice in the control, low-, medium-, and high-dose groups, respectively. Similar lesions were seen in 2, 3, 7, and 10 female mice in the 0, 75, 125, and 250 mg/kg treatment groups. The kidney lesions were described as minimal or mild in all dose groups. Relative and absolute kidney weights were reduced in the two higher dosage groups. Based on the results of this study, the low dose (75 mg/kg/day) was considered the NOAEL and 125 mg/kg/day the LOAEL for nephropathy and decreased kidney weights. The IRIS RfD of $3\text{E-}2$ mg/kg-day was calculated using a composite uncertainty factor of 3000, including 10 each for intra- and interspecies variability, and an additional 3 to account for the lack of both toxicity studies in a second species and developmental/reproductive studies and 10 for extrapolation from subchronic to chronic.

White and White (1939) fed six male rats (unspecified strain) a diet containing 2000 mg pyrene/kg for 40 days. The average reported food intake for two animals was 6.1 g/day, and the average body weight for these two animals was 94.3 g. A decrease in body weight gain was observed in two animals. The authors stated that this body weight gain was representative of the whole group; although there was no change in food intake. White and White (1939) also observed enlarged livers and increased hepatic lipid content in animals treated with pyrene, benzo(a)pyrene or methylcholanthrene in the diet; however, incidence data were not reported and it is unclear whether this effect occurred in the pyrene treated rats. Interpretation of this study is further complicated by the lack of experimental controls and statistical analysis, small sample size, and incomplete reporting of histopathology results.

No other useful studies are available that examine only pyrene exposure

Other studies

Pyrene has been assayed for genotoxicity in a number of tests with both positive and negative results. These have been extensively reviewed by EPA (U.S. EPA, 1984, 1987, 2000, 2001) and only those studies published since the most recent EPA review was performed are included in the following text.

In vitro genotoxicity tests of pyrene in prokaryotic systems have produced mixed results. The consensus conclusion on the WHO international collaborative study (which involved 20 bacterial test sets) was that protocol or evaluation criteria were critical factors in individual test verdicts (WHO, 1990). Pyrene has been shown to bind to DNA (Chen, 1983) and to form DNA adducts (Segerback and Vodicka, 1993), but was not mutagenic in DNA damage assays in *Escherichia coli* and *Bacillus subtilis* (Hellmer and Bolcsfold, 1992; Kranendonk et al., 1994, 1996; Mersch-Sundermann et al., 1992, 1993; Rossman et al., 1991). Both positive (Johnson, 1992) and negative (Rexroat et al., 1995; Rusina et al., 1992; Van der Lelie et al., 1997) results have been reported in bacterial gene mutation tests. Pyrene induced increased incidence of mitotic gene conversion but not other genetic endpoints in yeast (deSerres et al., 1981).

Most *in vitro* tests in mammalian cells have given negative results. Pyrene gave mixed results in tests of unscheduled DNA synthesis (Heil and Reifferscheid, 1992; Selden et al., 1994) and was mostly negative in tests for sister chromatid exchange and negative for chromosome aberrations (Darroudi and Natarajan, 1993; Natarajan and Darroudi, 1991). Pyrene was mutagenic in the L5178Y mouse lymphoma gene mutation assay when metabolically activated (Oberly et al., 1993), but was not mutagenic in metabolically competent human lymphoblastoid cells (Busby et al., 1994; Durant et al., 1996) and did not induce micronucleus formation in a variety of mammalian cell types (Crofton-Sleigh et al., 1993; Fritzenschaf et al., 1993; Muller-Tegethoff et al., 1995; Natarajan and Darroudi, 1991; Neslany and Marzin, 1999). Results of mammalian cell transformation assays have also been negative (U.S. EPA, 2000).

In vivo genotoxicity tests of pyrene have also produced mostly negative results. Pyrene produced no increase or only a slight increase in sex-linked recessive lethals in *Drosophila* and was negative in the *Drosophila* eye mosaic assay (Fujikawa et al., 1993; Vogel and Nivard, 1993). Application of pyrene to the skin of hairless mice produced no increase in micronucleus induction in keratinocytes (He and Baker, 1991). Pyrene was positive in the newt micronucleus test (Fernandez et al., 1989).

DERIVATION OF A PROVISIONAL SUBCHRONIC OR CHRONIC RfD FOR PYRENE

A U.S. EPA (1989) study conducted by Toxicity Research Laboratories, Muskegon MI for the Office of Solid Waste, Washington DC was utilized by IRIS for development of a chronic RfD. This study was selected for development of a provisional subchronic RfD. Based on the results of this study, the low dose (75 mg/kg/day) was considered the NOAEL and 125 mg/kg/day the LOAEL for nephropathy and decreased kidney weights.

A composite uncertainty factor of 300 was applied to the NOAEL of 75 mg/kg-day; 10 each for intra- and interspecies variability, and an additional 3 to account for the lack of both toxicity studies in a second species and developmental/reproductive studies providing a **subchronic RfD of 0.25 mg/kg-day or 3E-1 mg/kg-day**.

NOAEL/ Uncertainty Factors = $75/300 = 0.25$ or 3E-1 mg/kg-day

Confidence in the principal study is medium, as it was a well-designed experiment that examined a variety of toxicological endpoints and identified both a NOAEL and LOAEL for the critical effect. Confidence in the database is low, due to the lack of supporting subchronic, chronic, and developmental/reproductive studies. Accordingly, confidence in the provisional subchronic RfD is low.

DERIVATION OF A PROVISIONAL SUBCHRONIC OR CHRONIC RfC FOR PYRENE

No provisional RfC is developed due to lack of usable information.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR PYRENE

IRIS (U.S. EPA, 2007) provides no quantitative assessments (OSF or IUR) for pyrene and classifies it as classification of D, not classifiable as to human carcinogenicity based on no human data and inadequate data from animal experiments. Based on the U.S. EPA (2005) Cancer Guidelines, pyrene can be classified as “not likely to be a human carcinogen”.

No data is currently available and suitable for developing cancer values.

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Provisional Peer Reviewed Toxicity Values for

n-Butylbenzene (CASRN 104-51-8),
sec-Butylbenzene (CASRN 135-98-8)
and tert-Butylbenzene (CASRN 98-06-6)

Derivation of Subchronic and Chronic Oral RfDs

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
n-BUTYLBENZENE (CASRN 104-51-8), sec-BUTYLBENZENE (CASRN 135-98-8)
AND tert-BUTYLBENZENE (CASRN 98-06-6)
Derivation of Subchronic and Chronic Oral RfDs**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Subchronic or chronic RfDs for the isomeric butylbenzenes ($C_{10}H_{14}$) n-butylbenzene, sec-butylbenzene or tert-butylbenzene are not listed on IRIS (U.S. EPA, 2002a), the Drinking Water Standards and Health Advisories table (U.S. EPA, 2002b), or the HEAST (U.S. EPA, 1997). The only documents listed in the CARA database (U.S. EPA, 1991, 1994) for these chemicals are Health Advisories (U.S. EPA 1987a,b,c,d), wherein the data were considered inadequate for risk assessment for these chemicals. ATSDR (2002) has not published toxicological profiles for butylbenzenes and these chemicals were not listed in the NTP (2002) Management Status Reports. IARC (2002) and WHO (2002) have not published reviews for butylbenzenes. A review by Henderson (2001) was consulted for relevant information. Literature searches of the following databases were conducted from 1965 to July 2002 for relevant studies: TOXLINE, MEDLINE, TSCATS, GENETOX, HSDB, CANCERLIT, CCRIS, RTECS, EMIC/EMICBACK,

DART/ETICBACK and BIOSIS. An updated literature search was conducted through May 2004 and no relevant information was found.

REVIEW OF PERTINENT LITERATURE

Human Studies

No information was located regarding the adverse effects of n-butylbenzene, sec-butylbenzene, or tert-butylbenzene in humans.

Animal Studies

No studies on the chronic or subchronic toxicity of n-butylbenzene, sec-butylbenzene or tert-butylbenzene were located in the literature search. Limited acute data suggest that the branch-chained butylbenzenes are more lethal than the straight-chained butylbenzene. Gerarde (1959) observed mortality in 8/10 rats orally dosed with 4.3 g/kg of sec-butylbenzene and 7/10 at this dose of tert-butylbenzene, but only 2/10 rats treated with this dose of n-butylbenzene. Rat LD₅₀ values were lower for sec-butylbenzene (2.24 g/kg) and tert-butylbenzene (2.5-5.0 g/kg) (Henderson, 2001; Dupont, 1978; Rhone-Poulenc, Inc., 1981; NIOSH, 2002; Shell Oil Company, 1979). A rat LD₅₀ was not located for n-butylbenzene, but a mouse i.p. LD₅₀ for this chemical of 1.995 g/kg was obtained by Tanii et al., 1995. The leading cause of death in rats in the acute oral studies conducted by Gerarde (1959) was chemical induced pneumonitis with pulmonary edema and hemorrhage, the latter often associated with hemorrhage in other tissues such as thymus, adrenal, and bladder. Hyperemia and vasodilation of the blood vessels of the gastrointestinal tract were also reported.

FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC OR CHRONIC RfDs FOR n-BUTYLBENZENE, sec-BUTYLBENZENE AND tert-BUTYLBENZENE

Data on the oral toxicity of the three butylbenzene isomers under investigation are limited to acute lethality studies that are considered inadequate for derivation of provisional RfDs for butylbenzenes.

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U.S. EPA. 1987c. Drinking Water Health Advisory for sec-Butylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. December.

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Provisional Peer Reviewed Toxicity Values for
n-Butylbenzene (CASRN 104-51-8),
sec-Butylbenzene (CASRN 135-98-8)
and tert-Butylbenzene (CASRN 98-06-6)

Derivation of Subchronic and Chronic Inhalation RfCs

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
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MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
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INTRODUCTION

Subchronic or chronic RfCs the isomeric butylbenzenes ($C_{10}H_{14}$) n-butylbenzene, sec-butylbenzene or tert-butylbenzene are not listed on IRIS (U.S. EPA, 2002) or the HEAST (U.S. EPA, 1997). The only documents listed in the CARA database (U.S. EPA, 1991, 1994) for these chemicals are Health Advisories (U.S. EPA 1987a,b,c), wherein the data were considered inadequate for risk assessment for these chemicals. ATSDR (2002) has not published toxicological profiles for butylbenzenes and these chemicals were not listed in the NTP (2002) Management Status Reports. ACGIH (2001), NIOSH (2002) and OSHA (2002a,b) have not established occupational exposure limits for these chemicals. IARC (2002) and WHO (2002) have not published reviews for butylbenzenes. A review by Henderson (2001) was consulted for relevant information. Literature searches of the following databases were conducted from 1965 to July 2002 for relevant studies: TOXLINE, MEDLINE, TSCATS, GENETOX, HSDB,

CANCERLIT, CCRIS, RTECS, EMIC/EMICBACK, DART/ETICBACK and BIOSIS. An updated literature search was conducted through May 2004 and no relevant information was found.

REVIEW OF PERTINENT LITERATURE

Human Studies

No information was located regarding the adverse effects of n-butylbenzene, sec-butylbenzene, or tert-butylbenzene in humans.

Animal Studies

No studies on the subchronic or chronic inhalation toxicity of n-butylbenzene, sec-butylbenzene, or tert-butylbenzene were located in the literature search.

Available data on the toxicity of these alkylbenzenes are limited to acute duration exposures. In a review, Gerarde (1959) stated that direct contact (intratracheal instillation) of the liquid alkylbenzenes with pulmonary tissue causes chemical induced pneumonitis characterized by pulmonary edema, hemorrhage, and tissue necrosis.

n-Butylbenzene. The RD_{50} (concentration necessary to depress the respiratory rate by 50% during acute exposure in response to sensory irritation) for sensory irritation by n-butylbenzene was 710 ppm in a 30 minute exposure; the chemical did not produce pulmonary irritation (defined as a decrease in respiratory rate during exposure via tracheal cannula) at the RD_{50} (Nielsen and Alarie, 1982).

sec-Butylbenzene. Dow Chemical (1954) reported that no deaths occurred among 3 rats exposed to a saturated atmosphere (about 3400 ppm) of sec-butylbenzene for 7 hours. Slight eye irritation, drowsiness, and unsteadiness were observed, and moderate (unspecified) gross pathology of the liver and kidneys was seen at necropsy.

tert-Butylbenzene. A 4-hour LC_{50} of 4.6 mg/L (840 ppm) was reported for tert-butylbenzene in rats (Shell Oil Company, 1979). Clinical signs observed during exposure included lacrimation, salivation, tremors, and convulsions; necropsy of animals that survived the 14-day observation period revealed no gross pathological changes. The RD_{50} for tert-butylbenzene was 760 ppm in a 30 minute exposure; the chemical did not produce pulmonary irritation at the RD_{50} (Nielsen and Alarie, 1982).

**FEASIBILITY OF DERIVING PROVISIONAL SUBCHRONIC OR CHRONIC RfCs
FOR n-BUTYLBENZENE, sec-BUTYLBENZENE, tert-BUTYLBENZENE, and n-
PROPYLBENZENE**

Data on the inhalation toxicity of the butylbenzenes are limited to acute studies that are considered inadequate for derivation of provisional RfCs.

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Provisional Peer Reviewed Toxicity Values for
n-Butylbenzene (CASRN 104-51-8),
sec-Butylbenzene (CASRN 135-98-8)
and tert-Butylbenzene (CASRN 98-06-6)

Derivation of a Carcinogenicity Assessment

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration
p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

**PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR
n-BUTYLBENZENE (CASRN 104-51-8), sec-BUTYLBENZENE (CASRN 135-98-8)
AND tert-BUTYLBENZENE (CASRN 98-06-6)
Derivation of a Carcinogenicity Assessment**

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).
2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.
3. Other (peer-reviewed) toxicity values, including:
 - ▶ Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
 - ▶ California Environmental Protection Agency (CalEPA) values, and
 - ▶ EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for the Office of Superfund Remediation and Technology Innovation (OSRTI). Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or to EPA's Office of Superfund Remediation and Technology Information (OSRTI).

INTRODUCTION

Carcinogenicity assessments for the isomeric butylbenzenes ($C_{10}H_{14}$) n-butylbenzene, sec-butylbenzene or tert-butylbenzene are not listed on IRIS (U.S. EPA, 2002a), the Drinking Water Standards and Health Advisories table (U.S. EPA, 2002b), or the HEAST (U.S. EPA, 1997). The only documents listed in the CARA database (U.S. EPA, 1991, 1994) for these chemicals are Health Advisories (U.S. EPA 1987a,b,c), wherein the data were considered inadequate for risk assessment for these chemicals. ATSDR (2002) has not published toxicological profiles for butylbenzenes and these chemicals were not listed in the NTP (2002) Management Status Reports. IARC (2002) and WHO (2002) have not published reviews for butylbenzenes. A review by Henderson (2001) was consulted for relevant information. Literature searches of the following databases were conducted from 1965 to July 2002 for relevant studies: TOXLINE,

MEDLINE, TSCATS, GENETOX, HSDB, CANCERLIT, CCRIS, RTECS, EMIC/EMICBACK, DART/ETICBACK and BIOSIS. An updated literature search was conducted through May 2004 and no relevant information was found.

REVIEW OF PERTINENT LITERATURE

Human Studies

No data regarding the potential carcinogenicity of n-butylbenzene, sec-butylbenzene, or tert-butylbenzene in humans were located.

Animal Studies

No studies regarding the carcinogenicity of n-butylbenzene, sec-butylbenzene, or tert-butylbenzene in animals were located.

Other Studies

The only relevant information comes from a small number of genotoxicity studies for tert-butylbenzene.

tert-Butylbenzene. tert-Butylbenzene was not mutagenic in *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 or in *Escherichia coli* strains WP2 and WP2uvrA with or without metabolic activation when tested at concentrations of 0.2-2000 µg/plate (Dean et al., 1985). tert-Butylbenzene did not affect mitotic gene conversion (inactive alleles to wild-type alleles) in *Saccharomyces cerevisiae* at concentrations between 0.01 and 5.0 mg/ml in the presence or absence of metabolic activation by induced rat liver S9 fraction (Dean et al., 1985). *In vitro* exposure of rat liver cells (RL1) to 10, 20, or 40 µg/ml of tert-butylbenzene had no significant effect on the frequency of chromatid gaps, chromatid breaks or total chromosome aberrations (Dean et al., 1985). tert-Butylbenzene did not induce morphological transformation, or potentiate the morphological transformation frequency induced by positive control benzo(a)pyrene in Syrian hamster embryo (SHE) cells in culture (Rivedal et al., 1992).

PROVISIONAL WEIGHT-OF-EVIDENCE CLASSIFICATION

No studies of the potential carcinogenicity of n-butylbenzene, sec-butylbenzene, or tert-butylbenzene in humans or animals were located. Limited genotoxicity testing of tert-butylbenzene found no genotoxic activity. Therefore, n-butylbenzene, sec-butylbenzene, and

tert-butylbenzene data, under the proposed U.S. EPA (1999) guidelines, are inadequate for an assessment of human carcinogenic potential.

QUANTITATIVE ESTIMATES OF CARCINOGENIC RISK

Derivation of quantitative estimates of cancer risk for n-butylbenzene, sec-butylbenzene, or tert-butylbenzene is precluded by the lack of data regarding carcinogenicity of these chemicals.

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**Derivation of Exposure Point
Concentrations for Inhalation of
Potable Water Via Showering
Showering/Bathing**

APPENDIX D
CALCULATION OF EXPOSURE POINT CONCENTRATIONS FOR INHALATION OF POTABLE WATER VIA SHOWERING/BATHING
EXPOSURE POINT CONCENTRATIONS (EPC)
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

CASRN	Constituent	Concentration in		Adult EPC		Child EPC		Units
		Water		RME	CT	RME	CT	
		Cw	Units	Ca	Ca	Ca	Ca	
VOCs								
87-61-6	1,2,3-TRICHLOROBENZENE	1.21E-02	mg/L	1.48E-01	6.04E-02	2.63E-01	8.33E-02	mg/m3
120-82-1	1,2,4-TRICHLOROBENZENE	1.35E-02	mg/L	1.66E-01	6.77E-02	2.95E-01	9.33E-02	mg/m3
95-63-6	1,2,4-TRIMETHYLBENZENE	3.26E-01	mg/L	4.00E+00	1.63E+00	7.11E+00	2.25E+00	mg/m3
95-50-1	1,2-DICHLOROBENZENE	5.31E-01	mg/L	6.51E+00	2.65E+00	1.16E+01	3.66E+00	mg/m3
108-67-8	1,3,5-TRIMETHYLBENZENE	2.14E-01	mg/L	2.63E+00	1.07E+00	4.67E+00	1.48E+00	mg/m3
541-73-1	1,3-DICHLOROBENZENE	5.45E-03	mg/L	6.68E-02	2.73E-02	1.19E-01	3.76E-02	mg/m3
106-46-7	1,4-DICHLOROBENZENE	4.68E-01	mg/L	5.74E+00	2.34E+00	1.02E+01	3.23E+00	mg/m3
591-78-6	2-HEXANONE	1.95E-03	mg/L	2.39E-02	9.75E-03	4.25E-02	1.34E-02	mg/m3
67-64-1	ACETONE	7.80E-02	mg/L	9.56E-01	3.90E-01	1.70E+00	5.38E-01	mg/m3
71-43-2	BENZENE	5.83E+00	mg/L	7.15E+01	2.92E+01	1.27E+02	4.02E+01	mg/m3
75-27-4	BROMODICHLOROMETHANE	3.00E-03	mg/L	3.68E-02	1.50E-02	6.54E-02	2.07E-02	mg/m3
75-15-0	CARBON DISULFIDE	1.25E-02	mg/L	1.53E-01	6.25E-02	2.72E-01	8.61E-02	mg/m3
108-90-7	CHLOROBENZENE	1.81E-01	mg/L	2.22E+00	9.06E-01	3.95E+00	1.25E+00	mg/m3
75-00-3	CHLOROETHANE	4.58E-03	mg/L	5.61E-02	2.29E-02	9.98E-02	3.16E-02	mg/m3
67-66-3	CHLOROFORM	1.18E-02	mg/L	1.44E-01	5.88E-02	2.56E-01	8.10E-02	mg/m3
100-41-4	ETHYLBENZENE	1.47E-01	mg/L	1.80E+00	7.33E-01	3.20E+00	1.01E+00	mg/m3
98-82-8	ISOPROPYLBENZENE	3.95E-03	mg/L	4.84E-02	1.97E-02	8.60E-02	2.72E-02	mg/m3
75-09-2	METHYLENE CHLORIDE	7.44E-04	mg/L	9.12E-03	3.72E-03	1.62E-02	5.13E-03	mg/m3
99-87-6	P-ISOPROPYLTOLUENE	3.31E-03	mg/L	4.06E-02	1.66E-02	7.22E-02	2.28E-02	mg/m3
135-98-8	SEC-BUTYLBENZENE	1.19E-02	mg/L	1.46E-01	5.94E-02	2.59E-01	8.19E-02	mg/m3
100-42-5	STYRENE	8.20E-01	mg/L	1.00E+01	4.10E+00	1.79E+01	5.65E+00	mg/m3
127-18-4	TETRACHLOROETHENE	2.96E-04	mg/L	3.63E-03	1.48E-03	6.45E-03	2.04E-03	mg/m3
108-88-3	TOLUENE	1.27E+00	mg/L	1.56E+01	6.35E+00	2.77E+01	8.76E+00	mg/m3
75-01-4	VINYL CHLORIDE	1.10E-03	mg/L	1.35E-02	5.50E-03	2.40E-02	7.58E-03	mg/m3
1330-20-7	XYLENES, TOTAL	9.55E-01	mg/L	9.03E+00	3.78E+00	1.58E+01	5.11E+00	mg/m3

APPENDIX D
CALCULATION OF EXPOSURE POINT CONCENTRATIONS FOR INHALATION OF POTABLE WATER VIA SHOWERING/BATHING
EXPOSURE POINT CONCENTRATIONS (EPC)
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

a - Maximum air concentration in the bathroom derived by the following equation from Shaum et al. 1994.

$$C_{a \max} = \frac{C_w f F_w t_1}{V_a}$$

b - Concentration of contaminant in the air derived by the following equation from Shaum et al. 1994.

$$C_a = \frac{(C_{a \max} / 2)t_1 + C_{a \max} t_2}{t_1 + t_2}$$

Where (all scenarios): Fraction volatilized (f) = 1, water flow rate (F_w) = 750 L/day, bathroom volume (V_a) = 12 m³

Where (adult scenarios): time of shower (t_1) = 0.25 hr (RME), 0.1 hr (CT); time after shower (t_2) = 0.33 hr (RME), 0.15 hr (CT)

Where (child scenarios): time of shower (t_1) = 0.45 hr (RME), 0.14 hr (CT); time after shower (t_2) = 0.55 hr (RME), 0.19 hr (CT)

Source:

Schaum, J., K. Hoang, R. Kinerson, J. Moya, and R.G.M. Wang. 1994. Estimating Dermal and Inhalation Exposure to volatile Chemicals in Domestic Water. USEPA Region II.

**Derivation of Soil-to-Air
Volatilization Factors**

APPENDIX E, TABLE 1
SOIL-TO-AIR VOLATILIZATION FACTORS
Soil/Ditch Sediment Constituents of Potential Concern Subject to Inhalation Pathway
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

Soil COC List	COPC Evaluated for Soil Vapor or Dust
<i>Metals</i>	
ALUMINUM	Dust
ANTIMONY	Dust
ARSENIC	Dust
BARIUM	Dust
CADMIUM	Dust
CHROMIUM	Dust
COPPER	Dust
IRON	Dust
LEAD	Dust
MANGANESE	Dust
MERCURY	Dust
METHYL MERCURY	Dust
SILVER	Dust
THALLIUM	Dust
VANADIUM	Dust
<i>PCBs</i>	
Less Chlorinated PCBs	Dust
Highly Chlorinated PCBs	Dust
Total PCBs	Dust
<i>Pesticides</i>	
DIELDRIN	Dust
ENDOSULFAN SULFATE	Dust
ENDRIN ALDEHYDE	Dust
ENDRIN KETONE	Dust
<i>SVOCs</i>	
2,4-DIMETHYLPHENOL	Dust
2-METHYLNAPHTHALENE	Dust
3&4-METHYLPHENOL	Dust
ACENAPHTHENE	Dust
ACENAPHTHYLENE	Dust
ANTHRACENE	Dust
BENZO(A)ANTHRACENE	Dust
BENZO(A)PYRENE	Dust
BENZO(B)FLUORANTHENE	Dust
BENZO(G,H,I)PERYLENE	Dust
BENZO(K)FLUORANTHENE	Dust
CARBAZOLE	Dust
CHRYSENE	Dust
DIBENZO(A,H)ANTHRACENE	Dust
DIBENZOFURAN	Dust
FLUORANTHENE	Dust
FLUORENE	Dust

APPENDIX E, TABLE 1
SOIL-TO-AIR VOLATILIZATION FACTORS
Soil/Ditch Sediment Constituents of Potential Concern Subject to Inhalation Pathway
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

Soil COC List	COPC Evaluated for Soil Vapor or Dust
HEXACHLOROBENZENE	Dust
INDENO(1,2,3-CD)PYRENE	Dust
NAPHTHALENE	Dust
PHENANTHRENE	Dust
PYRENE	Dust
<i>VOCs</i>	
1,2,3-TRICHLOROBENZENE	Vapor
1,2,4-TRICHLOROBENZENE	Vapor
1,2,4-TRIMETHYLBENZENE	Vapor
1,3,5-TRIMETHYLBENZENE	Vapor
1,2-DICHLOROBENZENE	Vapor
1,3-DICHLOROBENZENE	Vapor
1,4-DICHLOROBENZENE	Vapor
ACETONE	Vapor
BENZENE	Vapor
BROMOMETHANE	Vapor
CHLOROBENZENE	Vapor
P-ISOPROPYLTOLUENE	Vapor
TOLUENE	Vapor
XYLENES, TOTAL	Vapor

Notes:

Compound list generated from surface and subsurface soil COPCs selected in RAGS 2 Series. Due to the ephemeral nature of the I-690 Drainage Ditch, surface sediment COPCs were also considered for the inhalation pathway.

Only volatile organic compounds are considered for the vapor inhalation pathway. All other compound classes are considered for the inhalation of fugitive dust particles.

APPENDIX E, TABLE 2
SOIL-TO-AIR VOLATILIZATION FACTORS
CALCULATING Q/C_{vol}
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

CALCULATING Q/C_{vol}

Exposure Unit	Receptor	Exposure Areas*	Area (acres)	Total Area for EU	Q/C _{vol} (g/m ² -s per kg/m ³)
1	Older Child Trespasser	Lakeshore Area	54.20	97.59	43.1
	Utility Worker	DSA #1	1.39		
	Construction Worker	DSA #2	1.50		
		AOS #1	10.20		
		AOS #2	2.26		
		I-690 Drainage Ditch	0.43		
		Penn-Can Property	13.63		
		Railroad Area	13.98		
2	Surveillance Worker	Lakeshore Area	54.20	57.09	46.3
		DSA #1	1.39		
		DSA #2	1.50		
3	Drainage Ditch Worker	Interstate-690 Drainage Ditch	0.43	0.43	101.2
4	Railroad Worker	Railroad Area	13.98	13.98	56.7
5	Commercial/Industrial Worker	Penn-Can Property	13.63	13.63	56.9
6	Recreator (Adult and Child)	Lakeshore Area	54.20	67.29	45.3
	Resident (Adult and Child)	DSA #1	1.39		
		DSA #2	1.50		
		AOS #1	10.20		
7	Commercial/Industrial Worker	Penn-Can Property	13.63	83.18	44.0
		Lakeshore Area	54.20		
		DSA #1	1.39		
		DSA #2	1.50		
		AOS #1	10.20		
		AOS #2	2.26		
9	Recreator (Adult and Child)	SYW-12	45.6	45.60	47.8
	Railroad Worker				
	Utility Worker				
	Resident (Adult and Child)				
	Commercial/Industrial Worker				
	Construction Worker				

$$Q/C_{vol} = A \times \exp \left[\frac{(\ln A_s - B)^2}{C} \right]$$

Exhibit D-3, USEPA 2002

Variable	Value	Units/Rationale/Source
A =	16.8653	unitless, Chicago Zone 7 values, Exhibit D-3, USEPA 2002
B =	18.7848	unitless, Chicago Zone 7 values, Exhibit D-3, USEPA 2002
C =	215.0624	unitless, Chicago Zone 7 values, Exhibit D-3, USEPA 2002

Notes:

* Only those areas within an exposure unit that contain soil are presented (sediment for I-690 Drainage Ditch).

Exposure Unit 8 is not exposed to volatile and particulate emission from soil.

Reference:

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Solid Waste and Emergency Response. OSWER 9355.4-24. December 2002.

APPENDIX E TABLE 3
SOIL-TO-AIR VOLATILIZATION FACTORS
APPARENT DIFFUSIVITY CALCULATIONS
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

Parameter	Units	Value	Source
Soil Particle Density	(g/cm ³)	2.65	default value, USEPA, 2002, Equation 4-8
Water-filled Soil Porosity	(L _{water} /L _{soil})	0.15	default value, USEPA, 2002, Equation 4-8
Total Soil Porosity	(L _{pore} /L _{soil})	0.43	default value, USEPA, 2002, Equation 4-8
Air-filled Soil Porosity	(L _{air} /L _{soil})	0.28	default value, USEPA, 2002, Equation 4-8
Dry Soil Bulk Density	(g/cm ³)	1.5	default value, USEPA, 2002, Equation 4-8
Exposure Interval	(s)	9.50E+08	default value, USEPA, 2002, Equation 4-8
Fraction Organic Carbon in Soil	g/g	6.00E-03	default value, USEPA, 2002, Equation 4-8

Compound	Diffusivity in Air (D _i) (cm ² /s)	Dimensionless Henry's Law (H') Constant	Diffusivity in Water (D _w) (cm ² /s)	Soil-Water Partition Coefficient (K _d) (cm ³ /g)	Soil Organic Carbon Partition Coefficient (K _{oc}) (cm ³ /g)	Apparent Diffusivity (D _A) (cm ² /s)
1,2,3-TRICHLOROBENZENE ^a	3.00E-02	5.82E-02	8.23E-06	1.07E+01	1.78E+03	8.39E-06
1,2,4-TRICHLOROBENZENE	3.00E-02	5.82E-02	8.23E-06	1.07E+01	1.78E+03	8.39E-06
1,2,4-TRIMETHYLBENZENE ^b	6.44E-02	2.52E-01	7.92E-06	1.29E+01	2.15E+03	6.44E-05
1,3,5-TRIMETHYLBENZENE ^b	6.02E-02	3.59E-01	8.67E-06	1.29E+01	2.15E+03	8.57E-05
1,2-DICHLOROBENZENE	6.90E-02	7.79E-02	7.90E-06	3.70E+00	6.17E+02	7.30E-05
1,3-DICHLOROBENZENE ^c	6.90E-02	8.88E-02	7.90E-06	3.70E+00	6.17E+02	8.31E-05
1,4-DICHLOROBENZENE	6.90E-02	9.96E-02	7.90E-06	3.70E+00	6.17E+02	9.32E-05
ACETONE	1.24E-01	1.59E-03	1.14E-05	3.45E-03	5.75E-01	9.92E-05
BENZENE	8.80E-02	2.28E-01	9.80E-06	3.53E-01	5.89E+01	2.10E-03
BROMOMETHANE ^b	7.28E-02	2.55E-01	1.21E-05	8.58E-02	1.43E+01	4.12E-03
CHLOROBENZENE	7.30E-02	1.52E-01	8.70E-06	1.31E-04	2.19E-02	4.47E-03
P-ISOPROPYLTOLUENE	NV	NV	NV	4.62E+00	7.70E+02	NV
TOLUENE	8.70E-02	2.72E-01	8.60E-06	1.09E+00	1.82E+02	9.86E-04
XYLENES, TOTAL ^d	7.80E-02	1.30E+02	8.75E-06	2.32E+00	3.86E+02	1.97E-02

Notes:

Values are from USEPA 2002 unless otherwise noted.

a = 1,2,4-Trichlorobenzene used as surrogate.

b = Values taken from Risk Assessment Information System: Chemical Specific Factors Database, Accessed February 2008.

c = Average of values for 1,4-Dichlorobenzene and 1,2-Dichlorobenzene

d = Average values for m-xylene, o-xylene, and p-xylene.

Apparent Diffusivity (D_A) calculated using equation 4-8 USEPA 2002.

K_d for organic was calculated as per USEPA 2002: K_{oc} × F_{oc}

Reference:

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Solid Waste and Emergency Response.

APPENDIX E, TABLE 4
SOIL-TO-AIR VOLATILIZATION FACTORS
VOLATILIZATION FACTOR CALCULATIONS
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

$$VF(m^3 / kg) = Q / C \times \frac{(3.14 \times D_A \times T)^{1/2}}{(2 \times \rho_b \times D_A)} \times 10^{-4} (m^2 / cm^2)$$

Constituent	Volatilization Factor (mg ³ /kg)							
	Exposure Unit 1	Exposure Unit 2	Exposure Unit 3	Exposure Unit 4	Exposure Unit 5	Exposure Unit 6	Exposure Unit 7	Exposure Unit 9
1,2,3-TRICHLOROBENZENE	2.71E+04	2.91E+04	6.36E+04	3.56E+04	3.58E+04	2.85E+04	2.77E+04	3.00E+04
1,2,4-TRICHLOROBENZENE	2.71E+04	2.91E+04	6.36E+04	3.56E+04	3.58E+04	2.85E+04	2.77E+04	3.00E+04
1,2,4-TRIMETHYLBENZENE	9.77E+03	1.05E+04	2.29E+04	1.29E+04	1.29E+04	1.03E+04	9.98E+03	1.08E+04
1,3,5-TRIMETHYLBENZENE	8.48E+03	9.11E+03	1.99E+04	1.11E+04	1.12E+04	8.91E+03	8.66E+03	9.40E+03
1,2-DICHLOROBENZENE	9.18E+03	9.87E+03	2.16E+04	1.21E+04	1.21E+04	9.65E+03	9.38E+03	1.02E+04
1,3-DICHLOROBENZENE	8.61E+03	9.25E+03	2.02E+04	1.13E+04	1.14E+04	9.05E+03	8.79E+03	9.54E+03
1,4-DICHLOROBENZENE	8.13E+03	8.73E+03	1.91E+04	1.07E+04	1.07E+04	8.54E+03	8.30E+03	9.01E+03
ACETONE	7.88E+03	8.47E+03	1.85E+04	1.04E+04	1.04E+04	8.28E+03	8.05E+03	8.74E+03
BENZENE	1.71E+03	1.84E+03	4.02E+03	2.25E+03	2.26E+03	1.80E+03	1.75E+03	1.90E+03
BROMOMETHANE	1.22E+03	1.31E+03	2.87E+03	1.61E+03	1.61E+03	1.28E+03	1.25E+03	1.36E+03
CHLOROBENZENE	1.17E+03	1.26E+03	2.75E+03	1.54E+03	1.55E+03	1.23E+03	1.20E+03	1.30E+03
P-ISOPROPYLTOLUENE	NV	NV	NV	NV	NV	NV	NV	NV
TOLUENE	2.50E+03	2.68E+03	5.86E+03	3.29E+03	3.30E+03	2.63E+03	2.55E+03	2.77E+03
XYLENES, TOTAL	5.59E+02	6.01E+02	1.31E+03	7.36E+02	7.39E+02	5.88E+02	5.71E+02	6.20E+02

Reference
Equation 4-8, USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Solid Waste and Emergency Response. OSWER 9355.4-24. December 2002.

**Derivation of Soil-to-Air Particulate
Emission Factors**

APPENDIX F, TABLE 1
PARTICULATE EMISSIONS FACTOR CALCULATIONS
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

PEF to be used for the following scenarios involving fugitive dust exposure:
Trespasser, Residents (Adult and Child), Recreator (Adult and Child), Surveillance
Worker, Railroad Worker, and Commercial/Industrial Worker.

$$Q/C_{wind} = A \times \exp \left[\frac{(\ln A_s - B)^2}{C} \right]$$

Equation D-1, USEPA 2002

Variable	Value	Units	Rationale
A =	16.8653	unitless	Chicago Zone 7 values
B =	18.7848	unitless	Chicago Zone 7 values
C =	215.0624	unitless	Chicago Zone 7 values
A _s =	97.5	acres	EU 1: Areal extent of contamination (Site Wide - Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area) ^a
A _s =	57.09	acres	EU 2: Areal extent of contamination (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2) ^a
A _s =	13.98	acres	EU 4: Areal extent of contamination (Railroad Area)
A _s =	13.63	acres	EU 5: Areal extent of contamination (Penn-Can Property)
A _s =	67.29	acres	EU 6: Areal extent of contamination (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1) ^a
A _s =	83.18	acres	EU 7: Areal extent of contamination (Penn-Can Property, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2) ^a
A _s =	45.6	acres	EU 9: Areal extent of contamination (SYW-12)

Q/C _{Sr} EU1 =	43.0990	g/m ² -s per kg/m ³
Q/C _{Sr} EU2 =	46.3182	g/m ² -s per kg/m ³
Q/C _{Sr} EU4 =	56.6907	g/m ² -s per kg/m ³
Q/C _{Sr} EU5 =	56.9071	g/m ² -s per kg/m ³
Q/C _{Sr} EU6 =	45.2919	g/m ² -s per kg/m ³
Q/C _{Sr} EU7 =	44.0181	g/m ² -s per kg/m ³
Q/C _{Sr} EU9 =	47.7784	g/m ² -s per kg/m ³

APPENDIX F, TABLE 1
PARTICULATE EMISSIONS FACTOR CALCULATIONS
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

PEF to be used for the following scenarios involving fugitive dust exposure:
Trespasser, Residents (Adult and Child), Recreator (Adult and Child), Surveillance
Worker, Railroad Worker, and Commercial/Industrial Worker.

$$PEF_{wind} = Q/C_{wind} \times \frac{3600s/h}{0.036 \times (1 - V) \times (U_m/U_t)^3 \times F(x)}$$

Equation 4-5, USEPA 2002

Variable	Value	Units	Rationale
$U_m =$	4.69	m/s	Mean annual windspeed (USEPA 2007)
$U_t =$	11.32	m/s	Threshold windspeed at 7m (USEPA 2007)
$F(x) =$	0.194	unitless	Function dependent on U_m/U_t derived using Cowherd et al. (1985)
$V_{EU1} =$	0.77	unitless	EU 1: Fraction Vegetative Cover Estimated (Site Wide - Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, I-690 Drainage Ditch, Penn-Can Property, Railroad Area) ^a
$V_{EU2} =$	0.90	unitless	EU 2: Fraction Vegetative Cover Estimated (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2) ^a
$V_{EU4} =$	0.50	unitless	EU 4: Fraction Vegetative Cover Estimated (Railroad Area)
$V_{EU5} =$	0.30	unitless	EU 5: Fraction Vegetative Cover Estimated (Penn-Can Property)
$V_{EU6} =$	0.92	unitless	EU 6: Fraction Vegetative Cover Estimated (Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1) ^a
$V_{EU7} =$	0.82	unitless	EU 7: Fraction Vegetative Cover Estimated (Penn-Can Property, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2) ^a
$V_{EU9} =$	0.85	unitless	EU 9: Fraction Vegetative Cover Estimated (SYW-12)

$PEF_{EU1} =$	1.37E+09	m ³ /kg
$PEF_{EU2} =$	3.44E+09	m ³ /kg
$PEF_{EU4} =$	8.22E+08	m ³ /kg
$PEF_{EU5} =$	5.89E+08	m ³ /kg
$PEF_{EU6} =$	3.97E+09	m ³ /kg
$PEF_{EU7} =$	1.74E+09	m ³ /kg
$PEF_{EU9} =$	2.31E+09	m ³ /kg

Notes:

a = Total affected area includes only those sites that contain surface soil.

Sources:

EPA 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24.
EPA 2007. Human Health Risk Assessment Onondaga Lake Wastebeds 1-8 Site: Bike Trail. Geddes, NY. November
Cowherd et al. (1985) Rapid Assessment of Exposure to Particulate Emissions from Surface Contaminated Sites
EPA/600/8-85/002

APPENDIX F, TABLE 2
PARTICULATE EMISSIONS FACTOR CALCULATIONS
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

PEF to be used for Construction & Utility worker/fugitive dust scenario

$$Q/C_{sr} = A \times \exp \left[\frac{(\ln A_s - B)^2}{C} \right]$$

Equation E-19, USEPA 2002

<u>Variable</u>	<u>Value</u>	<u>Units</u>	<u>Rationale</u>
A =	12.9351	unitless	default as per USEPA 2002
B =	5.7383	unitless	default as per USEPA 2002
C =	71.7711	unitless	default as per USEPA 2002
A_s =	0.946	acres	EU 1: Areal extent of contamination (Assumes 97.5 acre square site bisected by one construction access road 2060.85 feet long and 20 feet wide).
A_s =	0.647	acres	EU 9: Areal extent of contamination (Assumes 45.6 acre square site bisected by one construction access road feet long and 20 feet wide).
$Q/C_{sr \text{ EU1}}$ =	20.6488	$\text{g/m}^2\text{-s per kg/m}^3$	
$Q/C_{sr \text{ EU9}}$ =	21.9986	$\text{g/m}^2\text{-s per kg/m}^3$	

$$F_D = 0.1852 + \frac{5.3537}{t} + \frac{-9.6318}{t^2}$$

Equation E-16, USEPA 2002

<u>Variable</u>	<u>Value</u>	<u>Units</u>	<u>Rationale</u>
$t_{(EU \ 1)}$ =	262	hours	Time vehicles are on the road during the year. (assumes 10 vehicles traversing the length (628.13 meters) of the road 4 times per day for 250 days traveling an average of 24 kph).
$t_{(EU \ 9)}$ =	179	hours	Time vehicles are on the road during the year. (assumes 10 vehicles traversing the length (628.13 meters) of the road 4 times per day for 250 days traveling an average of 24 kph).
$F_{D \ (EU \ 1)}$ =	0.205493654	unitless	
$F_{D \ (EU \ 9)}$ =	0.214809886	unitless	

APPENDIX F, TABLE 2
PARTICULATE EMISSIONS FACTOR CALCULATIONS
Honeywell Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

$$PEF_{const} = Q/C_{sr} \times \frac{1}{F_D} \times \frac{T \times A_R}{556 \times (W/3)^{0.4} \times \left[\frac{(365 - p)}{365} \right] \times \sum VKT}$$

Equation 5-5, USEPA 2002

<u>Variable</u>	<u>Value</u>	<u>Units</u>	<u>Rationale</u>
T =	7200000	seconds	Total time of construction (8 hours/day X 250 days X 1 year)
A _R (EU 1) =	3831.59	m ²	Area of contaminated road (L _R = 628.13 meters, W _R = 6.1 meters)
A _R (EU 9) =	2619.16	m ²	Area of contaminated road (L _R = 429.37 meters, W _R = 6.1 meters)
W =	11.5	tons	Mean vehicle weight (average of 5 pickup trucks (3 tons) and 5 dump trucks (20 tons))
ΣVKT (EU 1) =	6281.3	km	Sum of vehicle km traveled (10 vehicles X 4 trips/day/vehicle X 628.13 meters/trip X 250 days)
ΣVKT (EU 9) =	4293.7	km	Sum of vehicle km traveled (10 vehicles X 4 trips/day/vehicle X 429.37 meters/trip X 250 days)
p =	171	days	Mean number of days with 0.01 inches precipitation or more. Data for Syracuse, NY collected by Cornell University NRCC.
PEF_{const}: EU1	8.72E+05	m³/kg	
PEF_{const}: EU9	8.89E+05	m³/kg	

Sources:

EPA 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24. December

Northeast Regional Climate Center. Cornell University. Accessed 1/15/08 <<http://www.nrcc.cornell.edu/ccd.html>>

Exposure Parameters Summary

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Child Resident		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	200	USEPA 1997; Table 4-23 (suggested conservative value for children)	100	USEPA 1997; Table 4-23 (mean value for children)
Ingestion Rate of Water	IR	L/day	1	USEPA 1989; Exhibit 6-11	1	USEPA 1989; Exhibit 6-11
Inhalation Rate	InR	m ³ /hr	0.42	USEPA 1997; Table 5-11 (10 m ³ /day inhalation)	0.42	USEPA 1997; Table 5-11 (10 m ³ /day inhalation)
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area (showering/bathing pathway)	SA	cm ²	6600	USEPA 2004; Exhibit 3-2, Entire body skin surface area for a child bathing	6600	USEPA 2004; Exhibit 3-2, Entire body skin surface area for a child bathing
Skin Surface Area Available for Contact	SA	cm ² /day	2800	USEPA 2004, Exhibit C-1	2800	USEPA 2004, Exhibit C-1
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.2	USEPA 2004, Exhibit 3-3 and page 3-14	0.04	USEPA 2004, Exhibit 3-3 and page 3-14
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	NA		NA	
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	24	Assumes 24 hour a day exposure	24	Assumes 24 hour a day exposure
Event Duration	t _{event}	hr/event	1	Schaum et al. 2004 (RME for a child showering/bathing)	0.33	Schaum et al. 2004 (CT for a child showering/bathing)
Lag Time per Event	t _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	350	USEPA 2004, Exhibit 3-2	350	USEPA 2004, Exhibit 3-2
Exposure Duration	ED	years	6	USEPA 2004, Exhibit 3-2	6	USEPA 2004, Exhibit 3-2
Body Weight	BW	kg	15	USEPA 1991, Section 6.0 Summary Table	15	USEPA 1991, Section 6.0 Summary Table
Averaging Time - Non-Cancer	AT-NC	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	2190	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Adult Resident		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	50	USEPA 1997; Table 4-23 (mean value for adults)	50	USEPA 1997; Table 4-23 (mean value for adults)
Ingestion Rate of Water	IR	L/day	2	USEPA 1989; Exhibit 6-11	2	USEPA 1989; Exhibit 6-11
Inhalation Rate	InR	m ³ /hr	0.8	USEPA 1997; Table 5-11 (20 m ³ /day inhalation)	0.8	USEPA 1997; Table 5-11 (20 m ³ /day inhalation)
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area (showering/bathing pathway)	SA	cm ²	18000	USEPA 2004; Exhibit 3-2, Entire body skin surface area for an adult showering	18000	USEPA 2004; Exhibit 3-2, Entire body skin surface area for an adult showering
Skin Surface Area Available for Contact	SA	cm ² /day	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.07	USEPA 2004, Exhibit 3-3 and page 3-14	0.01	USEPA 2004, Exhibit 3-3 and page 3-14
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	NA		NA	
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	16	BPJ, assumes 24 hours minus 8 hours working offsite	16	BPJ, assumes 24 hours minus 8 hours working offsite
Event Duration	t _{event}	hr/event	0.58	Schaum et al. 1994 (RME for an adult showering/bathing)	0.25	Schaum et al. 1994 (CT for an adult showering/bathing)
Lag Time per Event	t _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	350	USEPA 2004, Exhibit 3-2	350	USEPA 2004, Exhibit 3-2
Exposure Duration	ED	years	30	USEPA 2004, Exhibit 3-2	9	USEPA 2004, Exhibit 3-2
Body Weight	BW	kg	70	USEPA 1991, Section 6.0 Summary Table	70	USEPA 1991, Section 6.0 Summary Table
Averaging Time - Non-Cancer	AT-NC	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

Receptor:		Child Recreator				
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	200	USEPA 1997; Table 4-23 (suggested conservative value for children)	100	USEPA 1997; Table 4-23 (mean value for children)
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1.2	USEPA 1997; Table 5-23 (mean value for children, moderate activities)	1	USEPA 1997; Table 5-23 (mean value for children, light activities)
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	0.5	BPJ, assumes half of the dose comes from onsite due to a potentially larger geographic range
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area (dermal exposure to water)	SA	cm ²	2800	USEPA 2004, Exhibit C-1	2800	USEPA 2004, Exhibit C-1
Skin Surface Area Available for Contact	SA	cm ² /day	2800	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	2800	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(e)	0.2	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(b)
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(e)	0.2	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(b)
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Event Duration	t _{event}	hr/event	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Lag Time per Event	t _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	NA		NA	
Exposure Frequency	EF	days/year	42	BPJ, Assumes site access 2 days per week during the summer months and 1 days per week when school is in session (2X10 + 1X22) = 42	32	BPJ, (NYSDEC/TAMS Ninemile Creek HHRA), see comment 4.13(b)
Exposure Duration	ED	years	6	USEPA, 2004; Exhibit 3-2 (RME exposure duration for child resident)	6	USEPA, 2004; Exhibit 3-2 (CT exposure duration for child resident)
Body Weight	BW	kg	15	USEPA 1991, Section 6.0 Summary Table	15	USEPA 1991, Section 6.0 Summary Table
Averaging Time - Non-Cancer	AT-NC	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	2190	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Cooking Loss (PCBs and PCDD/PCDFs only)	CL	unitless	NA		0.33	USEPA 2004, Exhibit A-9
Exposure Frequency (For Fish Consumption)	EF _{fish}	days/year	365	USEPA 1997, Page 10-26	365	USEPA 1997, Page 10-26
Fraction Ingested from Fish	FI _{fish}	unitless	1	BPJ, assumes all fish consumed comes from onsite.	1	BPJ, assumes all fish consumed comes from onsite.
Ingestion Rate (For Fish Consumption)	IR _{fish}	g fish/day	8.3	USEPA 1997, 10-26. 1/3 of the Adult value to account for difference in body weight.	2.7	USEPA 1997, 10-26. 1/3 of the Adult value to account for difference in body weight.

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Adult Recreator		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	50	USEPA 1997; Table 4-23 (mean value for adults)	50	USEPA 1997; Table 4-23 (mean value for adults)
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1.6	USEPA 1997; Table 5-23 (mean value for adults, moderate activities)	1	USEPA 1997; Table 5-23 (mean value for adults, light activities)
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	0.5	BPJ, assumes half of the dose comes from onsite due to a potentially larger geographic range
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area (dermal exposure to water)	SA	cm ²	5700	USEPA 2004, Exhibit C-1; NYSDC 2002, Onondaga Lake HHRA	5700	USEPA 2004, Exhibit C-1; NYSDC 2002, Onondaga Lake HHRA
Skin Surface Area Available for Contact	SA	cm ² /day	5700	USEPA 2004, Exhibit C-1; NYSDC 2002, Onondaga Lake HHRA	5700	USEPA 2004, Exhibit C-1; NYSDC 2002, Onondaga Lake HHRA
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(d)	0.15	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(a)
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	0.3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(d)	0.15	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(a)
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Event Duration	t _{event}	hr/event	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Lag Time per Event	T _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	42	BPJ, Assumes adult recreator will accompany the child recreator and therefore have the same exposure frequency	32	BPJ, (NYSDEC/TAMS Ninemile Creek HHRA), see comment 4.13(b)
Exposure Duration	ED	years	30	USEPA, 2004; Exhibit 3-2 (RME exposure duration for adult resident)	9	USEPA, 2004; Exhibit 3-2 (CT exposure duration for adult resident)
Body Weight	BW	kg	70	USEPA 1991, Section 6.0 Summary Table	70	USEPA 1991, Section 6.0 Summary Table
Averaging Time - Non-Cancer	AT-NC	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Cooking Loss (PCBs and PCDD/PCDFs only)	CL	unitless	NA		0.33	USEPA 1997, Section 10.9
Exposure Frequency (For Fish Consumption)	EF _{fish}	days/year	365	USEPA 1997, Page 10-26	365	USEPA 1997, Page 10-26
Fraction Ingested from Fish	FI _{fish}	unitless	1	BPJ, assumes all fish consumed comes from onsite.	1	BPJ, assumes all fish consumed comes from onsite.
Ingestion Rate (For Fish Consumption)	IR _{fish}	g fish/day	25	USEPA 1997, Page 10-26	8	USEPA 1997, Page 10-26

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Construction Worker		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	330	USEPA 2002; Exhibit 1-2, as construction worker	330	USEPA 2002; Exhibit 1-2, as construction worker
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	3.2	USEPA 1997, Table 5-23, mean value for adults, heavy activities. See comment 4.6RME.	1.6	USEPA 1997, Table 5-23, mean value for adults, moderate activities. See comment 4.6CT.
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area	SA	cm ²	3300	USEPA 2002; Exhibit 1-2, as construction worker	3300	USEPA 2002; Exhibit 1-2, as construction worker
Skin Surface Area Available for Contact	SA	cm ² /day	3300	USEPA 2002; Exhibit 1-2, as construction worker	3300	USEPA 2002; Exhibit 1-2, as construction worker
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.3	USEPA, 2004; Exhibit 3-3, 95th percentile for construction worker	0.1	USEPA, 2004; Exhibit 3-3, geometric mean for construction worker
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	0.9	USEPA, 2004; Exhibit 3-3	0.1	USEPA, 2004; Exhibit 3-3
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)
Event Duration	t _{event}	hr/event	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)
Lag Time per Event	t _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	250	USEPA, 2004, Exhibit 3-5, RME for industrial worker, see comment 4.5(b)	125	BPJ, half of the RME assumes half of the working days of the year
Exposure Duration	ED	years	1	BPJ, see comment 4.5(b)	1	BPJ, see comment 4.5(b)
Body Weight	BW	kg	70	USEPA 1997; Table 7-11	70	USEPA 1997; Table 7-11
Averaging Time - Non-Cancer	AT-NC	days	365	USEPA 1989, Exhibits 6-11 through 6-16	365	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Surveillance Worker		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	100	USEPA 2002; Exhibit 1-2, as outdoor worker	100	USEPA 2002; Exhibit 1-2, as outdoor worker
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1	USEPA 1997, Table 5-23, mean value for adult, light activities (most surveillance is done from the vehicle).	1	USEPA 1997, Table 5-23, mean value for adult, light activities (most surveillance is done from the vehicle).
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	NA		NA	
Skin Surface Area	SA	cm ²	NA		NA	
Skin Surface Area Available for Contact	SA	cm ² /day	2480	USEPA 2004; Exhibit C-1 (hands, forearms, and face)	1930	USEPA 2004; Exhibit C-1
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.07	USEPA 2004, Exhibit 3-3 and page 3-14	0.01	USEPA 2004, Exhibit 3-3 and page 3-14
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	NA		NA	
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	NA		NA	
Exposure Time	ET	hours/day	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	1	BPJ, Surveillance workers are likely only onsite for 1 hour per day as they are disposed to other locations
Event Duration	t _{event}	hr/event	NA		NA	
Lag Time per Event	t _{event}	hr/event	NA		NA	
Time to Reach Steady-State	t*	hr	NA		NA	
Event Frequency	EV	event/day	NA		NA	
Exposure Frequency	EF	days/year	37	BPJ, 1 day/week x 50 weeks/year and 25% snow coverage throughout. See comment 4.3RME(b) and more recent comment 6 of 5/9/08 letter.	37	BPJ, 1 day/week x 50 weeks/year and 25% snow coverage throughout. See comment 4.3RME(b) and more recent comment 6 of 5/9/08 letter.
Exposure Duration	ED	years	25	USEPA 2004, Exhibit 3-5, RME for industrial workers	9	USEPA 2004, Exhibit 3-5, CT for industrial workers
Body Weight	BW	kg	70	USEPA 1997; Table 7-11	70	USEPA 1997; Table 7-11
Averaging Time - Non-Cancer	AT-NC	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	NA		NA	

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Railroad Worker		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	100	USEPA 2002; Exhibit 1-2, as outdoor worker	100	USEPA 2002; Exhibit 1-2, as outdoor worker
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	2.5	USEPA 1997, Table 5-23, mean value for outdoor worker, heavy activities.	1.5	USEPA 1997, Table 5-23, mean value for outdoor worker, moderate activities.
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	NA		NA	
Skin Surface Area	SA	cm ²	NA		NA	
Skin Surface Area Available for Contact	SA	cm ² /day	3300	USEPA 2002; Exhibit 1-2, as outdoor worker	3300	USEPA 2002; Exhibit 1-2, as outdoor worker
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.2	USEPA, 2004; Exhibit 3-3, 95th percentile for staged activity pipe layer (dry soil)	0.07	USEPA, 2004; Exhibit 3-3, geometric mean for staged activity pipe layer (dry soil)
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	NA		NA	
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	NA		NA	
Exposure Time	ET	hours/day	2	BPJ, assumes only 25% of 8-hour day is spend onsite, comment 4.3RME(c)	2	BPJ, assumes only 25% of 8-hour day is spend onsite, comment 4.3RME(c)
Event Duration	t _{event}	hr/event	NA		NA	
Lag Time per Event	t _{event}	hr/event	NA		NA	
Time to Reach Steady-State	t*	hr	NA		NA	
Event Frequency	EV	event/day	NA		NA	
Exposure Frequency	EF	days/year	188	BPJ, assumes 250 days/year x 25% snow coverage throughout the year (comment 6 of 5/9/08 comment letter)	164	BPJ, assumes 219 days/year x 25% snow coverage throughout the year (comment 6 of 5/9/08 comment letter)
Exposure Duration	ED	years	25	USEPA 2004, Exhibit 3-5, RME for industrial workers	9	USEPA 2004, Exhibit 3-5, CT for industrial workers
Body Weight	BW	kg	70	USEPA 1997; Table 7-11	70	USEPA 1997; Table 7-11
Averaging Time - Non-Cancer	AT-NC	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	NA		NA	

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Utility Worker		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	330	USEPA 2002; Exhibit 1-2, as construction worker	100	USEPA 2002; Exhibit 1-2, as outdoor worker
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1.5	USEPA 1997, Table 5-23, mean value for outdoor worker, moderate activities.	1.5	USEPA 1997, Table 5-23, mean value for outdoor worker, moderate activities.
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area	SA	cm ²	3300	USEPA 2002; Exhibit 1-2, as construction worker	3300	USEPA 2002; Exhibit 1-2, as construction worker
Skin Surface Area Available for Contact	SA	cm ² /day	3300	USEPA 2002; Exhibit 1-2, as construction worker	3300	USEPA 2002; Exhibit 1-2, as construction worker
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.3	USEPA, 2004; Exhibit 3-3, 95th percentile for construction workers	0.2	USEPA, 2004; Exhibit 3-3, geometric mean for utility workers
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	0.9	USEPA, 2004; Exhibit 3-3, 95th percentile for utility workers	0.2	USEPA, 2004; Exhibit 3-3, geometric mean for utility workers
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)
Event Duration	t _{event}	hr/event	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)
Lag Time per Event	t _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	20	MADEP, 1995	5	BPJ, see comment 4.14
Exposure Duration	ED	years	25	USEPA 2004, Exhibit 3-5, RME for industrial workers	9	USEPA 2004, Exhibit 3-5, CT for industrial workers
Body Weight	BW	kg	70	USEPA 1997; Table 7-11	70	USEPA 1997; Table 7-11
Averaging Time - Non-Cancer	AT-NC	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Older Child Trespasser (12 to < 18 years old)		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	100	USEPA 2002; Exhibit 1-2, as outdoor worker	100	USEPA 2002; Exhibit 1-2, as outdoor worker
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1.2	USEPA 1997, Table 5-23, mean value for children, moderate activities.	1.2	USEPA 1997, Table 5-23, mean value for children, moderate activities.
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	0.5	BPJ, half of the RME given that the geographic range could be much larger than the site
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area	SA	cm ²	5400	NYSDEC 2002, Onondaga Lake HHRA	5400	NYSDEC 2002, Onondaga Lake HHRA
Skin Surface Area Available for Contact	SA	cm ² /day	5400	NYSDEC 2002, Onondaga Lake HHRA	5400	NYSDEC 2002, Onondaga Lake HHRA
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(e)	0.2	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(b)
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(e)	0.2	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(b)
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Event Duration	t _{event}	hr/event	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Lag Time per Event	t _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	42	BPJ, Assumes site access 2 days per week during the summer months and 1 days per week when school is in session (2X10 + 1X22) = 42	32	BPJ, (NYSDEC/TAMS Ninemile Creek HHRA), see comment 4.13(b)
Exposure Duration	ED	years	6	USEPA, 2004; Exhibit 3-2 (RME exposure duration for child resident)	6	USEPA, 2004; Exhibit 3-2 (CT exposure duration for child resident)
Body Weight	BW	kg	56	USEPA 1997; Table 7-3, mean body weight for boys and girls age 12 to 17	56	USEPA 1997; Table 7-3, mean body weight for boys and girls age 12 to 17
Averaging Time - Non-Cancer	AT-NC	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	2190	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Cooking Loss (PCBs and PCDD/PCDFs only)	CL	unitless	NA		0.33	USEPA 1997, Section 10.9
Exposure Frequency (For Fish Consumption)	EF _{fish}	days/year	365	USEPA 1997, Page 10-26	365	USEPA 1997, Page 10-26
Fraction Ingested from Fish	FI _{fish}	unitless	1	BPJ, assumes all fish consumed comes from onsite.	1	BPJ, assumes all fish consumed comes from onsite.
Ingestion Rate (For Fish Consumption)	IR _{fish}	g fish/day	16.7	USEPA 1997, 10-26. 2/3 of the Adult value to account for difference in body weight.	5.3	USEPA 1997, 10-26. 2/3 of the Adult value to account for difference in body weight.

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Adult Trespasser (> 18 years old)		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	50	USEPA 1997; Table 4-23 (mean value for adults)	50	USEPA 1997; Table 4-23 (mean value for adults)
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1.6	USEPA 1997; Table 5-23 (mean value for adults, moderate activities)	1	USEPA 1997; Table 5-23 (mean value for adults, light activities)
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	0.5	BPJ, half of the RME given that the geographic range could be much larger than the site
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area	SA	cm ²	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA
Skin Surface Area Available for Contact	SA	cm ² /day	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(d)	0.15	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(a)
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	0.3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(d)	0.15	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(a)
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Event Duration	t _{event}	hr/event	4	BPJ, based on an estimated 4 hours of recreational activities per day	2	BPJ, half the RME of 4 hours of recreational activities per day
Lag Time per Event	t _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	42	BPJ, Assumes site access 2 days per week during the summer months and 1 days per week when school is in session (2X10 + 1X22) = 42	32	BPJ, (NYSDEC/TAMS Ninemile Creek HHRA), see comment 4.13(b)
Exposure Duration	ED	years	30	USEPA, 2004; Exhibit 3-2 (RME exposure duration for adult resident)	9	USEPA, 2004; Exhibit 3-2 (CT exposure duration for adult resident)
Body Weight	BW	kg	70	USEPA 1991, Section 6.0 Summary Table	70	USEPA 1991, Section 6.0 Summary Table
Averaging Time - Non-Cancer	AT-NC	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Cooking Loss (PCBs and PCDD/PCDFs only)	CL	unitless	NA		0.33	USEPA 1997, Section 10.9
Exposure Frequency (For Fish Consumption)	EF _{fish}	days/year	365	USEPA 1997, Page 10-26	365	USEPA 1997, Page 10-26
Fraction Ingested from Fish	FI _{fish}	unitless	1	BPJ, assumes all fish consumed comes from onsite.	1	BPJ, assumes all fish consumed comes from onsite.
Ingestion Rate (For Fish Consumption)	IR _{fish}	g fish/day	25	USEPA, 1997; Page 10-26	8	USEPA, 1997; Page 10-26

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Commercial/Industrial Worker		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	100	USEPA 2002; Exhibit 1-2, as outdoor worker	50	USEPA 1991; Section 6.0 Summary Table
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1.6	USEPA 1997, Table 5-23, mean value for adult, moderate activities.	1.6	USEPA 1997, Table 5-23, mean value for adult, moderate activities.
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	NA		NA	
Skin Surface Area	SA	cm ²	NA		NA	
Skin Surface Area Available for Contact	SA	cm ² /day	3300	USEPA 2002; Exhibit 1-2, as outdoor worker	3300	USEPA 2002; Exhibit 1-2, as outdoor worker
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	0.3	USEPA 2004, Exhibit 3-3, In response to comment 4.3RME(i)	0.1	USEPA 2004, Exhibit 3-3, In response to comment 4.3CT(c)
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	NA		NA	
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	NA		NA	
Exposure Time	ET	hours/day	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)
Event Duration	t _{event}	hr/event	NA		NA	
Lag Time per Event	t _{event}	hr/event	NA		NA	
Time to Reach Steady-State	t*	hr	NA		NA	
Event Frequency	EV	event/day	NA		NA	
Exposure Frequency	EF	days/year	250	USEPA, 2004, Exhibit 3-5, RME for industrial worker	219	USEPA 2004, Exhibit 3-5, CT for industrial worker
Exposure Duration	ED	years	25	USEPA 2004, Exhibit 3-5, RME for industrial workers	9	USEPA 2004, Exhibit 3-5, CT for industrial workers
Body Weight	BW	kg	70	USEPA 1997; Table 7-11	70	USEPA 1997; Table 7-11
Averaging Time - Non-Cancer	AT-NC	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	NA		NA	

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

		Receptor:		Drainage Ditch Worker		
Parameter		Units	RME	Source/Rationale	CT	Source/Rationale
Ingestion Rate of Soil	IR	mg/day	330	USEPA 2002; Exhibit 1-2, NYSDEC 2002, Onondaga Lake HHRA	330	USEPA 2002; Exhibit 1-2, NYSDEC 2002, Onondaga Lake HHRA
Ingestion Rate of Water	IR	L/day	NA		NA	
Inhalation Rate	InR	m ³ /hr	1.5	USEPA 1997, Table 5-23, mean value for outdoor worker, heavy activities.	1.5	USEPA 1997, Table 5-23, mean value for outdoor worker, heavy activities.
Fraction Ingested from Soil	FI	unitless	1	BPJ, assumes entire dose comes from onsite	1	BPJ, assumes entire dose comes from onsite
Fraction Absorbed	FA	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Skin Surface Area	SA	cm ²	3300	USEPA 2002; Exhibit 1-2, as construction worker	3300	USEPA 2002; Exhibit 1-2, as construction worker
Skin Surface Area Available for Contact	SA	cm ² /day	3300	USEPA 2002; Exhibit 1-2, as construction worker	3300	USEPA 2002; Exhibit 1-2, as construction worker
Soil to Skin Adherence Factor (Soil)	AF	mg/cm ³	NA		NA	
Soil to Skin Adherence Factor (Sediment)	AF	mg/cm ³	0.9	USEPA, 2004; Exhibit 3-3, 95th percentile for utility workers	0.2	USEPA, 2004; Exhibit 3-3, geometric mean for utility workers
Dermal Absorption Factor	ABS	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	Chemical Specific	USEPA, 2004; Exhibit 3-4
Permeability Constant	K _p	cm/hour	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Exposure Time	ET	hours/day	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)
Event Duration	t _{event}	hr/event	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)	8	BPJ, assumes a standard 8-hour work day (USEPA 1991, Section 1.2 for commercial/industrial workers)
Lag Time per Event	T _{event}	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Time to Reach Steady-State	t*	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4
Event Frequency	EV	event/day	1	USEPA 2004, Exhibit A-9	1	USEPA 2004, Exhibit A-9
Exposure Frequency	EF	days/year	10	BPJ, ditch worker could access the site as often as 10 days/year	5	BPJ, ditch worker typically accesses the site only 5 days/year
Exposure Duration	ED	years	25	USEPA 2004, Exhibit 3-5, RME for industrial workers	9	USEPA 2004, Exhibit 3-5, CT for industrial workers
Body Weight	BW	kg	70	USEPA 1997; Table 7-11	70	USEPA 1997; Table 7-11
Averaging Time - Non-Cancer	AT-NC	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	3285	USEPA 1989, Exhibits 6-11 through 6-16
Averaging Time - Cancer	AT-C	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	25550	USEPA 1989, Exhibits 6-11 through 6-16
Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	B	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4

APPENDIX G
EXPOSURE PARAMETERS SUMMARY
Honeywell, Wastebed B/Harbor Brook Site, Geddes and Syracuse, New York

Notes:

BPJ = Best Professional Judgement
NA = Not Applicable

References:

Massachusetts Department of Environmental Protection (MADEP). 1995. *Guidance for Disposal Site Risk Characterization - in Support of the Massachusetts Contingency Plan (Interim final policy)*. BWSC/ORS-95-141.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

ATTACHMENT A

**RAGS D Table Series 1 through 7, 9,
and 10**

RAGS Table 1 Series

TABLE 1.1
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 1 - SITE-WIDE^a
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Soil (0-2 ft bgs)	Surface Soil	Site-wide Surface Soil	Trespasser	Older Child (Age 12 to <18)	Ingestion	Quantitative	There is potential for trespassers to incidentally ingest soil.
						Dermal	Quantitative	There is potential for trespassers to have dermal exposure to soil.
					Adult (Age >18)	Ingestion	Quantitative	There is potential for trespassers to incidentally ingest soil.
						Dermal	Quantitative	There is potential for trespassers to have dermal exposure to soil.
		Air	Ambient Air - Fugitive Dust	Trespasser	Older Child (Age 12 to <18)	Inhalation	Quantitative	There is potential for trespassers to inhale fugitive dusts.
					Adult (Age >18)	Inhalation	Quantitative	There is potential for trespassers to inhale fugitive dusts.
			Ambient Air -Volatile Emissions	Trespasser	Older Child (Age 12 to <18)	Inhalation	Quantitative	There is potential for trespassers to inhale vapors.
					Adult (Age >18)	Inhalation	Quantitative	There is potential for trespassers to inhale vapors.
	Surface and Subsurface Soil (0-10 ft bgs)	Surface and Subsurface Soil	Site-wide Surface and Subsurface Soil	Utility Worker	Adult (Age >18)	Ingestion	Quantitative	Utility workers could incidentally ingest soil to a depth of approximately 10 ft bgs repairing or installing on-site utilities.
						Dermal	Quantitative	Utility workers could have dermal exposure to soil to a depth of approximately 10 ft bgs repairing or installing on-site utilities.
		Air	Ambient Air - Fugitive Dust	Utility Worker	Adult (Age >18)	Inhalation	Quantitative	Utility workers could inhale dust originating from soil excavations as part of repairing or installing on-site utilities.
			Ambient Air -Volatile Emissions	Utility Worker	Adult (Age >18)	Inhalation	Quantitative	Utility workers could inhale vapors originating from soil excavations as part of repairing or installing on-site utilities.
	Surface Sediment (0-1 ft)	Surface Sediment	Site-wide Surface Sediment	Trespasser	Older Child (Age 12 to <18)	Ingestion	Quantitative	There is potential for trespassers to incidentally ingest surface sediment.
						Dermal	Quantitative	There is potential for trespassers to have dermal exposure to surface sediment.
					Adult (Age >18)	Ingestion	Quantitative	There is potential for trespassers to incidentally ingest surface sediment.
						Dermal	Quantitative	There is potential for trespassers to have dermal exposure to surface sediment.
	Surface and Subsurface Sediment (0-10 ft) ^b	Surface and Subsurface Sediment	Site-wide Surface and Subsurface Sediment	Utility Worker	Adult (Age >18)	Ingestion	Quantitative	Utility workers could incidentally ingest sediment during excavations as part activities related to on-site utilities.
						Dermal	Quantitative	Utility workers could have dermal exposure to sediment during excavations as part of activities related to on-site utilities.
	Surface Water	Surface Water	Site-wide Surface Water	Trespasser	Older Child (Age 12 to <18)	Dermal	Quantitative	Trespasser could have dermal exposure to surface water.
					Adult (Age >18)	Dermal	Quantitative	Trespasser could have dermal exposure to surface water.
				Utility Worker	Adult (Age >18)	Dermal	Quantitative	Utility workers could have dermal exposure to surface water during excavations as part of activities related to on-site utilities.

TABLE 1.1
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 1 - SITE-WIDE^a
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future (cont'd)	Onondaga Lake Fish Tissue ^c	Fish Tissue	Onondaga Lake Fish Tissue	Trespasser ^d	Older Child (Age 12 to <18)	Ingestion	Quantitative	Trespasser could ingest fish if recreational angling is practiced unlawfully.
					Adult (Age >18)	Ingestion	Quantitative	Trespasser could ingest fish if recreational angling is practiced unlawfully.
	Shallow Ground Water (0-10 ft bgs)	Shallow Ground Water	Site-wide Shallow Ground Water	Utility Worker	Adult (Age >18)	Ingestion	None	Incidental ingestion of shallow ground water present during excavations as part of repairing or installing on-site utilities is expected to be <i>de minimis</i> .
						Dermal	Quantitative	Utility workers could have dermal exposure to shallow ground water present during excavations as part of repairing or installing on-site utilities.
Future	Surface and Subsurface Soil (0-10 ft bgs)	Surface and Subsurface Soil	Site-wide Surface and Subsurface Soil	Construction Worker	Adult (Age >18)	Ingestion	Quantitative	Future construction workers could incidentally ingest soil to a depth of approximately 10 ft bgs as part of construction projects.
						Dermal	Quantitative	Future construction workers could have dermal exposure to soil to a depth of approximately 10 ft bgs as part of construction projects.
		Air	Ambient Air - Fugitive Dust	Construction Worker	Adult (Age >18)	Inhalation	Quantitative	Future construction workers could inhale dust originating from soil excavations as part of construction projects.
			Ambient Air - Volatile Emissions	Construction Worker	Adult (Age >18)	Inhalation	Quantitative	Future construction workers could inhale vapors originating from soil excavations as part of construction projects.
	Surface and Subsurface Sediment (0-1 ft) ^b	Surface and Subsurface Sediment	Site-wide Surface and Subsurface Sediment	Construction Worker	Adult (Age >18)	Ingestion	Quantitative	Construction workers could incidentally ingest sediment while conducting activities.
					Adult (Age >18)	Dermal	Quantitative	Construction workers could have dermal contact with sediment while conducting activities.
	Surface Water	Surface Water	Site-wide Surface Water	Construction Worker	Adult (Age >18)	Dermal	Quantitative	Construction workers could have dermal contact with surface water while conducting activities.
	Shallow Ground Water (0-10 ft bgs)	Shallow Ground Water	Site-wide Shallow Ground Water	Construction Worker	Adult (Age >18)	Dermal	Quantitative	Future construction workers could have dermal exposure to shallow ground water present during excavations as part of construction projects.

Notes:

a = Site wide designation does not include State wetland SYW-12 area, which is evaluated separately in this assessment (see Table 1.9).

b = Where construction or utility workers have may contact with the sediment of Harbor Brook, a depth interval of 0 - 10 ft bgs is applied. This reflects the potential for contact with deeper sediments for bridge reconstruction, which is anticipated and unique to the Harbor Brook exposure area. In a few instances, sediment samples with start depths of 0 ft and end depths ranging from >1 to 3 ft were also incorporated in the evaluation of surface sediment.

c = Fish tissue collected from Onondaga Lake is used herein, given the lack of available fish tissue data from Harbor Brook but recognizing the hydrologic connection between Harbor Brook and Onondaga Lake.

d = Recreation is not currently allowed; a trespasser is therefore evaluated in current scenario. Trespassing includes the fish ingestion pathway and will therefore be protective of a recreator.

References:

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

TABLE 1.2
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 2 - HARBOR BROOK, LAKESHORE AREA, EAST FLUME, DSA #1, AND DSA #2
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Soil (0-2 ft bgs) ^a	Surface Soil	EU-2 Surface Soils	Surveillance Worker	Adult (Age >18)	Ingestion	Quantitative	A surveillance worker may incidentally ingest surface soil while performing his/her duties.
						Dermal	Quantitative	A surveillance worker may have dermal exposure to soil while performing his/her duties.
		Air	Ambient Air - Fugitive Dust	Surveillance Worker	Adult (Age >18)	Inhalation	Quantitative	Surveillance workers could inhale fugitive dust.
			Ambient Air -Volatile Emissions	Surveillance Worker	Adult (Age >18)	Inhalation	Quantitative	Surveillance workers could inhale vapors originating from soil.

Notes:

a = Exposure to surface soil is not limited to vehicle paths of travel; soil data from the entire exposure unit is used to evaluate risk to the surveillance worker.

TABLE 1.3
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 3 - INTERSTATE 690 DRAINAGE DITCH
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Water	Storm Water	I-690 Drainage Ditch	Ditch Worker	Adult (Age >18)	Ingestion	None	Incidental ingestion of surface (storm) water is expected to be <i>de minimis</i> .
						Dermal	Quantitative	A drainage ditch worker may be dermally exposed to surface (storm) water while performing his/her duties.
	Sediment (0-1 ft bgs)	Sediment	I-690 Drainage Ditch Sediment	Ditch Worker	Adult (Age >18)	Ingestion	Quantitative	A ditch worker may incidentally ingest sediment while performing his/her duties.
						Dermal	Quantitative	A ditch worker may have dermal exposure to sediment while performing his/her duties.
		Air	Ambient Air -Volatile Emissions	Ditch Worker	Adult (Age >18)	Inhalation	Quantitative	Due to the ephemeral nature of the I-690 drainage ditch, periods of time where sediment is exposed are possible. Inhalation of volatile compounds originating from sediment could occur.

TABLE 1.4
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 4 - RAILROAD AREA
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Soil (0-2 ft bgs)	Surface Soil	EU-4 Surface Soils	Railroad Worker	Adult (Age >18)	Ingestion	Quantitative	A railroad worker may incidentally ingest soil while performing his/her duties.
						Dermal	Quantitative	A railroad worker may have dermal exposure to soil while performing his/her duties.
		Air	Ambient Air - Fugitive Dust	Railroad Worker	Adult (Age >18)	Inhalation	Quantitative	A railroad worker could inhale fugitive dust while performing his/her duties.
			Ambient Air -Volatile Emissions	Railroad Worker	Adult (Age >18)	Inhalation	Quantitative	A railroad worker could inhale vapors while performing his/her duties.

TABLE 1.5
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 5 - PENN-CAN PROPERTY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Soil (0-2 ft bgs)	Surface Soil	EU-5 Surface Soils	Commercial/ Industrial Worker	Adult (Age >18)	Ingestion	Quantitative	A commercial/industrial worker may incidentally ingest soil while performing his/her duties.
						Dermal	Quantitative	A commercial/industrial worker may have dermal exposure to soil while performing his/her duties.
		Air	Ambient Air - Fugitive Dust	Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	A commercial/industrial worker could inhale fugitive dust while performing his/her duties outside.
			Ambient Air - Volatile Emissions	Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	A commercial/industrial worker could inhale vapors while performing his/her duties outside.
	Surface and Subsurface Soil (0-10 ft bgs)	Air	Indoor Air - Vapor Intrusion	Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	Vapors originating from soil VOCs may enter building workspace. When soil vapor data is available, detected constituents are evaluated using the framework presented in USEPA (2004) Developing Indoor Air Decision Matrices for Screening and Interim Actions.
	Shallow Ground Water (0-10 ft bgs)	Air	Indoor Air - Vapor Intrusion	Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	Constituents in ground water also have the potential to migrate to the occupational workspace. When sub-surface soil vapor data is unavailable, ground water data will be screened with respect to USEPA OSWER (2002) ground water to indoor air criteria.

References:

USEPA. 2002. OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Ground Water and Soils (Subsurface Vapor Intrusion Guidance) November 2002 EPA530-D-02-004

USEPA. 2004. Developing Indoor Air Decision Matrices for Screening and Interim Actions. Region II. Final Draft. July.

TABLE 1.6
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 6 - HARBOR BROOK, LAKESHORE AREA, EAST FLUME, DSA #1, DSA #2, AND AOS #1
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Surface Soil (0-2 ft bgs)	Surface Soil	EU-6 Surface Soils	Recreational Visitor	Adult (Age >18)	Ingestion	Quantitative	The potential exists for future recreational visitors to incidentally ingest surface soil.
						Dermal	Quantitative	The potential exists for future recreational visitors to have dermal contact with surface soil.
					Child (Age 0 to <6)	Ingestion	Quantitative	The potential exists for future recreational visitors to incidentally ingest surface soil.
						Dermal	Quantitative	The potential exists for future recreational visitors to have dermal contact with surface soil.
				Resident	Adult (Age >18)	Ingestion	Quantitative	Although residential use of the Site is not expected, the potential for future residents to incidentally ingest surface soil will be evaluated in the analysis of uncertainty.
						Dermal	Quantitative	Although residential use of the Site is not expected, the potential for future residents to have dermal contact with surface soil will be evaluated in the analysis of uncertainty.
					Child (Age 0 to <6)	Ingestion	Quantitative	Although residential use of the Site is not expected, the potential for future residents to incidentally ingest surface soil will be evaluated in the analysis of uncertainty.
						Dermal	Quantitative	Although residential use of the Site is not expected, the potential for future residents to have dermal contact with surface soil will be evaluated in the analysis of uncertainty.
		Air	Ambient Air - Fugitive Dust	Recreational Visitor	Adult (Age >18)	Inhalation	Quantitative	There is potential for a recreational visitor to inhale fugitive dust.
					Child (Age 0 to <6)	Inhalation	Quantitative	There is potential for a recreational visitor to inhale fugitive dust.
				Resident	Adult (Age >18)	Inhalation	Quantitative	Residential use of the Site is not expected. Nonetheless, potential inhalation of fugitive dust by a resident will be evaluated in the analysis of uncertainty.
					Child (Age 0 to <6)	Inhalation	Quantitative	Residential use of the Site is not expected. Nonetheless, potential inhalation of fugitive dust by a resident will be evaluated in the analysis of uncertainty.
			Ambient Air - Volatile Emissions	Recreational Visitor	Adult (Age >18)	Inhalation	Quantitative	There is potential for a recreational visitor to inhale vapors.
					Child (Age 0 to <6)	Inhalation	Quantitative	There is potential for a recreational visitor to inhale vapors.
				Resident	Adult (Age >18)	Inhalation	Quantitative	Residential use of the Site is not expected. Nonetheless, potential inhalation of vapors by a resident will be evaluated in the analysis of uncertainty.
					Child (Age 0 to <6)	Inhalation	Quantitative	Residential use of the Site is not expected. Nonetheless, potential inhalation of vapors by a resident will be evaluated in the analysis of uncertainty.

TABLE 1.6
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 6 - HARBOR BROOK, LAKESHORE AREA, EAST FLUME, DSA #1, DSA #2, AND AOS #1
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future (cont'd)	Surface and Subsurface Soil (0-10 ft bgs)	Air	Indoor Air -Vapor Intrusion	Resident	Adult (Age >18)	Inhalation	Quantitative	Residential use of the Site is not anticipated. However, vapors originating from soil VOCs may enter residential buildings, if they were to exist. When soil data is available, detected constituents are evaluated using the framework presented in USEPA (2004) Developing Indoor Air Decision Matrices for Screening and Interim Actions.
					Child (Age 0 to <6)	Inhalation	Quantitative	Residential use of the Site is not anticipated. However, vapors originating from soil VOCs may enter residential buildings, if they were to exist. When soil data is available, detected constituents are evaluated using the framework presented in USEPA (2004) Developing Indoor Air Decision Matrices for Screening and Interim Actions.
	Surface Sediment (0-1 ft bgs)	Surface Sediment	EU-6 Surface Sediment	Recreational Visitor	Adult (Age >18)	Ingestion	Quantitative	The potential exists for future recreational visitors to incidentally ingest surface sediment.
						Dermal	Quantitative	The potential exists for future recreational visitors to have dermal contact with surface sediment.
					Child (Age 0 to <6)	Ingestion	Quantitative	The potential exists for future recreational visitors to incidentally ingest surface sediment.
						Dermal	Quantitative	The potential exists for future recreational visitors to have dermal contact with surface sediment.
	Surface Water	Surface Water	EU-6 Surface Water	Recreational Visitor	Adult (Age >18)	Ingestion	None	Incidental ingestion of surface water is expected to be <i>de minimis</i> .
						Dermal	Quantitative	The potential exists for future recreational visitors to have dermal contact with surface water.
					Child (Age 0 to <6)	Ingestion	None	Incidental ingestion of surface water is expected to be <i>de minimis</i> .
						Dermal	Quantitative	The potential exists for future recreational visitors to have dermal contact with surface water.
	Onondaga Lake Fish Tissue ^a	Fish Tissue	Onondaga Lake Fish Tissue	Recreational Visitor	Adult (Age >18)	Ingestion	Quantitative	The potential exists for future recreational visitors to eat fish caught in surface water bodies adjacent to the Site.
					Child (Age 0 to <6)	Ingestion	Quantitative	The potential exists for future recreational visitors to eat fish caught in surface water bodies adjacent to the Site.

TABLE 1.6
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 6 - HARBOR BROOK, LAKESHORE AREA, EAST FLUME, DSA #1, DSA #2, AND AOS #1
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future (cont'd)	Shallow Ground Water (0-10 ft bgs)	Air	Indoor Air -Vapor Intrusion	Resident	Adult (Age >18)	Inhalation	Quantitative	Residential use of the Site is not anticipated. However, vapors originating from ground water VOCs may enter residential buildings, if they were to exist. Constituents in ground water also have the potential to migrate to the occupational workspace. When sub-surface soil vapor data is unavailable, ground water data will be evaluated with respect to USEPA OSWER (2002) ground water to indoor air criteria.
					Child (Age 0 to <6)	Inhalation	Quantitative	Residential use of the Site is not anticipated. However, vapors originating from ground water VOCs may enter residential buildings, if they were to exist. Constituents in ground water also have the potential to migrate to the occupational workspace. When sub-surface soil vapor data is unavailable, ground water data will be evaluated with respect to USEPA OSWER (2002) ground water to indoor air criteria.

a = Fish tissue collected from Onondaga Lake is used herein, given the lack of available fish tissue data from Harbor Brook but recognizing the hydrologic connection between Harbor Brook and Onondaga Lake.

References:

USEPA. 2002. OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Ground Water and Soils (Subsurface Vapor Intrusion Guidance) November 2002 EPA530-D-02-004

USEPA. 2004. Developing Indoor Air Decision Matrices for Screening and Interim Actions. Region II. Final Draft. July.

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

TABLE 1.7
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 7 - PENN-CAN PROPERTY, LAKESHORE AREA, DSA #1, DSA #2, AOS #1, AND AOS #2
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Surface Soil (0-2 ft bgs)	Surface Soil	EU-7 Surface Soils	Commercial/Industrial Worker	Adult (Age >18)	Ingestion	Quantitative	A commercial/industrial worker may incidentally ingest soil while performing his/her duties.
					Adult (Age >18)	Dermal	Quantitative	A commercial/industrial worker may have dermal exposure to soil while performing his/her duties.
		Air	Ambient Air - Fugitive Dust	Commercial/Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	A commercial/industrial worker could inhale fugitive dust while performing his/her duties outside.
			Ambient Air - Volatile Emissions	Commercial/Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	A commercial/industrial worker could inhale vapors while performing his/her duties outside.
	Surface and Subsurface Soil (0-10 ft bgs)	Air	Indoor Air - Vapor Intrusion	Commercial/Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	Vapors originating from soil VOCs may enter building workspace. When soil vapor data is available, detected constituents are screened using the framework presented in USEPA (2004) Developing Indoor Air Decision Matrices for Screening and Interim Actions.
	Shallow Ground Water (0-10 ft bgs)	Air	Indoor Air - Vapor Intrusion	Commercial/Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	Constituents in ground water also have the potential to migrate to the occupational workspace. When sub-surface soil vapor data is unavailable, ground water data will be evaluated with respect to USEPA OSWER (2002) ground water to indoor air criteria.

References:

USEPA. 2002. OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Ground Water and Soils (Subsurface Vapor Intrusion Guidance) November 2002 EPA530-D-02-004

USEPA. 2004. Developing Indoor Air Decision Matrices for Screening and Interim Actions. Region II. Final Draft. July.

TABLE 1.8
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 8 - SITE-WIDE GROUND WATER^a
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Ground Water	Drinking Water	Potable Water Sites	Resident	Child (Age 0 to <6)	Ingestion	Quantitative	This is a hypothetical scenario. The Site is zoned as industrial and is unlikely to be developed as a residential area. However, this pathway is being evaluated because the use designation for this aquifer is as a potable water supply and the Nation Contingency Plan states the ground water must be returned to its most beneficial use. Children may ingest ground water during the course of normal activities such drinking potable water.
						Dermal	Quantitative	This is a hypothetical scenario. The Site is zoned as industrial and is unlikely to be developed as a residential area. However, this pathway is being evaluated because the use designation for this aquifer is as a potable water supply and the Nation Contingency Plan states the ground water must be returned to its most beneficial use. Children may have dermal contact with ground water during the course of normal activities such as bathing/showering.
						Inhalation	Quantitative	This is a hypothetical scenario. The Site is zoned as industrial and is unlikely to be developed as a residential area. However, this pathway is being evaluated because the use designation for this aquifer is as a potable water supply and the Nation Contingency Plan states the ground water must be returned to its most beneficial use. Children may inhale vapors originating from potable ground water during bathing/showering.
					Adult (Age >18)	Ingestion	Quantitative	This is a hypothetical scenario. The Site is zoned as industrial and is unlikely to be developed as a residential area. However, this pathway is being evaluated because the use designation for this aquifer is as a potable water supply and the Nation Contingency Plan states the ground water must be returned to its most beneficial use. Adults may ingest ground water during the course of normal activities such drinking potable water.
						Dermal	Quantitative	This is a hypothetical scenario. The Site is zoned as industrial and is unlikely to be developed as a residential area. However, this pathway is being evaluated because the use designation for this aquifer is as a potable water supply and the Nation Contingency Plan states the ground water must be returned to its most beneficial use. Adults may have dermal contact with potable ground water during the course of normal activities such as bathing/showering.

TABLE 1.8
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 8 - SITE-WIDE GROUND WATER^a
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future (cont'd)	Ground Water (cont'd)	Drinking Water (cont'd)	Potable Water Sites (cont'd)	Resident (cont'd)	Adult (Age >18) (cont'd)	Inhalation	Quantitative	This is a hypothetical scenario. The Site is zoned as industrial and is unlikely to be developed as a residential area. However, this pathway is being evaluated because the use designation for this aquifer is as a potable water supply and the Nation Contingency Plan states the ground water must be returned to its most beneficial use. Adults may inhale vapors originating from potable ground water during bathing/showering.
				Commercial/Industrial Worker	Adult (Age >18)	Ingestion	Quantitative	This is a hypothetical scenario. The Site is zoned as industrial and it is unlikely that ground water will be used as a potable water source. However, this pathway is being evaluated because the use designation for this aquifer is as a potable water supply and the Nation Contingency Plan states the ground water must be returned to its most beneficial use.

a = Includes SYW-12

TABLE 1.9
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 9 - STATE WETLAND SYW-12
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Soil (0-2 ft bgs)	Surface Soil	EU-9 Surface Soils	Recreational Visitor	Child (Age 0 to <6)	Ingestion	Quantitative	There is potential for recreators to incidentally ingest surface soil. An Older Child Trespasser may also ingest surface soil at SYW-12. However, the Child Recreational Visitor is protective of the Older Child Trespasser.
						Dermal	Quantitative	There is potential for recreators to have dermal exposure to surface soil. An Older Child Trespasser may also have dermal exposure to surface soil at SYW-12. However, the Child Recreational Visitor is protective of the Older Child Trespasser.
					Adult (Age >18)	Ingestion	Quantitative	There is potential for recreators to incidentally ingest surface soil. An Adult Trespasser may also ingest surface soil at SYW-12. However, the Adult Recreational Visitor is protective of the Adult Trespasser.
						Dermal	Quantitative	There is potential for recreators to have dermal exposure to surface soil. An Adult Trespasser may also have dermal exposure to surface soil at SYW-12. However, the Adult Recreational Visitor is protective of the Adult Trespasser.
				Railroad Worker	Adult (Age >18)	Ingestion	Quantitative	There is potential for railroad workers to incidentally ingest surface soil.
						Dermal	Quantitative	There is potential for railroad workers to have dermal exposure to surface soil.
		Air	Ambient Air - Fugitive Dust	Recreational Visitor	Child (Age 0 to <6)	Inhalation	Quantitative	Recreators could inhale fugitive dust while visiting SYW-12. An Older Child Trespasser may also inhale fugitive dust while visiting SYW-12. However, the Child Recreational Visitor is protective of the Older Child Trespasser.
					Adult (Age >18)	Inhalation	Quantitative	Recreators could inhale fugitive dust while visiting SYW-12. An Adult Trespasser may also inhale fugitive dust while visiting SYW-12. However, the Adult Recreational Visitor is protective of the Adult Trespasser.
				Railroad Worker	Adult (Age >18)	Inhalation	Quantitative	A railroad worker could inhale fugitive dust while performing his/her duties.
			Ambient Air - Volatile Emissions	Recreational Visitor	Child (Age 0 to <6)	Inhalation	Quantitative	Recreators could inhale vapors while visiting SYW-12. An Older Child Trespasser may also inhale vapors while visiting SYW-12. However, the Child Recreational Visitor is protective of the Older Child Trespasser.
					Adult (Age >18)	Inhalation	Quantitative	Recreators could inhale vapors while visiting SYW-12. An Adult Trespasser may also inhale vapors while visiting SYW-12. However, the Adult Recreational Visitor is protective of the Adult Trespasser.
				Railroad Worker	Adult (Age >18)	Inhalation	Quantitative	A railroad worker could inhale vapors while performing his/her duties outside.

TABLE 1.9
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 9 - STATE WETLAND SYW-12
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future (Cont'd)	Surface and Subsurface Soil (0-10 ft bgs)	Surface and Subsurface Soil	EU-9 Surface and Subsurface Soil	Utility Worker	Adult (Age >18)	Ingestion	Quantitative	Utility workers could incidentally ingest soil to a depth of approximately 10 ft bgs repairing or installing on-site utilities.
						Dermal	Quantitative	Utility workers could have dermal exposure to soil to a depth of approximately 10 ft bgs repairing or installing on-site utilities.
	Surface and Subsurface Soil (0-10 ft bgs) (cont'd)	Air	Ambient Air - Fugitive Dust	Utility Worker	Adult (Age >18)	Inhalation	Quantitative	Utility workers could inhale dust originating from soil excavations as part of repairing or installing on-site utilities.
			Ambient Air - Volatile Emissions	Utility Worker	Adult (Age >18)	Inhalation	Quantitative	Utility workers could inhale vapors originating from soil excavations as part of repairing or installing on-site utilities.
	Shallow Ground Water (0-10 ft bgs)	Shallow Ground Water	EU-9 Shallow Ground Water	Utility Worker	Adult (Age >18)	Ingestion	None	Incidental ingestion of shallow ground water present during excavations as part of repairing or installing on-site utilities is expected to be <i>de minimis</i> .
						Dermal	Quantitative	Utility workers could have dermal exposure to shallow ground water present during excavations as part of repairing or installing on-site utilities.
Future	Surface Soil (0-2 ft bgs)	Surface Soil	EU-9 Surface Soils	Resident	Child (Age 0 to <6)	Ingestion	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, there is potential for them to incidentally ingest surface soil.
						Dermal	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, there is potential for them to have dermal contact with surface soil.
					Adult (Age >18)	Ingestion	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, there is potential for them to incidentally ingest surface soil.
						Dermal	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, there is potential for them to have dermal contact with surface soil.
				Commercial/ Industrial Worker	Adult (Age >18)	Ingestion	Quantitative	If future buildings were constructed, a commercial/industrial worker may incidentally ingest surface soil while performing his/her duties outside.
						Dermal	Quantitative	If future buildings were constructed, a commercial/industrial worker may have dermal exposure to surface soil while performing his/her duties outside.
		Air	Ambient Air - Fugitive Dust	Resident	Child (Age 0 to <6)	Inhalation	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, a resident could inhale fugitive dust during the course of his/her activities.
					Adult (Age >18)	Inhalation	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, a resident could inhale fugitive dust during the course of his/her activities.
				Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	A commercial/industrial worker could inhale fugitive dust while performing his/her duties outside.
						Inhalation	Quantitative	

TABLE 1.9
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 9 - STATE WETLAND SYW-12
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future (Cont'd)	Surface Soil (0-2 ft bgs) (cont'd)	Air (cont'd)	Ambient Air - Volatile Emissions	Resident	Child (Age 0 to <6)	Inhalation	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, a resident could inhale vapors during the course of his/her activities.
					Adult (Age >18)	Inhalation	Quantitative	Although residential use of the Site is not expected, if residential occupancy were to occur, a resident could inhale vapors during the course of his/her activities.
				Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	A commercial/industrial worker could inhale vapors while performing his/her duties outside.
	Surface and Subsurface Soil (0-10 ft bgs)	Surface and Subsurface Soil	EU-9 Surface and Subsurface Soil	Construction Worker	Adult (Age >18)	Ingestion	Quantitative	Construction workers could incidentally ingest soil to a depth of approximately 10 ft bgs while conducting activities.
						Dermal	Quantitative	Construction workers could have dermal exposure to soil to a depth of approximately 10 ft bgs while conducting activities.
		Air	Ambient Air - Fugitive Dust	Construction Worker	Adult (Age >18)	Inhalation	Quantitative	Construction workers could inhale dust originating from soil excavations as part of construction projects.
			Ambient Air -Volatile Emissions	Construction Worker	Adult (Age >18)	Inhalation	Quantitative	Construction workers could inhale vapors originating from soil excavations as part of construction projects.
			Indoor Air - Vapor Intrusion	Resident	Adult (Age >18)	Inhalation	Qualitative	Residential use of the Site is not anticipated. However, vapors originating from soil VOCs may enter residential buildings, if they were to exist. When soil data is available, detected constituents are evaluated using the framework presented in USEPA (2004) Developing Indoor Air Decision Matrices for Screening and Interim Actions.
					Child (Age 0 to <6)	Inhalation	Qualitative	Residential use of the Site is not anticipated. However, vapors originating from soil VOCs may enter residential buildings, if they were to exist. When soil data is available, detected constituents are evaluated using the framework presented in USEPA (2004) Developing Indoor Air Decision Matrices for Screening and Interim Actions.
				Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	Vapors originating from soil VOCs may enter building workspace. When soil data is available, detected constituents are screened using the framework presented in USEPA (2004) Developing Indoor Air Decision Matrices for Screening and Interim Actions.
		Shallow Ground Water	EU-9 Shallow Ground Water	Construction Worker	Adult (Age >18)	Ingestion	None	Incidental ingestion of shallow ground water present during excavations is expected to be <i>de minimis</i> .
						Dermal	Quantitative	Construction workers could have dermal exposure to shallow ground water present during excavations as part of construction projects.

TABLE 1.9
SELECTION OF EXPOSURE PATHWAYS
EXPOSURE UNIT 9 - STATE WETLAND SYW-12
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future (Cont'd)	Shallow Ground Water (0-10 ft bgs) (cont'd)	Air	Indoor Air - Vapor Intrusion	Resident	Adult (Age >18)	Inhalation	Quantitative	Residential use of the Site is not anticipated. However, vapors originating from ground water VOCs may enter residential buildings, if they were to exist. Constituents in ground water also have the potential to migrate to the occupational workspace. When sub-surface soil vapor data is unavailable, ground water data will be evaluated with respect to USEPA OSWER (2002) ground water to indoor air criteria.
					Child (Age 0 to <6)	Inhalation	Quantitative	Residential use of the Site is not anticipated. However, vapors originating from ground water VOCs may enter residential buildings, if they were to exist. Constituents in ground water also have the potential to migrate to the occupational workspace. When sub-surface soil vapor data is unavailable, ground water data will be evaluated with respect to USEPA OSWER (2002) ground water to indoor air criteria.
				Commercial/ Industrial Worker	Adult (Age >18)	Inhalation	Quantitative	Constituents in ground water also have the potential to migrate to the occupational workspace. When sub-surface soil vapor data is unavailable, ground water data will be evaluated with respect to USEPA OSWER (2002) ground water to indoor air criteria.

References:

USEPA. 2002. OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Ground Water and Soils (Subsurface Vapor Intrusion Guidance) November 2002 EPA530-D-02-004

USEPA. 2004. Developing Indoor Air Decision Matrices for Screening and Interim Actions. Region II. Final Draft. July.

RAGS Table 2 Series

TABLE 2.1a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Ground Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
Sitewide Ground Water	METALS																	
	7429-90-5	ALUMINUM	0.042 J	291 J	mg/L	HB-HB-04D	131/156	0.0439-4	2.91E+02		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	Y	ASL
	7440-36-0	ANTIMONY	0.0016 J	0.0058 J	mg/L	HB-GWS-08	7/170	0.0014-2.4	5.80E-03		6.00E-03	1.46E-03	N	1.46E-03	nc	1.46E-03	Y	ASL
	7440-38-2	ARSENIC	0.002 J	0.102	mg/L	HB-HB-04D	37/170	0.0016-0.4	1.02E-01		1.00E-02	4.46E-05	C	4.48E-05	ca	4.46E-05	Y	TOX
	7440-39-3	BARIUM	0.0015 J	20.3	mg/L	HB-HB-06S	166/171	0.003-0.02	2.03E+01		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	Y	ASL
	7440-41-7	BERYLLIUM	0.00012 J	0.0073 J	mg/L	HB-HB-13D	21/171	0.000076-0.4	7.30E-03		4.00E-03	7.30E-03	N	7.30E-03	nc	7.30E-03	Y	ASL
	7440-43-9	CADMIUM	0.00037 J	0.027	mg/L	HB-GWS-08	16/168	0.00024-0.4	2.70E-02		5.00E-03	1.83E-03	N	1.82E-03	nc	1.82E-03	Y	ASL
	7440-70-2	CALCIUM	0.19	7970	mg/L	HB-HB-06S	171/171	-	7.97E+03		NV	NV	NV	NV	NV	N	NUT	
	7440-47-3	CHROMIUM ⁶	0.0014 J	0.856	mg/L	HB-HB-17D	99/169	0.0011-0.2	8.56E-01		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX
	7440-48-4	COBALT	0.0029 J	0.133	mg/L	HB-HB-04D	14/169	0.00093-2	1.33E-01			NV	NV	7.30E-02	nc	7.30E-02	Y	ASL
	7440-50-8	COPPER	0.0015 J	1.23 J	mg/L	HB-HB-21I	83/170	0.00049-0.4	1.23E+00		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	Y	ASL
	57-12-5	CYANIDE	0.0101	0.533	mg/L	HB-HB-05I	50/167	0.01-0.02	5.33E-01		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	Y	ASL
	7439-89-6	IRON	0.0105 J	446 J	mg/L	HB-HB-04D	167/171	0.05-0.3	4.46E+02		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL
	7439-92-1	LEAD	0.0012 J	1.7	mg/L	HB-GWS-09	85/170	0.00066-0.4	1.70E+00		1.50E-02	NV	NV	NV	NV	1.50E-02	Y	ASL
	7439-95-4	MAGNESIUM	0.0393 J	925 J	mg/L	HB-HB-04D	164/171	0.08-1.5	9.25E+02			NV	NV	NV	NV	N	NUT	
	7439-96-5	MANGANESE	0.0023 J	16.1 J	mg/L	HB-HB-04D	146/171	0.0031-0.05	1.61E+01		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	Y	ASL
	7439-97-6	MERCURY ²	0.00003 J	0.0308	mg/L	HB-HB-05I	54/168	0.00017-0.0321	3.08E-02		2.00E-03	3.65E-04	N	3.65E-04	nc	3.65E-04	Y	ASL
	7440-02-0	NICKEL	0.0012 J	0.394	mg/L	HB-HB-13D	79/169	0.0031-2	3.94E-01			7.30E-02	N	7.30E-02	nc	7.30E-02	Y	ASL
	9177-44-0	POTASSIUM	1.2 J	580 J	mg/L	HB-HB-20D	167/172	0.81-110	5.80E+02			NV	NV	NV	NV	N	NUT	
	7782-49-2	SELENIUM	0.0019 J	0.022	mg/L	HB-GWS-09	18/168	0.0018-0.4	2.20E-02		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	Y	ASL
	7440-22-4	SILVER	0.00085 J	0.0245	mg/L	HB-HB-12S	16/170	0.00073-0.4	2.45E-02		1.00E-01	1.83E-02	N	1.82E-02	nc	1.82E-02	Y	ASL
	7440-23-5	SODIUM	0.27	75520	mg/L	HB-HB-20D	171/171		7.55E+04			NV	NV	NV	NV	N	NUT	
	7440-28-0	THALLIUM	0.0051 J	0.0883 J	mg/L	HB-HB-02I	4/169	0.002-0.8	8.83E-02		2.00E-03	2.56E-04	N	2.41E-04	nc	2.41E-04	Y	ASL
	7440-62-2	VANADIUM	0.00052 J	0.568	mg/L	HB-HB-04D	67/171	0.00039-2	5.88E-01			3.65E-03	N	3.65E-03	nc	3.65E-03	Y	ASL
	7440-66-6	ZINC	0.0011 J	1.9	mg/L	HB-GWS-08	78/171	0.0011-0.8	1.90E+00		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	Y	ASL
	PCBs																	
		HIGHLY CHLORINATED PCBs ^c	0.07 J	0.5 J	ug/l	HB-HP-07	3/161	0.07-1	5.00E-01			3.35E-02	C	3.36E-02	ca ^a	3.35E-02	Y	ASL
		TOTAL PCBs ^d	0.07 J	0.5 J	ug/l	HB-HP-07	3/161	0.07-1	5.00E-01			3.35E-02	C	3.36E-02	ca ^a	3.35E-02	Y	ASL
	PESTICIDES																	
	72-54-8	4,4'-DDD	0.015 J	2.2	ug/l	HB-WB-BU	5/161	0.093-2.1	2.20E+00			2.79E-01	C	2.80E-01	ca	2.79E-01	Y	ASL
	50-29-3	4,4'-DDT	0.018 J	20 J	ug/l	HB-HB-04S	3/160	0.093-2.1	2.00E+01			1.97E-01	C	1.98E-01	ca	1.97E-01	Y	ASL
	309-00-2	ALDRIN	0.03 J	0.17	ug/l	HB-WB-BU	3/161	0.047-1	1.70E-01			3.94E-03	C	3.95E-03	ca	3.94E-03	Y	ASL
	319-84-6	ALPHA-BHC	0.19 J	0.19 J	ug/l	HB-HB-12D	1/160	0.047-1	1.90E-01			1.06E-02	C	1.07E-02	ca	1.06E-02	Y	ASL
	57-74-9	TOTAL CHLORDANE ^e	0.002 J	0.002 J	ug/l	HB-HB-12I	1/161	0.047-1	2.00E-03			1.91E-01	C	1.92E-01	ca	1.91E-01	N	BSL
	33213-65-9	ENDOSULFAN II ^f	0.06 J	0.2	ug/l	HB-HB-01S	2/161	0.093-2.1	2.00E-01			NV	N	NV	nc	NV	Y	NTX
	1031-07-8	ENDOSULFAN SULFATE ^f	0.013 J	0.18 J	ug/l	HB-HB-14D	2/161	0.093-2.1	1.80E-01			NV	N	NV	nc	NV	Y	NTX
	72-20-8	ENDRIN	0.14	0.14	ug/l	HB-WB-BU	1/161	0.093-2.1	1.40E-01		2.00E-03	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	1024-57-3	HEPTACHLOR EPOXIDE	0.01 J	0.01 J	ug/l	HB-HP-06	1/161	0.047-1	1.00E-02		2.00E-04	7.36E-03	C	7.39E-03	ca	7.36E-03	Y	ASL
	SVOCs																	
	92-52-4	1,1'-BIPHENYL	1.2 J	83 J	ug/l	HB-HB-04S	12/56	10-54	8.30E+01			3.04E+01	N	3.04E+01	nc	3.04E+01	Y	ASL
	95-95-4	2,4,5-TRICHLOROPHENOL	2 J	7 J	ug/l	HB-HB-01S	2/167	9.3-5100	7.00E+00			3.65E+02	N	3.65E+02	nc	3.65E+02	N	BSL
	120-83-2	2,4-DICHLOROPHENOL	7 J	75	ug/l	HB-WA-03S	8/166	9.3-1000	7.50E+01			1.10E+01	N	1.09E+01	nc	1.09E+01	Y	ASL
	105-67-9	2,4-DIMETHYLPHENOL	1 J	38000	ug/l	HB-HB-12D	52/164	9.3-260	3.80E+04			7.30E+01	N	7.30E+01	nc	7.30E+01	Y	ASL
	95-57-8	2-CHLOROPHENOL	1 J	2 J	ug/l	HB-WA-03S	2/166	9.3-1000	2.00E+00			3.04E+00	N	3.04E+00	nc	3.04E+00	N	BSL
	91-57-6	2-METHYLNAPHTHALENE	1 J	9800	ug/l	HB-HB-04S	82/164	9.3-260	9.80E+03			2.43E+00	N	NV	NV	2.43E+00	Y	ASL
	95-48-7	2-METHYLPHENOL	1.2 J	15000	ug/l	HB-HB-13D	52/164	9.3-260	1.50E+04			1.83E+02	N	1.82E+02	nc	1.82E+02	Y	ASL
	88-75-5	2-NITROPHENOL	2.6 J	6 J	ug/l	HB-HP-02	3/166	9.3-1000	6.00E+00			NV	NV	NV	NV	NV	Y	NTX
	34METPH	3&4-METHYLPHENOL ^g	1 J	24000	ug/l	HB-HB-13D	59/110	9.3-260	2.40E+04			1.83E+01	N	1.82E+01	nc	1.82E+01	Y	ASL
	59-50-7	4-CHLORO-3-METHYLPHENOL	1 J	1 J	ug/l	HB-WA-08D	1/166	9.3-1000	1.00E+00			NV	NV	NV	NV	NV	Y	NTX
	106-44-5	4-METHYLPHENOL	1.8 J	30000	ug/l	HB-HB-13D	21/56	10-22	3.00E+04			1.83E+01	N	1.82E+01	nc	1.82E+01	Y	ASL
	100-02-7	4-NITROPHENOL	1.1 J	18 J	ug/l	HB-HB-11I	7/168	26-5200	1.80E+01			NV	NV	NV	NV	NV	Y	NTX
	83-32-9	ACENAPHTHENE	1 J	2200	ug/l	HB-HB-04S	68/166	9.3-1000	2.20E+03			3.65E+01	N	3.65E+01	nc	3.65E+01	Y	ASL

TABLE 2.1a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Ground Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	208-96-8	ACENAPHTHYLENE	1.2 J	2700	ug/l	HB-HB-04S	55/165	9.3-970	2.70E+03		3.00E-03	NV	NV	NV	NV	Y	NTX	
	120-12-7	ANTHRACENE	1 J	2000	ug/l	HB-HB-04S	37/166	9.3-1000	2.00E+03			1.83E+02	N	1.83E+02	nc	1.83E+02	Y	ASL
	1912-24-9	ATRAZINE	53	53	ug/l	HB-GWS-05	1/56	10-110	5.30E+01			3.04E-01	C	3.03E-01	ca	3.03E-01	Y	ASL
	100-52-7	BENZALDEHYDE	2.3 J	37	ug/l	HB-GWS-05	4/56	10-100	3.70E+01			3.65E+02	N	3.65E+02	nc	3.65E+02	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	1 J	700	ug/l	HB-HB-13D	23/167	9.3-1000	7.00E+02			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	50-32-8	BENZO(A)PYRENE	1.4 J	310 J	ug/l	HB-HB-04S	17/167	9.3-1000	3.10E+02		2.00E-04	3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	1.3 J	360	ug/l	HB-HB-13D	17/167	9.3-1000	3.60E+02			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	1.5 J	80 J	ug/l	HB-HB-13D	11/167	9.3-1000	8.00E+01			NV	NV	NV	NV	Y	NTX	
	207-08-9	BENZO(K)FLUORANTHENE	1.1 J	340 J	ug/l	HB-HB-04S	13/167	9.3-1000	3.40E+02			3.00E-01	C	9.21E-01	ca	3.00E-01	Y	ASL
	65-85-0	BENZOIC ACID	2 J	2300 J	ug/l	HB-HB-02S	13/30	50-5100	2.30E+03		6.00E-03	1.46E+04	N	1.46E+04	nc	1.46E+04	N	BSL
	100-51-6	BENZYL ALCOHOL	2 J	100	ug/l	HB-HB-05I	7/108	9.3-1000	1.00E+02			1.83E+03	N	1.09E+03	nc	1.09E+03	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1 J	110	ug/l	HB-HB-08S	54/168	1-1000	1.10E+02			4.78E+00	C	4.80E+00	ca	4.78E+00	Y	ASL
	105-60-2	CAPROLACTAM	9.6 J	42	ug/l	HB-GWS-05	5/56	10-110	4.20E+01			1.83E+03	N	1.82E+03	nc	1.82E+03	N	BSL
	86-74-8	CARBOLACTONE	1 J	930	ug/l	HB-HB-13D	56/162	9.3-260	9.30E+02			3.35E+00	C	3.36E+00	ca	3.35E+00	Y	ASL
	218-01-9	CHRYSENE	1 J	590 J	ug/l	HB-HB-04S	25/167	9.3-1000	5.90E+02			3.00E+00	C	9.21E+00	ca	3.00E+00	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	2 J	32 J	ug/l	HB-HB-13D	4/167	9.3-1000	3.20E+01			3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL
	132-64-9	DIBENZOFURAN	1 J	3400	ug/l	HB-HB-04S	59/166	9.3-970	3.40E+03			3.65E+00	N	1.22E+00	nc	1.22E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.6 J	3.7 J	ug/l	HB-HB-20I	7/167	9.3-1000	3.70E+00			3.65E+02	N	3.65E+02	nc	3.65E+02	N	BSL
	206-44-0	FLUORANTHENE	0.97 J	3200	ug/l	HB-HB-04S	47/166	9.3-1000	3.20E+03			1.46E+02	N	1.46E+02	nc	1.46E+02	Y	ASL
	86-73-7	FLUORENE	1 J	4200	ug/l	HB-HB-04S	64/166	9.3-970	4.20E+03		2.00E-01	2.43E+01	N	2.43E+01	nc	2.43E+01	Y	ASL
	87-68-3	HEXACHLOROBUTADIENE	1 J	1 J	ug/l	HB-HB-07S	1/199	1-1000	1.00E+00			8.59E-01	C	8.62E-01	ca	8.59E-01	Y	ASL
	193-39-5	INDENO(1,2,3-CD)PYRENE	1.2 J	110 J	ug/l	HB-HB-04S	12/167	9.3-1000	1.10E+02			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	91-20-3	NAPHTHALENE	0.3 J	35000	ug/l	HB-HB-04S	126/193	1-97	3.50E+04			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	98-95-3	NITROBENZENE	2.6 J	2.6 J	ug/l	HB-HB-11I	1/167	9.3-1000	2.60E+00			3.53E-01	N	3.40E-01	nc	3.40E-01	Y	ASL
	85-01-8	PHENANTHRENE	0.7 J	8300	ug/l	HB-HB-04S	68/166	9.3-1000	8.30E+03			NV	NV	NV	NV	Y	NTX	
	108-95-2	PHENOL	1 J	23000	ug/l	HB-HP-06	90/165	9.3-140	2.30E+04			1.10E+03	N	1.09E+03	nc	1.09E+03	Y	ASL
	129-00-0	PYRENE	1 J	1900	ug/l	HB-HB-04S	44/166	9.3-1000	1.90E+03			1.83E+01	N	1.83E+01	nc	1.83E+01	Y	ASL
	VOCs																	
	71-55-6	1,1,1-TRICHLOROETHANE	6	32 J	ug/l	HB-HB-12S	4/179	0.5-500	3.20E+01		2.00E-01	9.13E+02	N	3.17E+02	nc	3.17E+02	N	BSL
	75-34-3	1,1-DICHLOROETHANE	0.1 J	1.04	ug/l	HB-GWS-01	4/179	0.5-500	1.04E+00			8.96E+01	N	8.11E+01	nc	8.11E+01	N	BSL
	75-35-4	1,1-DICHLOROETHENE	0.1 J	0.1 J	ug/l	HB-HB-12S	1/179	0.5-500	1.00E-01			3.53E+01	N	3.39E+01	nc	3.39E+01	N	BSL
	87-61-6	1,2,3-TRICHLOROBENZENE	1 J	19	ug/l	HB-HB-01S	2/34	1-1000	1.90E+01		7.00E-03	NV	NV	NV	NV	Y	NTX	
	120-82-1	1,2,4-TRICHLOROBENZENE	1 J	468	ug/l	HB-WA-03S	12/199	1-1000	4.68E+02			6.08E+00	N	7.16E-01	nc	7.16E-01	Y	ASL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.1 J	900	ug/l	HB-HB-12I	27/35	0.5-0.5	9.00E+02			1.46E+00	N	1.23E+00	nc	1.23E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	0.12 J	7560	ug/l	HB-WA-03S	46/211	0.5-1000	7.56E+03		6.00E-01	2.68E+01	N	3.70E+01	nc	2.68E+01	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.2 J	320 J	ug/l	HB-HB-12I	23/35	0.5-500	3.20E+02			NV	NV	1.23E+00	nc	1.23E+00	Y	ASL
	541-73-1	1,3-DICHLOROBENZENE	3.6	62 J	ug/l	HB-WA-03S	13/212	0.5-1000	6.20E+01			1.83E+00	N	1.83E+01	nc	1.83E+00	Y	ASL
	106-46-7	1,4-DICHLOROBENZENE	0.1 J	8700	ug/l	HB-WA-03S	47/211	0.5-1000	8.70E+03		7.50E-02	2.81E-01	C	5.02E-01	ca	2.81E-01	Y	ASL
	78-93-3	2-BUTANONE	0	100 J	ug/l	HB-HB-05I	33/171	10-10000	1.00E+02			6.97E+02	N	6.97E+02	nc	6.97E+02	N	BSL
	591-78-6	2-HEXANONE	0	6.28	ug/l	HB-HB-20D	6/170	5-5000	6.28E+00			NV	NV	NV	NV	Y	NTX	
	106-43-4	4-CHLOROTOLUENE	2 J	2 J	ug/l	HB-HB-06S, HB-HB-05I	1/34	0.5-500	2.00E+00		5.00E-03	4.26E+01	N	NV	NV	4.26E+01	N	BSL
	108-10-1	4-METHYL-2-PENTANONE	0	3.2 J	ug/l	HB-HB-05I	11/171	5-5000	3.20E+00			6.28E+02	N	1.99E+02	nc	1.99E+02	N	BSL
	67-64-1	ACETONE	0	560	ug/l	HB-HB-05I	62/170	10-10000	5.60E+02			5.48E+02	N	5.48E+02	nc	5.48E+02	Y	ASL
	98-86-2	ACETOPHENONE	4.2 J	38	ug/l	HB-GWS-05	5/56	10-110	3.80E+01			6.08E+01	N	NV	NV	6.08E+01	N	BSL
	71-43-2	BENZENE	0.3 J	126000	ug/l	HB-HB-12D	93/178	0.5-250	1.26E+05			3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	75-27-4	BROMODICHLOROMETHANE	0.6	3 J	ug/l	HB-WA-08I	2/179	0.5-500	3.00E+00			1.70E-01	C	1.81E-01	ca	1.70E-01	Y	ASL
	104-51-8	BUTYLBENZENE	5 J	5 J	ug/l	HB-HB-01S	1/34	0.5-500	5.00E+00			NV	NV	2.43E+01	nc	2.43E+01	N	BSL
	75-15-0	CARBON DISULFIDE	0	200	ug/l	HB-WA-03I	19/136	0.5-1000	2.00E+02			1.04E+02	N	1.04E+02	nc	1.04E+02	Y	ASL
	108-90-7	CHLOROBENZENE	0.1 J	3080	ug/l	HB-HB-01S	34/179	0.5-500	3.08E+03			8.96E+00	N	1.06E+01	nc	8.96E+00	Y	ASL
	75-00-3	CHLOROETHANE	0.3 J	32.6	ug/l	HB-HB-20D	14/179	1-1000	3.26E+01			3.64E+00	C	4.64E+00	ca	3.64E+00	Y	ASL
	67-66-3	CHLOROFORM	0.13 J	240 J	ug/l	HB-HB-12D	11/179	0.5-500	2.40E+02		7.00E-02	1.55E-01	C	1.66E-01	ca	1.55E-01	Y	ASL
	156-59-2	CIS-1,2-DICHLOROETHENE	0.1 J	1.12	ug/l	HB-GWS-01	3/161	0.5-500	1.12E+00			6.08E+00	N	6.08E+00	nc	6.08E+00	N	BSL
	110-82-7	CYCLOHEXANE	0.5	0.55	ug/l	HB-MW-22	2/56	0.5-250	5.50E-01			1.24E+03	N	1.03E+03	nc	1.03E+03	N	BSL
	100-41-4	ETHYLBENZENE	0.1 J	1000	ug/l	HB-HB-13D	72/179	0.5-125	1.00E+03		7.00E-01	1.34E+02	N	1.34E+02	nc	1.34E+02	Y	ASL

TABLE 2.1a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Ground Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	98-82-8	ISOPROPYLBENZENE	0.1 J	68	ug/l	HB-HB-01S	16/90	0.5-500	6.80E+01			6.58E+01	N	6.58E+01	nc	6.58E+01	Y	ASL
	79-20-9	METHYL ACETATE	0.36 J	6.46	ug/l	HB-HB-20D	2/56	0.5-250	6.46E+00			6.08E+02	N	6.08E+02	nc	6.08E+02	N	BSL
	1634-04-4	METHYL TERT-BUTYL ETHER	0.16 J	0.94	ug/l	HB-HB-05D	9/56	0.5-250	9.40E-01			2.64E+00	C	1.10E+01	ca	2.64E+00	N	BSL
	108-87-2	METHYLCYCLOHEXANE	0.46 J	0.46 J	ug/l	HB-HB-21I	1/56	0.5-250	4.60E-01			6.28E+02	N	5.22E+02	nc	5.22E+02	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.11 J	25 J	ug/l	HB-HB-01S	3/179	1-2000	2.50E+01		5.00E-03	4.10E+00	C	4.28E+00	ca	4.10E+00	Y	ASL
	103-65-1	N-PROPYLBENZENE	0.2 J	12 J	ug/l	HB-HB-08I	7/34	0.5-500	1.20E+01			NV	NV	2.43E+01	nc	2.43E+01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.3 J	20	ug/l	HB-HB-01S	8/35	0.5-500	2.00E+01			NV	NV	NV	NV	Y	NTX	
	135-98-8	SEC-BUTYLBENZENE	0.1 J	120	ug/l	HB-HB-01S	6/35	0.5-500	1.20E+02			NV	NV	2.43E+01	nc	2.43E+01	Y	ASL
	100-42-5	STYRENE	0	17000	ug/l	HB-HB-13D	43/170	0.5-500	1.70E+04		1.00E-01	1.62E+02	N	1.64E+02	nc	1.62E+02	Y	ASL
	98-06-6	TERT-BUTYLBENZENE	11	11	ug/l	HB-HB-01S	1/34	0.5-500	1.10E+01			NV	NV	2.43E+01	nc	2.43E+01	N	BSL
	127-18-4	TETRACHLOROETHENE	0.1 J	1.7 J	ug/l	HB-HB-12S	6/180	0.5-500	1.70E+00		5.00E-03	1.04E-01	C	1.04E-01	ca	1.04E-01	Y	ASL
	108-88-3	TOLUENE	0.1 J	6500	ug/l	HB-HB-12D	103/179	0.5-100	6.50E+03		1.00E+00	2.27E+02	N	7.23E+01	nc	7.23E+01	Y	ASL
	75-01-4	VINYL CHLORIDE	0.7 J	4.1 J	ug/l	HB-HB-03S	6/179	1-1000	4.10E+00		2.00E-03	1.50E-02	C	1.98E-02	ca	1.50E-02	Y	TOX
	1330-20-7	XYLENES, TOTAL	0.1 J	4800	ug/l	HB-HB-12I	100/179	0.25-150	4.80E+03		1.00E+01	2.13E+01	N	2.06E+01	nc	2.06E+01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) N/A - No background screening performed.
(4) United States Environmental Protection Agency, 2008. National Primary and Secondary Drinking Water Regulations.
(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
f = RBC and PRG values for endosulfan (CAS# 115297) utilized.
g = RBC and PRG values for 4-methylphenol (CAS# 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals; USEPA, 2004
RBC: Risk Based Concentration; USEPA, October 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-B-04W	3/7/2007	6	11	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-B-04W	3/7/2007	6	11	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-B-08W	3/5/2007	6	11	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.05
HB-B-08W	3/5/2007	6	11	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-B-10	3/7/2007	6	11	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.051
HB-B-10	3/7/2007	6	11	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.051
Total Chlordane =									ND
HB-GWS-01	12/18/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.051
HB-GWS-01	12/18/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.051
Total Chlordane =									ND
HB-GWS-02	12/18/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-GWS-02	12/18/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-GWS-03	12/19/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.051
HB-GWS-03	12/19/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.051
Total Chlordane =									ND
HB-GWS-04	12/20/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.05
HB-GWS-04	12/20/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-GWS-05	12/11/2006	10	12	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.05
HB-GWS-05	12/11/2006	10	12	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-GWS-06	12/15/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	UJ	ug/l	0.052
HB-GWS-06	12/15/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.052
Total Chlordane =									ND
HB-GWS-07	12/14/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.051
HB-GWS-07	12/14/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.051
Total Chlordane =									ND
HB-GWS-08	12/13/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.54
HB-GWS-08	12/13/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.54
Total Chlordane =									ND
HB-GWS-09	12/12/2006	10	12	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-GWS-09	12/12/2006	10	12	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-01D	5/22/2001	86.38	91.38	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-01D	5/22/2001	86.38	91.38	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-01D	5/14/2003	86.38	91.38	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-01D	5/14/2003	86.38	91.38	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-01D	8/19/2003	86.38	91.38	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-01D	8/19/2003	86.38	91.38	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-01D	3/12/2007	86.38	91.38	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-01D	3/12/2007	86.38	91.38	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-01S	5/22/2001	4.95	9.95	57-74-9	CHLORDANE	N	UJ	ug/l	0.05
HB-HB-01S	5/22/2001	4.95	9.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.05
Total Chlordane =									ND
HB-HB-01S	5/14/2003	4.95	9.95	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-01S	5/14/2003	4.95	9.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-01S	8/19/2003	4.95	9.95	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-01S	8/19/2003	4.95	9.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-01S	3/12/2007	4.95	9.95	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-01S	3/12/2007	4.95	9.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-02I	5/17/2001	22.1	32.1	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-02I	5/17/2001	22.1	32.1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-02I	5/20/2003	22.1	32.1	57-74-9	CHLORDANE	N	U	ug/l	0.051
HB-HB-02I	5/20/2003	22.1	32.1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.051
Total Chlordane =									ND
HB-HB-02I	8/22/2003	22.1	32.1	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-02I	8/22/2003	22.1	32.1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-02I	3/15/2007	22.1	32.1	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-02I	3/15/2007	22.1	32.1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-02S	5/17/2001	4.01	14.01	57-74-9	CHLORDANE	N	UJ	ug/l	0.05
HB-HB-02S	5/17/2001	4.01	14.01	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.05
Total Chlordane =									ND
HB-HB-02S	5/20/2003	4.01	14.01	57-74-9	CHLORDANE	N	U	ug/l	0.25
HB-HB-02S	5/20/2003	4.01	14.01	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.25
Total Chlordane =									ND
HB-HB-02S	8/22/2003	4.01	14.01	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-02S	8/22/2003	4.01	14.01	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-02S	3/15/2007	4.01	14.01	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	1
HB-HB-02S	3/15/2007	4.01	14.01	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	1
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-03S	5/22/2001	4.96	14.96	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-03S	5/22/2001	4.96	14.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-03S	5/14/2003	4.96	14.96	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-03S	5/14/2003	4.96	14.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-03S	8/19/2003	4.96	14.96	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-03S	8/19/2003	4.96	14.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-03S	3/8/2007	4.96	14.96	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.11
HB-HB-03S	3/8/2007	4.96	14.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.11
Total Chlordane =									ND
HB-HB-04D	5/16/2003	87.99	97.99	57-74-9	CHLORDANE	N	U	ug/l	0.051
HB-HB-04D	5/16/2003	87.99	97.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.051
Total Chlordane =									ND
HB-HB-04D	8/20/2003	87.99	97.99	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-04D	8/20/2003	87.99	97.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-04D	3/14/2007	87.99	97.99	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-04D	3/14/2007	87.99	97.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-04S	5/17/2001	8.59	18.59	57-74-9	CHLORDANE	N	UJ	ug/l	0.05
HB-HB-04S	5/17/2001	8.59	18.59	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.05
Total Chlordane =									ND
HB-HB-04S	5/16/2003	8.59	18.59	57-74-9	CHLORDANE	N	U	ug/l	0.96
HB-HB-04S	5/16/2003	8.59	18.59	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.96
Total Chlordane =									ND
HB-HB-04S	8/20/2003	8.59	18.59	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-04S	8/20/2003	8.59	18.59	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-04S	3/14/2007	8.59	18.59	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.52
HB-HB-04S	3/14/2007	8.59	18.59	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.52
Total Chlordane =									ND
HB-HB-05D	5/20/2003	97.99	107.99	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-05D	5/20/2003	97.99	107.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-05D	8/19/2003	97.99	107.99	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-05D	8/19/2003	97.99	107.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-05D	3/13/2007	97.99	107.99	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-05D	3/13/2007	97.99	107.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-05I	5/23/2001	44.09	54.09	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-05I	5/23/2001	44.09	54.09	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-05I	5/20/2003	44.09	54.09	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-05I	5/20/2003	44.09	54.09	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-05I	8/19/2003	44.09	54.09	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-05I	8/19/2003	44.09	54.09	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-05I	3/13/2007	44.09	54.09	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.054
HB-HB-05I	3/13/2007	44.09	54.09	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.054
Total Chlordane =									ND
HB-HB-05S	5/23/2001	7.03	17.03	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-05S	5/23/2001	7.03	17.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-05S	5/20/2003	7.03	17.03	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-05S	5/20/2003	7.03	17.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-05S	8/19/2003	7.03	17.03	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-05S	8/19/2003	7.03	17.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-05S	3/13/2007	7.03	17.03	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.05
HB-HB-05S	3/13/2007	7.03	17.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-06S	5/23/2001	3	13	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-06S	5/23/2001	3	13	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-06S	5/22/2003	3	13	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-06S	5/22/2003	3	13	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-06S	8/25/2003	3	13	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-06S	8/25/2003	3	13	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-06S	3/20/2007	3	13	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-06S	3/20/2007	3	13	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-07S	5/10/2001	3	8	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-07S	5/10/2001	3	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-07S	5/19/2003	3	8	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-07S	5/19/2003	3	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-07S	8/22/2003	3	8	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-07S	8/22/2003	3	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-07S	3/19/2007	3	8	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-07S	3/19/2007	3	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-08D	5/19/2003	57.98	67.98	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-08D	5/19/2003	57.98	67.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-08D	8/26/2003	57.98	67.98	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-08D	8/26/2003	57.98	67.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-08D	3/19/2007	57.98	67.98	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-08D	3/19/2007	57.98	67.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-08I	5/11/2001	11.95	21.95	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-08I	5/11/2001	11.95	21.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-08I	5/19/2003	11.95	21.95	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-08I	5/19/2003	11.95	21.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-08I	8/26/2003	11.95	21.95	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-08I	8/26/2003	11.95	21.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-08I	3/19/2007	11.95	21.95	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.52
HB-HB-08I	3/19/2007	11.95	21.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.52
Total Chlordane =									ND
HB-HB-08S	5/11/2001	5	10	57-74-9	CHLORDANE	N	U	ug/l	0.06
HB-HB-08S	5/11/2001	5	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.06
Total Chlordane =									ND
HB-HB-08S	5/19/2003	5	10	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-08S	5/19/2003	5	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-08S	8/27/2003	5	10	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-08S	8/27/2003	5	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-08S	3/19/2007	5	10	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-08S	3/19/2007	5	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-09	5/10/2001	5	15	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-09	5/10/2001	5	15	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-09	5/19/2003	5	15	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-09	5/19/2003	5	15	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-09	8/22/2003	5	15	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-09	8/22/2003	5	15	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-09S	3/19/2007	4.96	14.96	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-09S	3/19/2007	4.96	14.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-11I	5/11/2001	34.95	44.95	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-11I	5/11/2001	34.95	44.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-11I	5/13/2003	34.95	44.95	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-11I	5/13/2003	34.95	44.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-11I	8/18/2003	34.95	44.95	57-74-9	CHLORDANE	N	UJ	ug/l	0.049
HB-HB-11I	8/18/2003	34.95	44.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.049
Total Chlordane =									ND
HB-HB-11I	3/15/2007	34.95	44.95	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.26
HB-HB-11I	3/15/2007	34.95	44.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.26
Total Chlordane =									ND
HB-HB-11S	5/11/2001	3.98	13.98	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-11S	5/11/2001	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-11S	3/15/2007	3.98	13.98	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.054
HB-HB-11S	3/15/2007	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.054
Total Chlordane =									ND
HB-HB-12D	5/11/2001	78	88	57-74-9	CHLORDANE	N	UJ	ug/l	0.05
HB-HB-12D	5/11/2001	78	88	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.05
Total Chlordane =									ND
HB-HB-12D	5/12/2003	78	88	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-12D	5/12/2003	78	88	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-12D	8/13/2003	78	88	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-12D	8/13/2003	78	88	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-12D	3/16/2007	78	88	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.26
HB-HB-12D	3/16/2007	78	88	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.26
Total Chlordane =									ND
HB-HB-12I	5/14/2001	35.03	50.03	57-74-9	CHLORDANE	N	UJ	ug/l	0.05
HB-HB-12I	5/14/2001	35.03	50.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	ug/l	0.02
Total Chlordane =									0.02

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-12I	5/12/2003	35.03	50.03	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-12I	5/12/2003	35.03	50.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-12I	8/13/2003	35.03	50.03	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-12I	8/13/2003	35.03	50.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-12I	3/16/2007	35.03	50.03	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-12I	3/16/2007	35.03	50.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-12S	5/14/2001	5.96	15.96	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-12S	5/14/2001	5.96	15.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-12S	5/12/2003	5.96	15.96	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-12S	5/12/2003	5.96	15.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-12S	8/13/2003	5.96	15.96	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-12S	8/13/2003	5.96	15.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-12S	3/16/2007	5.96	15.96	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-12S	3/16/2007	5.96	15.96	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-13D	5/14/2001	76	86	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-13D	5/14/2001	76	86	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-13D	5/15/2003	76	86	57-74-9	CHLORDANE	N	U	ug/l	0.24
HB-HB-13D	5/15/2003	76	86	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.24
Total Chlordane =									ND
HB-HB-13D	8/18/2003	76	86	57-74-9	CHLORDANE	N	UJ	ug/l	0.048
HB-HB-13D	8/18/2003	76	86	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.048
Total Chlordane =									ND
HB-HB-13D	3/16/2007	76	86	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.26
HB-HB-13D	3/16/2007	76	86	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.26
Total Chlordane =									ND
HB-HB-14D	5/16/2001	28	38	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-14D	5/16/2001	28	38	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-14D	5/13/2003	28	38	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-14D	5/13/2003	28	38	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-14D	8/18/2003	28	38	57-74-9	CHLORDANE	N	UJ	ug/l	0.049
HB-HB-14D	8/18/2003	28	38	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.049
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-14S	5/13/2003	6.95	11.95	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-14S	5/13/2003	6.95	11.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-14S	8/20/2003	6.95	11.95	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HB-14S	8/20/2003	6.95	11.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HB-16D	5/14/2003	97.02	107.02	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-16D	5/14/2003	97.02	107.02	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-16D	8/20/2003	97.02	107.02	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-16D	8/20/2003	97.02	107.02	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-16D	3/13/2007	97.02	107.02	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-16D	3/13/2007	97.02	107.02	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-17D	5/13/2003	67	77	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-17D	5/13/2003	67	77	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-17D	8/13/2003	67	77	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-17D	8/13/2003	67	77	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-17D	3/16/2007	67	77	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.054
HB-HB-17D	3/16/2007	67	77	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.054
Total Chlordane =									ND
HB-HB-18S	5/21/2003	3.98	13.98	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-18S	5/21/2003	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-18S	8/27/2003	3.98	13.98	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-18S	8/27/2003	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-18S	3/20/2007	3.98	13.98	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-18S	3/20/2007	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-19S	5/21/2003	4.01	14.01	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-19S	5/21/2003	4.01	14.01	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-19S	8/27/2003	4.01	14.01	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-19S	8/27/2003	4.01	14.01	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-19S	3/20/2007	4.01	14.01	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-19S	3/20/2007	4.01	14.01	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-20D	5/22/2003	125	135	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-20D	5/22/2003	125	135	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-20D	8/25/2003	125	135	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-20D	8/25/2003	125	135	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-20D	3/22/2007	125	135	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-20D	3/22/2007	125	135	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HB-20I	5/22/2003	27.99	37.99	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-20I	5/22/2003	27.99	37.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-20I	8/25/2003	27.99	37.99	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-HB-20I	8/25/2003	27.99	37.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-HB-20I	3/22/2007	27.99	37.99	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-20I	3/22/2007	27.99	37.99	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-20S	5/22/2003	3.98	13.98	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-20S	5/22/2003	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-20S	8/25/2003	3.98	13.98	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-20S	8/25/2003	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-20S	3/22/2007	3.98	13.98	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-HB-20S	3/22/2007	3.98	13.98	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-HB-21I	5/21/2003	20.03	30.03	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-HB-21I	5/21/2003	20.03	30.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-HB-21I	8/22/2003	20.03	30.03	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-HB-21I	8/22/2003	20.03	30.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-HB-21I	3/20/2007	20.03	30.03	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-HB-21I	3/20/2007	20.03	30.03	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-HP-01	7/6/2000	17	17	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HP-01	7/6/2000	16	16	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HP-02	7/5/2000			57-74-9	CHLORDANE	N	U	ug/l	0.07
HB-HP-02	7/5/2000			12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.07
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HP-03	7/5/2000			57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HP-03	7/5/2000			12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HP-04	7/5/2000			57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HP-04	7/5/2000			12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HP-05	7/5/2000			57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HP-05	7/5/2000			12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HP-06	7/6/2000			57-74-9	CHLORDANE	N	U	ug/l	0.07
HB-HP-06	7/6/2000			12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.07
Total Chlordane =									ND
HB-HP-07	7/6/2000			57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HP-07	7/6/2000			12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-HP-08	7/6/2000			57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-HP-08	7/6/2000			12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-MW-22	3/5/2007	4	14	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-MW-22	3/5/2007	4	14	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-MW-23	3/5/2007	4	14	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.055
HB-MW-23	3/5/2007	4	14	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.055
Total Chlordane =									ND
HB-MW-24	3/7/2007	4	14	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.05
HB-MW-24	3/7/2007	4	14	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-MW-25	3/7/2007	4	14	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-MW-25	3/7/2007	4	14	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-MW-26	3/5/2007	5	15	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.054
HB-MW-26	3/5/2007	5	15	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.054
Total Chlordane =									ND
HB-MW-27	3/7/2007	4	14	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-MW-27	3/7/2007	4	14	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-WA-03D	3/8/2007	53.5	63.5	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.055
HB-WA-03D	3/8/2007	53.5	63.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.055
Total Chlordane =									ND
HB-WA-03I	3/8/2007	20	30	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-WA-03I	3/8/2007	20	30	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WA-03S	3/8/2007	20	30	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.052
HB-WA-03S	3/8/2007	20	30	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.052
Total Chlordane =									ND
HB-WA-08D	5/21/2001	70.5	80.5	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-WA-08D	5/21/2001	70.5	80.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-WA-08D	5/15/2003	70.5	80.5	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-WA-08D	5/15/2003	70.5	80.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-WA-08D	8/14/2003	70.5	80.5	57-74-9	CHLORDANE	N	UJ	ug/l	0.048
HB-WA-08D	8/14/2003	70.5	80.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.048
Total Chlordane =									ND
HB-WA-08D	3/12/2007	70.5	80.5	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.054
HB-WA-08D	3/12/2007	70.5	80.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.054
Total Chlordane =									ND
HB-WA-08I	5/21/2001	30.5	40.5	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-WA-08I	5/21/2001	30.5	40.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-WA-08I	5/15/2003	30.5	40.5	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-WA-08I	5/15/2003	30.5	40.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-WA-08I	8/14/2003	30.5	40.5	57-74-9	CHLORDANE	N	UJ	ug/l	0.048
HB-WA-08I	8/14/2003	30.5	40.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.048
Total Chlordane =									ND
HB-WA-08I	3/12/2007	30.5	40.5	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-WA-08I	3/12/2007	30.5	40.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-WA-08S	5/21/2001	8.95	18.95	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-WA-08S	5/21/2001	8.95	18.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND
HB-WA-08S	5/15/2003	8.95	18.95	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-WA-08S	5/15/2003	8.95	18.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-WA-08S	8/14/2003	8.95	18.95	57-74-9	CHLORDANE	N	UJ	ug/l	0.048
HB-WA-08S	8/14/2003	8.95	18.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.048
Total Chlordane =									ND
HB-WA-08S	3/12/2007	8.95	18.95	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.053
HB-WA-08S	3/12/2007	8.95	18.95	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.053
Total Chlordane =									ND
HB-WB-BL	5/22/2001	80.5	85.5	57-74-9	CHLORDANE	N	U	ug/l	0.05
HB-WB-BL	5/22/2001	80.5	85.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.05
Total Chlordane =									ND

TABLE 2.1b
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WB-BL	5/21/2003	80.5	85.5	57-74-9	CHLORDANE	N	U	ug/l	0.049
HB-WB-BL	5/21/2003	80.5	85.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.049
Total Chlordane =									ND
HB-WB-BL	8/26/2003	80.5	85.5	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-WB-BL	8/26/2003	80.5	85.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-WB-BL	3/14/2007	80.5	85.5	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.051
HB-WB-BL	3/14/2007	80.5	85.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.051
Total Chlordane =									ND
HB-WB-BU	5/21/2001	18.8	23.8	57-74-9	CHLORDANE	N	UJ	ug/l	0.05
HB-WB-BU	5/21/2001	18.8	23.8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	ug/l	0.05
Total Chlordane =									ND
HB-WB-BU	5/21/2003	18.8	23.8	57-74-9	CHLORDANE	N	U	ug/l	0.048
HB-WB-BU	5/21/2003	18.8	23.8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.048
Total Chlordane =									ND
HB-WB-BU	8/26/2003	18.8	23.8	57-74-9	CHLORDANE	N	U	ug/l	0.047
HB-WB-BU	8/26/2003	18.8	23.8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.047
Total Chlordane =									ND
HB-WB-BU	3/14/2007	18.8	23.8	5103-71-9	ALPHA-CHLORDANE	N	U	ug/l	0.51
HB-WB-BU	3/14/2007	18.8	23.8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	ug/l	0.51
Total Chlordane =									ND

TABLE 2.1c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-B-04W	3/7/2007	6	11	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	15.2	15.2
HB-B-08W	3/5/2007	6	11	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.58	0.58
HB-B-10	3/7/2007	6	11	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-01	12/18/2006	8	10	1330-20-7	XYLENES, TOTAL	Y		ug/l	1.68	1.68
HB-GWS-02	12/18/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-03	12/19/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-04	12/20/2006	8	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.38	0.38
HB-GWS-05	12/11/2006	10	12	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-06	12/15/2006	8	10	1330-20-7	XYLENES, TOTAL	N	UJ	ug/l	1	0.5
HB-GWS-07	12/14/2006	8	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.11	0.11
HB-GWS-08	12/13/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-09	12/12/2006	10	12	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-01D	5/22/2001	86.38	91.38	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.1	0.1
HB-HB-01D	5/14/2003	86.38	91.38	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-01D	5/14/2003	86.38	91.38	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-01D	5/14/2003	86.38	91.38	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-01D	8/19/2003	86.38	91.38	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-01D	8/19/2003	86.38	91.38	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-01D	8/19/2003	86.38	91.38	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-01D	3/12/2007	86.38	91.38	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-01S	5/14/2003	4.95	9.95	XYLENES1314	XYLENES, M & P	Y		ug/l	650	
HB-HB-01S	5/14/2003	4.95	9.95	95-47-6	O-XYLENE	Y		ug/l	300	
HB-HB-01S	5/14/2003	4.95	9.95	CALCULATED	TOTAL	Y		ug/l		950
HB-HB-01S	8/19/2003	4.95	9.95	XYLENES1314	XYLENES, M & P	Y		ug/l	830	
HB-HB-01S	8/19/2003	4.95	9.95	95-47-6	O-XYLENE	Y		ug/l	400	
HB-HB-01S	8/19/2003	4.95	9.95	CALCULATED	TOTAL	Y		ug/l		1230
HB-HB-01S	3/12/2007	4.95	9.95	1330-20-7	XYLENES, TOTAL	Y		ug/l	475	475
HB-HB-02I	5/17/2001	22.1	32.1	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	6	6
HB-HB-02I	5/20/2003	22.1	32.1	XYLENES1314	XYLENES, M & P	N	U	ug/l	50	
HB-HB-02I	5/20/2003	22.1	32.1	95-47-6	O-XYLENE	N	U	ug/l	50	
HB-HB-02I	5/20/2003	22.1	32.1	CALCULATED	TOTAL	N	U	ug/l		50
HB-HB-02I	8/22/2003	22.1	32.1	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-02I	8/22/2003	22.1	32.1	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-02I	8/22/2003	22.1	32.1	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-02I	3/15/2007	22.1	32.1	1330-20-7	XYLENES, TOTAL	Y		ug/l	2.27	2.27
HB-HB-02S	5/17/2001	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y		ug/l	2800	2800
HB-HB-02S	5/20/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	Y		ug/l	2100	
HB-HB-02S	5/20/2003	4.01	14.01	95-47-6	O-XYLENE	Y		ug/l	810	
HB-HB-02S	5/20/2003	4.01	14.01	CALCULATED	TOTAL	Y		ug/l		2910
HB-HB-02S	8/22/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	Y		ug/l	2000	
HB-HB-02S	8/22/2003	4.01	14.01	95-47-6	O-XYLENE	Y		ug/l	770	
HB-HB-02S	8/22/2003	4.01	14.01	CALCULATED	TOTAL	Y		ug/l		2770
HB-HB-02S	3/15/2007	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y		ug/l	3380	3380
HB-HB-03S	5/22/2001	4.96	14.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	23	23
HB-HB-03S	5/14/2003	4.96	14.96	XYLENES1314	XYLENES, M & P	Y		ug/l	32	
HB-HB-03S	5/14/2003	4.96	14.96	95-47-6	O-XYLENE	Y		ug/l	11	
HB-HB-03S	5/14/2003	4.96	14.96	CALCULATED	TOTAL	Y		ug/l		43
HB-HB-03S	8/19/2003	4.96	14.96	XYLENES1314	XYLENES, M & P	Y		ug/l	51	
HB-HB-03S	8/19/2003	4.96	14.96	95-47-6	O-XYLENE	Y		ug/l	15	
HB-HB-03S	8/19/2003	4.96	14.96	CALCULATED	TOTAL	Y		ug/l		66
HB-HB-03S	3/8/2007	4.96	14.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	33	33
HB-HB-04D	5/16/2003	87.99	97.99	XYLENES1314	XYLENES, M & P	N	UJ	ug/l	5	
HB-HB-04D	5/16/2003	87.99	97.99	95-47-6	O-XYLENE	N	UJ	ug/l	5	
HB-HB-04D	5/16/2003	87.99	97.99	CALCULATED	TOTAL	N	UJ	ug/l		5
HB-HB-04D	8/20/2003	87.99	97.99	XYLENES1314	XYLENES, M & P	N	UJ	ug/l	5	
HB-HB-04D	8/20/2003	87.99	97.99	95-47-6	O-XYLENE	N	UJ	ug/l	5	
HB-HB-04D	8/20/2003	87.99	97.99	CALCULATED	TOTAL	N	UJ	ug/l		5
HB-HB-04D	3/14/2007	87.99	97.99	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-04S	5/17/2001	8.59	18.59	1330-20-7	XYLENES, TOTAL	Y		ug/l	800	800
HB-HB-04S	5/16/2003	8.59	18.59	XYLENES1314	XYLENES, M & P	Y		ug/l	1400	
HB-HB-04S	5/16/2003	8.59	18.59	95-47-6	O-XYLENE	Y		ug/l	540	
HB-HB-04S	5/16/2003	8.59	18.59	CALCULATED	TOTAL	Y		ug/l		1940
HB-HB-04S	8/20/2003	8.59	18.59	XYLENES1314	XYLENES, M & P	Y		ug/l	2300	
HB-HB-04S	8/20/2003	8.59	18.59	95-47-6	O-XYLENE	Y		ug/l	870	
HB-HB-04S	8/20/2003	8.59	18.59	CALCULATED	TOTAL	Y		ug/l		3170
HB-HB-04S	3/14/2007	8.59	18.59	1330-20-7	XYLENES, TOTAL	Y		ug/l	3220	3220
HB-HB-05D	5/20/2003	97.99	107.99	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-05D	5/20/2003	97.99	107.99	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-05D	5/20/2003	97.99	107.99	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-05D	8/19/2003	97.99	107.99	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-05D	8/19/2003	97.99	107.99	95-47-6	O-XYLENE	N	U	ug/l	5	

TABLE 2.1c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-05D	8/19/2003	97.99	107.99	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-05D	3/13/2007	97.99	107.99	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.33	0.33
HB-HB-05I	5/23/2001	44.09	54.09	1330-20-7	XYLENES, TOTAL	N	UJ	ug/l	2	1
HB-HB-05I	5/20/2003	44.09	54.09	XYLENES1314	XYLENES, M & P	N	U	ug/l	13	
HB-HB-05I	5/20/2003	44.09	54.09	95-47-6	O-XYLENE	N	U	ug/l	13	
HB-HB-05I	5/20/2003	44.09	54.09	CALCULATED	TOTAL	N	U	ug/l		13
HB-HB-05I	8/19/2003	44.09	54.09	XYLENES1314	XYLENES, M & P	N	U	ug/l	25	
HB-HB-05I	8/19/2003	44.09	54.09	95-47-6	O-XYLENE	N	U	ug/l	25	
HB-HB-05I	8/19/2003	44.09	54.09	CALCULATED	TOTAL	N	U	ug/l		25
HB-HB-05I	3/13/2007	44.09	54.09	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-05S	5/23/2001	7.03	17.03	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-05S	5/20/2003	7.03	17.03	XYLENES1314	XYLENES, M & P	N	U	ug/l	50	
HB-HB-05S	5/20/2003	7.03	17.03	95-47-6	O-XYLENE	N	U	ug/l	50	
HB-HB-05S	5/20/2003	7.03	17.03	CALCULATED	TOTAL	N	U	ug/l		50
HB-HB-05S	8/19/2003	7.03	17.03	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-05S	8/19/2003	7.03	17.03	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-05S	8/19/2003	7.03	17.03	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-05S	3/13/2007	7.03	17.03	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-06S	5/23/2001	3	13	1330-20-7	XYLENES, TOTAL	Y		ug/l	55	55
HB-HB-06S	5/22/2003	3	13	XYLENES1314	XYLENES, M & P	Y		ug/l	35	
HB-HB-06S	5/22/2003	3	13	95-47-6	O-XYLENE	Y		ug/l	34	
HB-HB-06S	5/22/2003	3	13	CALCULATED	TOTAL	Y		ug/l		69
HB-HB-06S	8/25/2003	3	13	XYLENES1314	XYLENES, M & P	Y		ug/l	11	
HB-HB-06S	8/25/2003	3	13	95-47-6	O-XYLENE	Y		ug/l	12	
HB-HB-06S	8/25/2003	3	13	CALCULATED	TOTAL	Y		ug/l		23
HB-HB-06S	3/20/2007	3	13	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2.2	2.2
HB-HB-07S	5/10/2001	3	8	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-07S	5/19/2003	3	8	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-07S	5/19/2003	3	8	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-07S	5/19/2003	3	8	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-07S	8/22/2003	3	8	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-07S	8/22/2003	3	8	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-07S	8/22/2003	3	8	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-07S	3/19/2007	3	8	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-08D	5/19/2003	57.98	67.98	XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.1	
HB-HB-08D	5/19/2003	57.98	67.98	95-47-6	O-XYLENE	Y	J	ug/l	4.3	
HB-HB-08D	5/19/2003	57.98	67.98	CALCULATED	TOTAL	Y	J	ug/l		5.4
HB-HB-08D	8/26/2003	57.98	67.98	XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.1	
HB-HB-08D	8/26/2003	57.98	67.98	95-47-6	O-XYLENE	Y	J	ug/l	4.4	
HB-HB-08D	8/26/2003	57.98	67.98	CALCULATED	TOTAL	Y	J	ug/l		5.5
HB-HB-08D	3/19/2007	57.98	67.98	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	1.96	1.96
HB-HB-08I	5/11/2001	11.95	21.95	1330-20-7	XYLENES, TOTAL	Y		ug/l	1500	1500
HB-HB-08I	5/19/2003	11.95	21.95	XYLENES1314	XYLENES, M & P	Y		ug/l	1200	
HB-HB-08I	5/19/2003	11.95	21.95	95-47-6	O-XYLENE	Y		ug/l	530	
HB-HB-08I	5/19/2003	11.95	21.95	CALCULATED	TOTAL	Y		ug/l		1730
HB-HB-08I	8/26/2003	11.95	21.95	XYLENES1314	XYLENES, M & P	Y		ug/l	1000	
HB-HB-08I	8/26/2003	11.95	21.95	95-47-6	O-XYLENE	Y		ug/l	440	
HB-HB-08I	8/26/2003	11.95	21.95	CALCULATED	TOTAL	Y		ug/l		1440
HB-HB-08I	3/19/2007	11.95	21.95	1330-20-7	XYLENES, TOTAL	Y		ug/l	760	760
HB-HB-08S	5/11/2001	5	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.2	0.2
HB-HB-08S	5/19/2003	5	10	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-08S	5/19/2003	5	10	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-08S	5/19/2003	5	10	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-08S	8/26/2003	5	10	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-08S	8/26/2003	5	10	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-08S	8/26/2003	5	10	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-08S	3/19/2007	5	10	1330-20-7	XYLENES, TOTAL	Y		ug/l	1.23	1.23
HB-HB-09	5/10/2001	5	15	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-09	5/19/2003	5	15	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-09	5/19/2003	5	15	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-09	5/19/2003	5	15	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-09	8/22/2003	5	15	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-09	8/22/2003	5	15	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-09	8/22/2003	5	15	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-09S	3/19/2007	4.96	14.96	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-11I	5/11/2001	34.95	44.95	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2	2
HB-HB-11I	5/13/2003	34.95	44.95	XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.1	
HB-HB-11I	5/13/2003	34.95	44.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-11I	5/13/2003	34.95	44.95	CALCULATED	TOTAL	Y	J	ug/l		1.1
HB-HB-11I	8/18/2003	34.95	44.95	XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.3	
HB-HB-11I	8/18/2003	34.95	44.95	95-47-6	O-XYLENE	N	U	ug/l	5	

TABLE 2.1c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-11I	8/18/2003	34.95	44.95	CALCULATED	TOTAL	Y	J	ug/l		1.3
HB-HB-11I	3/15/2007	34.95	44.95	1330-20-7	XYLENES, TOTAL	Y		ug/l	17.7	17.7
HB-HB-11S	5/11/2001	3.98	13.98	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-11S	3/15/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-12D	5/11/2001	78	88	1330-20-7	XYLENES, TOTAL	Y		ug/l	1500	1500
HB-HB-12D	5/12/2003	78	88	XYLENES1314	XYLENES, M & P	Y	J	ug/l	1300	
HB-HB-12D	5/12/2003	78	88	95-47-6	O-XYLENE	Y	J	ug/l	640	
HB-HB-12D	5/12/2003	78	88	CALCULATED	TOTAL	Y	J	ug/l		1940
HB-HB-12D	8/13/2003	78	88	XYLENES1314	XYLENES, M & P	Y		ug/l	1800	
HB-HB-12D	8/13/2003	78	88	95-47-6	O-XYLENE	Y		ug/l	830	
HB-HB-12D	8/13/2003	78	88	CALCULATED	TOTAL	Y		ug/l		2630
HB-HB-12D	3/16/2007	78	88	1330-20-7	XYLENES, TOTAL	Y		ug/l	1960	1960
HB-HB-12I	5/14/2001	35.03	50.03	1330-20-7	XYLENES, TOTAL	Y		ug/l	4800	4800
HB-HB-12I	5/12/2003	35.03	50.03	XYLENES1314	XYLENES, M & P	Y		ug/l	2800	
HB-HB-12I	5/12/2003	35.03	50.03	95-47-6	O-XYLENE	Y		ug/l	1300	
HB-HB-12I	5/12/2003	35.03	50.03	CALCULATED	TOTAL	Y		ug/l		4100
HB-HB-12I	8/13/2003	35.03	50.03	XYLENES1314	XYLENES, M & P	Y		ug/l	3100	
HB-HB-12I	8/13/2003	35.03	50.03	95-47-6	O-XYLENE	Y		ug/l	1400	
HB-HB-12I	8/13/2003	35.03	50.03	CALCULATED	TOTAL	Y		ug/l		4500
HB-HB-12I	3/16/2007	35.03	50.03	1330-20-7	XYLENES, TOTAL	Y		ug/l	660	660
HB-HB-12S	5/14/2001	5.96	15.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2	2
HB-HB-12S	5/12/2003	5.96	15.96	XYLENES1314	XYLENES, M & P	Y		ug/l	8	
HB-HB-12S	5/12/2003	5.96	15.96	95-47-6	O-XYLENE	Y	J	ug/l	3.7	
HB-HB-12S	5/12/2003	5.96	15.96	CALCULATED	TOTAL	Y		ug/l		11.7
HB-HB-12S	8/13/2003	5.96	15.96	XYLENES1314	XYLENES, M & P	Y	J	ug/l	4.4	
HB-HB-12S	8/13/2003	5.96	15.96	95-47-6	O-XYLENE	Y	J	ug/l	1.8	
HB-HB-12S	8/13/2003	5.96	15.96	CALCULATED	TOTAL	Y	J	ug/l		6.2
HB-HB-12S	3/16/2007	5.96	15.96	1330-20-7	XYLENES, TOTAL	Y		ug/l	5.06	5.06
HB-HB-13D	5/14/2001	76	86	1330-20-7	XYLENES, TOTAL	Y		ug/l	4500	4500
HB-HB-13D	5/15/2003	76	86	XYLENES1314	XYLENES, M & P	Y		ug/l	2000	
HB-HB-13D	5/15/2003	76	86	95-47-6	O-XYLENE	Y		ug/l	880	
HB-HB-13D	5/15/2003	76	86	CALCULATED	TOTAL	Y		ug/l		2880
HB-HB-13D	8/18/2003	76	86	XYLENES1314	XYLENES, M & P	Y		ug/l	1500	
HB-HB-13D	8/18/2003	76	86	95-47-6	O-XYLENE	Y		ug/l	690	
HB-HB-13D	8/18/2003	76	86	CALCULATED	TOTAL	Y		ug/l		2190
HB-HB-13D	3/16/2007	76	86	1330-20-7	XYLENES, TOTAL	Y		ug/l	1070	1070
HB-HB-14D	5/16/2001	28	38	1330-20-7	XYLENES, TOTAL	Y		ug/l	180	180
HB-HB-14D	5/13/2003	28	38	XYLENES1314	XYLENES, M & P	Y		ug/l	2800	
HB-HB-14D	5/13/2003	28	38	95-47-6	O-XYLENE	Y		ug/l	1000	
HB-HB-14D	5/13/2003	28	38	CALCULATED	TOTAL	Y		ug/l		3800
HB-HB-14D	8/18/2003	28	38	XYLENES1314	XYLENES, M & P	Y		ug/l	1400	
HB-HB-14D	8/18/2003	28	38	95-47-6	O-XYLENE	Y		ug/l	530	
HB-HB-14D	8/18/2003	28	38	CALCULATED	TOTAL	Y		ug/l		1930
HB-HB-14S	5/16/2001	6.95	11.95	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-14S	5/13/2003	6.95	11.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-14S	5/13/2003	6.95	11.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-14S	5/13/2003	6.95	11.95	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-14S	8/18/2003	6.95	11.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-14S	8/18/2003	6.95	11.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-14S	8/18/2003	6.95	11.95	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-16D	5/14/2003	97.02	107.02	XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.5	
HB-HB-16D	5/14/2003	97.02	107.02	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-16D	5/14/2003	97.02	107.02	CALCULATED	TOTAL	Y	J	ug/l		1.5
HB-HB-16D	8/20/2003	97.02	107.02	XYLENES1314	XYLENES, M & P	Y	J	ug/l	2	
HB-HB-16D	8/20/2003	97.02	107.02	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-16D	8/20/2003	97.02	107.02	CALCULATED	TOTAL	Y	J	ug/l		2
HB-HB-16D	3/13/2007	97.02	107.02	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.11	0.11
HB-HB-17D	5/13/2003	67	77	XYLENES1314	XYLENES, M & P	Y		ug/l	13	
HB-HB-17D	5/13/2003	67	77	95-47-6	O-XYLENE	Y		ug/l	34	
HB-HB-17D	5/13/2003	67	77	CALCULATED	TOTAL	Y		ug/l		47
HB-HB-17D	8/13/2003	67	77	XYLENES1314	XYLENES, M & P	Y		ug/l	10	
HB-HB-17D	8/13/2003	67	77	95-47-6	O-XYLENE	Y		ug/l	27	
HB-HB-17D	8/13/2003	67	77	CALCULATED	TOTAL	Y		ug/l		37
HB-HB-17D	3/16/2007	67	77	1330-20-7	XYLENES, TOTAL	Y		ug/l	11.4	11.4
HB-HB-18S	5/21/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-18S	5/21/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-18S	5/21/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-18S	8/27/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-18S	8/27/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-18S	8/27/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-18S	3/20/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.73	0.73

TABLE 2.1c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-19S	5/21/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-19S	5/21/2003	4.01	14.01	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-19S	5/21/2003	4.01	14.01	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-19S	8/27/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-19S	8/27/2003	4.01	14.01	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-19S	8/27/2003	4.01	14.01	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-19S	3/20/2007	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.57	0.57
HB-HB-20D	5/22/2003	125	135	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20D	5/22/2003	125	135	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20D	5/22/2003	125	135	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20D	8/25/2003	125	135	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20D	8/25/2003	125	135	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20D	8/25/2003	125	135	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20D	3/22/2007	125	135	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-20I	5/22/2003	27.99	37.99	XYLENES1314	XYLENES, M & P	N	U	ug/l	50	
HB-HB-20I	5/22/2003	27.99	37.99	95-47-6	O-XYLENE	N	U	ug/l	50	
HB-HB-20I	5/22/2003	27.99	37.99	CALCULATED	TOTAL	N	U	ug/l		50
HB-HB-20I	8/25/2003	27.99	37.99	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20I	8/25/2003	27.99	37.99	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20I	8/25/2003	27.99	37.99	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20I	3/22/2007	27.99	37.99	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.21	0.21
HB-HB-20S	5/22/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20S	5/22/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20S	5/22/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20S	8/25/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20S	8/25/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20S	8/25/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20S	3/22/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.52	0.52
HB-HB-21I	5/21/2003	20.03	30.03	XYLENES1314	XYLENES, M & P	Y		ug/l	130	
HB-HB-21I	5/21/2003	20.03	30.03	95-47-6	O-XYLENE	Y		ug/l	120	
HB-HB-21I	5/21/2003	20.03	30.03	CALCULATED	TOTAL	Y		ug/l		250
HB-HB-21I	8/22/2003	20.03	30.03	XYLENES1314	XYLENES, M & P	Y		ug/l	91	
HB-HB-21I	8/22/2003	20.03	30.03	95-47-6	O-XYLENE	Y		ug/l	87	
HB-HB-21I	8/22/2003	20.03	30.03	CALCULATED	TOTAL	Y		ug/l		178
HB-HB-21I	3/20/2007	20.03	30.03	1330-20-7	XYLENES, TOTAL	Y		ug/l	92.7	92.7
HB-HP-01	7/6/2000			1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.3	0.3
HB-HP-01	7/6/2000			1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.4	0.4
HB-HP-02	7/5/2000			1330-20-7	XYLENES, TOTAL	Y		ug/l	1	1
HB-HP-03	7/5/2000			1330-20-7	XYLENES, TOTAL	Y		ug/l	1	1
HB-HP-04	7/5/2000			1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2	2
HB-HP-05	7/5/2000			1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.3	0.3
HB-HP-06	7/6/2000			1330-20-7	XYLENES, TOTAL	Y		ug/l	700	700
HB-HP-07	7/6/2000			1330-20-7	XYLENES, TOTAL	Y		ug/l	150	150
HB-HP-08	7/6/2000			1330-20-7	XYLENES, TOTAL	Y		ug/l	130	130
HB-HP-1(WA)	12/5/1994	20	20	1330-20-7	XYLENES, TOTAL	N	U	ug/l	5	2.5
HB-HP-1(WA)	12/5/1994	35	35	1330-20-7	XYLENES, TOTAL	N	U	ug/l	5	2.5
HB-HP-2(WA)	12/7/1994	20	20	1330-20-7	XYLENES, TOTAL	N	U	ug/l	5	2.5
HB-HP-2(WA)	12/7/1994	30	30	1330-20-7	XYLENES, TOTAL	N	U	ug/l	5	2.5
HB-HP-3(WA)	12/6/1994	20	20	1330-20-7	XYLENES, TOTAL	N	U	ug/l	5	2.5
HB-HP-3(WA)	12/6/1994	35	35	1330-20-7	XYLENES, TOTAL	N	U	ug/l	5	2.5
HB-HP-4(WA)	12/7/1994	27	27	1330-20-7	XYLENES, TOTAL	Y		ug/l	54	54
HB-MW-22	3/5/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-23	3/5/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-24	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-25	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-26	3/5/2007	5	15	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.45	0.45
HB-MW-27	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-WA-03D	4/7/1992	53.5	63.5	1330-20-7	XYLENES, TOTAL	N	U	ug/l	5	2.5
HB-WA-03D	10/14/1992	53.5	63.5	1330-20-7	XYLENES, TOTAL	N	U	ug/l	3	1.5
HB-WA-03D	3/8/2007	53.5	63.5	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-WA-03I	4/7/1992	20	30	1330-20-7	XYLENES, TOTAL	Y		ug/l	40	40
HB-WA-03I	10/14/1992	20	30	1330-20-7	XYLENES, TOTAL	N	U	ug/l	300	150
HB-WA-03I	3/8/2007	20	30	1330-20-7	XYLENES, TOTAL	N	U	ug/l	10	5
HB-WA-03S	10/14/1992	3	13	1330-20-7	XYLENES, TOTAL	N	U	ug/l	300	150
HB-WA-03S	4/6/1992	3	13	1330-20-7	XYLENES, TOTAL	Y		ug/l	98	98
HB-WA-03S	3/8/2007	20	30	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	125	125
HB-WA-08D	1/5/1995	70.5	80.5	1330-20-7	XYLENES, TOTAL	N	U	ug/l	10	5
HB-WA-08D	5/21/2001	70.5	80.5	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-WA-08D	5/15/2003	70.5	80.5	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-WA-08D	5/15/2003	70.5	80.5	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-WA-08D	5/15/2003	70.5	80.5	CALCULATED	TOTAL	N	U	ug/l		5

TABLE 2.1c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SITE WIDE GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-WA-08D	8/14/2003	70.5	80.5	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-WA-08D	8/14/2003	70.5	80.5	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-WA-08D	8/14/2003	70.5	80.5	CALCULATED	TOTAL	N	U	ug/l		5
HB-WA-08D	3/12/2007	70.5	80.5	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-WA-08I	1/5/1995	30.5	40.5	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	7	7
HB-WA-08I	5/21/2001	30.5	40.5	1330-20-7	XYLENES, TOTAL	Y		ug/l	20	20
HB-WA-08I	5/15/2003	30.5	40.5	XYLENES1314	XYLENES, M & P	N	U	ug/l	50	
HB-WA-08I	5/15/2003	30.5	40.5	95-47-6	O-XYLENE	N	U	ug/l	50	
HB-WA-08I	5/15/2003	30.5	40.5	CALCULATED	TOTAL	N	U	ug/l		50
HB-WA-08I	8/14/2003	30.5	40.5	XYLENES1314	XYLENES, M & P	Y	J	ug/l	14	
HB-WA-08I	8/14/2003	30.5	40.5	95-47-6	O-XYLENE	N	U	ug/l	50	
HB-WA-08I	8/14/2003	30.5	40.5	CALCULATED	TOTAL	Y	J	ug/l		14
HB-WA-08I	3/12/2007	30.5	40.5	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	7.5	7.5
HB-WA-08S	12/9/1994	40	40	1330-20-7	XYLENES, TOTAL	Y		ug/l	21	21
HB-WA-08S	1/5/1995	8.95	18.95	1330-20-7	XYLENES, TOTAL	N	U	ug/l	10	5
HB-WA-08S	5/21/2001	8.95	18.95	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	5	5
HB-WA-08S	5/15/2003	8.95	18.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-WA-08S	5/15/2003	8.95	18.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-WA-08S	5/15/2003	8.95	18.95	CALCULATED	TOTAL	N	U	ug/l		5
HB-WA-08S	8/14/2003	8.95	18.95	XYLENES1314	XYLENES, M & P	Y		ug/l	6.4	
HB-WA-08S	8/14/2003	8.95	18.95	95-47-6	O-XYLENE	Y	J	ug/l	3.3	
HB-WA-08S	8/14/2003	8.95	18.95	CALCULATED	TOTAL	Y		ug/l		9.7
HB-WA-08S	3/12/2007	8.95	18.95	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.29	0.29
HB-WB-BL	5/22/2001	80.5	85.5	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-WB-BL	5/21/2003	80.5	85.5	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-WB-BL	5/21/2003	80.5	85.5	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-WB-BL	5/21/2003	80.5	85.5	CALCULATED	TOTAL	N	U	ug/l		5
HB-WB-BL	8/26/2003	80.5	85.5	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-WB-BL	8/26/2003	80.5	85.5	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-WB-BL	8/26/2003	80.5	85.5	CALCULATED	TOTAL	N	U	ug/l		5
HB-WB-BL	3/14/2007	80.5	85.5	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.18	0.18
HB-WB-BU	5/21/2001	18.8	23.8	1330-20-7	XYLENES, TOTAL	Y		ug/l	3500	3500
HB-WB-BU	5/21/2003	18.8	23.8	XYLENES1314	XYLENES, M & P	Y		ug/l	2300	
HB-WB-BU	5/21/2003	18.8	23.8	95-47-6	O-XYLENE	Y		ug/l	890	
HB-WB-BU	5/21/2003	18.8	23.8	CALCULATED	TOTAL	Y		ug/l		3190
HB-WB-BU	8/26/2003	18.8	23.8	XYLENES1314	XYLENES, M & P	Y		ug/l	2100	
HB-WB-BU	8/26/2003	18.8	23.8	95-47-6	O-XYLENE	Y		ug/l	760	
HB-WB-BU	8/26/2003	18.8	23.8	CALCULATED	TOTAL	Y		ug/l		2860
HB-WB-BU	3/14/2007	18.8	23.8	1330-20-7	XYLENES, TOTAL	Y		ug/l	2440	2440

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.2a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)	
Lakeshore Area - Surface Soil	DIOXIN/FURAN (9)																
	1746-01-6	2,3,7,8-TCDD Equivalent	0.000003	0.0006	mg/kg	HB-SS-04	8/8		5.75E-04			4.26E-06	C	3.90E-06	ca	3.90E-06 Y ASL	
	METALS																
	7429-90-5	ALUMINUM	1870	24400	mg/Kg	HB-HBW-03	58/58	-	2.44E+04			7.82E+03	N	7.61E+03	nc	7.61E+03 Y ASL	
	7440-36-0	ANTIMONY	0.31 J	0.97 J	mg/Kg	HB-HB-02I	18/58	0.19-15.37	9.70E-01			3.13E+00	N	3.13E+00	nc	3.13E+00 N BSL	
	7440-38-2	ARSENIC	2.5 J	21.4	mg/Kg	HB-GP-08	58/58	-	2.14E+01		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01 Y TOX	
	7440-39-3	BARIUM	32.5	4880 J	mg/Kg	HB-SEEP-2	58/58	-	4.88E+03		4.00E+02	1.56E+03	N	5.37E+02	nc	5.37E+02 Y ASL	
	7440-41-7	BERYLLIUM	0.3 J	1.4	mg/Kg	HB-HBW-01	33/58	0.6-1.28	1.40E+00		7.20E+01	1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL	
	7440-43-9	CADMIUM	0.055 J	110 J	mg/Kg	HB-SS-06	48/58	0.6-1.17	1.10E+02		4.30E+00	3.91E+00	N	3.70E+00	nc	3.70E+00 Y ASL	
	7440-70-2	CALCIUM	59100	352000	mg/Kg	HB-SS-08	58/58	-	3.52E+05			NV	NV	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	6.7	391 J	mg/Kg	HB-SS-06	58/58	-	3.91E+02		1.10E+02	2.35E+01	N	3.01E+00	nc	3.01E+00 Y TOX	
	7440-48-4	COBALT	3.5 J	13.3 J	mg/Kg	HB-GP-20	36/58	7.21-12.81	1.33E+01			NV	9.03E+01	nc	9.03E+01 N BSL		
	7440-50-8	COPPER	13.4	744 J	mg/Kg	HB-SS-06	58/58	-	7.44E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02 Y ASL	
	57-12-5	CYANIDE	0.76	5.6 J	mg/Kg	HB-GP-06	23/58	0.51-2.64	5.60E+00			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL	
	7439-89-6	IRON	2690	24400 J	mg/Kg	HB-HBW-04	58/58	-	2.44E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL	
	7439-92-1	LEAD	6.8 J	1800 J	mg/Kg	HB-GP-06	58/58	-	1.80E+03			NV	4.00E+02	nc	4.00E+02 Y ASL		
	7439-95-4	MAGNESIUM	3600	36300	mg/Kg	HB-GP-20	58/58	-	3.83E+04			NV	NV	NV	N	NUT	
	7439-96-5	MANGANESE	128	722	mg/Kg	HB-GP-05	58/58	-	7.22E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL	
	7439-97-6	MERCURY ^b	0.09	64.3	mg/Kg	HB-SS-08	58/58	-	6.43E+01			2.35E+00	N	2.35E+00	nc	2.35E+00 Y ASL	
	22967-92-6	METHYL MERCURY	0.001	0.096	mg/Kg	HB-SS-08	12/12	-	9.61E+01			7.82E+02	N	6.11E+02	nc	6.11E+02 N BSL	
	7440-02-0	NICKEL	10.1	72.3 J	mg/Kg	HB-GP-06	58/58	-	7.23E+01		3.10E+02	1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL	
	7440-09-7	POTASSIUM	275 J	8170 J	mg/Kg	HB-HBW-03	52/58	323.56-448	8.17E+03			NV	NV	NV	N	NUT	
	7782-49-2	SELENIUM	0.35 J	3.3	mg/Kg	HB-GP-10	36/58	0.6-5.95	3.30E+00		1.80E+02	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL	
	7440-22-4	SILVER	0.28 J	91.9 J	mg/Kg	HB-SS-06	36/58	0.076-2.33	9.19E+01		1.80E+02	3.91E+01	N	3.91E+01	nc	3.91E+01 Y ASL	
	7440-23-5	SODIUM	326 J	4910 J	mg/Kg	HB-RISB-01	57/58	568-568	4.91E+03			NV	NV	NV	N	NUT	
	7440-28-0	THALLIUM	0.57 J	2.3	mg/Kg	HB-HBW-05	12/58	0.5-3.3	2.30E+00			5.48E-01	N	5.16E-01	nc	5.16E-01 Y ASL	
	7440-62-2	VANADIUM	8.1 J	49.1	mg/Kg	HB-HBW-03	54/58	8.09-11.66	4.91E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL	
	7440-66-6	ZINC	14.8	1520 J	mg/Kg	HB-SS-06	58/58	-	1.52E+03		1.00E+04	2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL	
	PCBs																
		LESS CHLORINATED PCBs ^c	0.03	0.4	mg/kg	HB-GP-08	8/58	0.003-5.3	4.00E-01			5.48E-01	N	3.93E-01	nc	3.93E-01 Y ASL	
		HIGHLY CHLORINATED PCBs ^d	0.02	6	mg/kg	HB-GP-06	46/58	0.003-6	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL	
		TOTAL PCBs ^e	0.02	6	mg/kg	HB-GP-06	46/58	0.003-6	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL	
	PESTICIDES																
	50-29-3	4,4'-DDT	0.016 J	0.016 J	mg/kg	HB-SS-10	1/58	0.003-0.4	1.60E-02		7.90E+00	1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL	
	57-74-9	TOTAL CHLORDANE ^f	0.002 J	0.38 J	mg/kg	HB-GP-06	17/58	0.002-0.09	3.80E-01			1.82E+00	C	1.62E+00	ca	1.62E+00 N BSL	
	60-57-1	DIELDRIN	0.2 J	0.2 J	mg/kg	HB-GP-07	1/58	0.003-0.4	2.00E-01		2.00E-01	3.99E-02	C	3.04E-02	ca	3.04E-02 Y ASL	
	1031-07-8	ENDOSULFAN SULFATE ^g	0.2	0.2	mg/kg	HB-HB-05I	1/58	0.003-0.4	2.00E-01		2.40E+01	4.69E+01	N	3.67E+01	nc	3.67E+01 N BSL	
	7421-93-4	ENDRIN ALDEHYDE ^h	0.012 J	0.043 J	mg/kg	HB-HB-16D	2/58	0.003-0.4	4.30E-02			2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL	
	53494-70-5	ENDRIN KETONE ^h	0.39	0.56	mg/kg	HB-SS-11	2/58	0.003-0.4	5.60E-01			2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL	
	SVOCs																
	105-67-9	2,4-DIMETHYLPHENOL	0.076 J	7.3 J	mg/kg	HB-SEEP-2	4/57	0.34-180	7.30E+00			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL	
	51-28-5	2,4-DINITROPHENOL	0.067 J	0.067 J	mg/kg	HB-GP-05	1/55	1.7-910	6.70E-02			1.56E+01	N	1.22E+01	nc	1.22E+01 N BSL	
	91-57-6	2-METHYLNAPHTHALENE	0.06 J	130 J	mg/kg	HB-GP-01	33/57	0.34-180	1.30E+02			3.13E+01	N	NV	nc	3.13E+01 Y ASL	
	95-48-7	2-METHYLPHENOL	2 J	9.2 J	mg/kg	HB-SEEP-2	3/57	0.34-180	9.20E+00		1.00E+02	3.91E+02	N	3.06E+02	nc	3.06E+02 N BSL	
	34METPH	3,4-METHYLPHENOL ⁱ	0.091 J	19 J	mg/kg	HB-SEEP-2	6/56	0.34-180	1.90E+01			3.91E+01	N	3.06E+01	nc	3.06E+01 N BSL	
	106-47-8	4-CHLOROANILINE	0.12 J	1.7 J	mg/kg	HB-SS-05	16/57	0.34-180	1.70E+00			3.13E+01	N	2.44E+01	nc	2.44E+01 N BSL	
	83-32-9	ACENAPHTHENE	0.064 J	31 J	mg/kg	HB-GP-01	21/57	0.34-180	3.10E+01		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02 N BSL	
	208-96-8	ACENAPHTHYLENE	0.088 J	37 J	mg/kg	HB-GP-01	41/57	0.34-180	3.70E+01			NV	NV	NV	Y	NTX	
	120-12-7	ANTHRACENE	0.08 J	28 J	mg/kg	HB-GP-01	42/57	0.34-180	2.80E+01			1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03 N BSL
	56-55-3	BENZ(A)ANTHRACENE	0.063 J	6.9 J	mg/kg	HB-GP-01	49/57	0.53-180	6.90E+00		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL	
	50-32-8	BENZO(A)PYRENE	0.06 J	6.4	mg/kg	HB-HBW-01	49/58	0.57-180	6.40E+00		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL	
	205-99-2	BENZO(B)FLUORANTHENE	0.071 J	9.5	mg/kg	HB-HBW-01	50/58	0.57-180	9.50E+00		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL	
	191-24-2	BENZO(G,H,I)PERYLENE	0.042 J	4.7	mg/kg	HB-SS-10	44/57	0.53-180	4.70E+00		1.00E+02	NV	NV	NV	Y	NTX	
	207-08-9	BENZO(K)FLUORANTHENE	0.061 J	5	mg/kg	HB-SS-10	47/57	0.53-180	5.00E+00		3.90E+00	2.20E+00	C	6.21E+00	ca	2.20E+00 Y ASL	
	65-85-0	BENZOIC ACID	0.074 J	3 J	mg/kg	HB-GP-09	18/29	1.7-29	3.00E+00			3.13E+04	N	1.00E+04	nc	1.00E+04 N BSL	
	100-51-6	BENZYL ALCOHOL	0.066 J	0.13 J	mg/kg	HB-GP-14	2/56	0.34-180	1.30E-01			3.91E+03	N	1.83E+03	nc	1.83E+03 N BSL	

TABLE 2.2a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	111-44-4	BIS(2-CHLOROETHYL)ETHER	0.05 J	0.05 J	mg/kg	HB-HBW-06	1/57	0.34-180	5.00E-02			5.81E-01	C	2.18E-01	ca	2.18E-01	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.11 J	4.4 J	mg/kg	HB-HB-021	7/57	0.069-180	4.40E+00			4.56E+01	C	3.47E+01	ca	3.47E+01	N	BSL
	85-68-7	BUTYLBENZYL PHTHALATE	0.11 J	0.2 J	mg/kg	HB-HBW-03	3/57	0.34-180	2.00E-01			1.56E+03	N	1.22E+03	nc	1.22E+03	N	BSL
	86-74-8	CARBAZOLE	0.065 J	15 J	mg/kg	HB-GP-01	28/57	0.34-180	1.50E+01			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL
	218-01-9	CHRYSENE	0.07 J	6.1	mg/kg	HB-HBW-01	51/58	0.53-180	6.10E+00		3.90E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	N	BSL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.072 J	1.4 J	mg/kg	HB-SS-10	30/57	0.34-180	1.40E+00		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.051 J	53 J	mg/kg	HB-GP-01	23/57	0.34-180	5.30E+01		5.90E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.047 J	0.22 J	mg/kg	HB-GP-07	7/57	0.34-180	2.20E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	117-84-0	DI-N-OCTYL PHTHALATE	0.3 J	0.3 J	mg/kg	HB-HBW-03	1/57	0.34-180	3.00E-01			NV		2.44E+02	nc	2.44E+02	N	BSL
	206-44-0	FLUORANTHENE	0.076 J	43 J	mg/kg	HB-GP-01	54/58	2.9-180	4.30E+01		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	86-73-7	FLUORENE	0.05 J	61 J	mg/kg	HB-GP-01	20/57	0.34-180	6.10E+01		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	118-74-1	HEXACHLOROBENZENE	0.083 J	4.1 J	mg/kg	HB-HBW-02	11/57	0.4-180	4.10E+00		1.20E+00	3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.047 J	4.1	mg/kg	HB-SS-10	45/57	0.53-180	4.10E+00		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.034 J	300 J	mg/kg	HB-GP-01	45/85	0.004-160	3.00E+02		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	87-86-5	PENTACHLOROPHENOL	0.13 J	0.13 J	mg/kg	HB-GP-05	1/55	1.7-910	1.30E-01		6.70E+00	5.32E+00	C	2.98E+00	ca	2.98E+00	N	BSL
	85-01-8	PHENANTHRENE	0.065 J	120 J	mg/kg	HB-GP-01	49/58	0.53-180	1.20E+02		1.00E+02	NV		NV		NV	Y	NTX
	108-95-2	PHENOL	0.055 J	26 J	mg/kg	HB-SEEP-2	10/57	0.34-180	2.60E+01		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.058 J	26 J	mg/kg	HB-GP-01	54/58	2.9-180	2.60E+01		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																	
	120-82-1	1,2,4-TRICHLOROBENZENE	0.002 J	48 J	mg/kg	HB-SS-11	26/86	0.005-180	4.80E+01			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.006 J	0.006 J	mg/kg	HB-HBW-04	1/28	0.003-0.006	6.00E-03		5.20E+01	NV		5.16E+00	nc	5.16E+00	N	BSL
	95-50-1	1,2-DICHLOROBENZENE	0.001 J	210	mg/kg	HB-SS-11	41/87	0.003-8.7	2.10E+02		1.00E+02	7.04E+02	N	6.00E+01	nc	6.00E+01	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.003 J	0.003 J	mg/kg	HB-HBW-04	1/28	0.003-0.006	3.00E-03		5.20E+01	NV		2.13E+00	nc	2.13E+00	N	BSL
	541-73-1	1,3-DICHLOROBENZENE	0.001 J	7	mg/kg	HB-SB-65	9/86	0.003-180	7.00E+00		4.90E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.002 J	350	mg/kg	HB-SS-11	49/87	0.003-8.7	3.50E+02		1.30E+01	2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL
	78-93-3	2-BUTANONE	0.001 J	0.023 J	mg/kg	HB-RISB-01	6/58	0.01-2.9	2.30E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.007 J	0.17 J	mg/kg	HB-SS-09	8/57	0.01-5.9	1.70E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.001 J	4.2 J	mg/kg	HB-SEEP-2	15/58	0.003-0.022	4.20E+00		4.80E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.0026 J	0.11 J	mg/kg	HB-SB-65	2/26	0.012-2.9	1.10E-01			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL
	108-90-7	CHLOROBENZENE	0.001 J	3.4	mg/kg	HB-SB-65	21/58	0.003-0.022	3.40E+00		1.00E+02	1.56E+02	N	1.51E+01	nc	1.51E+01	N	BSL
	100-41-4	ETHYLBENZENE	0.005 J	0.57 J	mg/kg	HB-SEEP-2	6/57	0.003-0.022	5.70E-01		4.10E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.13 J	0.13 J	mg/kg	HB-SB-65	1/29	0.003-0.006	1.30E-01			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
108-87-2	METHYLCYCLOHEXANE	0.33 J	0.33 J	mg/kg	HB-SB-65	1/1	-	3.30E-01			NV		2.59E+02	nc	2.59E+02	N	BSL	
75-09-2	METHYLENE CHLORIDE	0.002 J	0.16	mg/kg	HB-HBW-01	2/58	0.002-1.5	1.60E-01		1.00E+02	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL	
99-87-6	P-ISOPROPYLTOLUENE	0.002 J	0.002 J	mg/kg	HB-GP-12	1/29	0.003-0.006	2.00E-03			NV		NV		NV	Y	NTX	
135-98-8	SEC-BUTYLBENZENE	0.002 J	0.002 J	mg/kg	HB-HBW-04	1/28	0.003-0.006	2.00E-03		1.00E+02	NV		2.20E+01	nc	2.20E+01	N	BSL	
100-42-5	STYRENE	1.2 J	2.4 J	mg/kg	HB-SEEP-2	2/58	0.003-0.42	2.40E+00			1.56E+03	N	1.70E+02	nc	1.70E+02	N	BSL	
108-88-3	TOLUENE	0.001 J	9.4 J	mg/kg	HB-SEEP-2	14/58	0.003-0.022	9.40E+00		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL	
79-01-6	TRICHLOROETHENE	0.002 J	0.002 J	mg/kg	HB-HBW-04	1/58	0.003-1.5	2.00E-03		2.10E+01	1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL	
1330-20-7	XYLENES, TOTAL	0.001 J	12.4 J	mg/kg	HB-SEEP-2	16/58	0.0015-0.022	1.24E+01		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL	

Footnotes:
(1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) Values are from New York Subpart 375-6 Soil Cleanup Objectives. Values reflect residential restricted use for the protection of human health.
(5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.2b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for mercury compounds utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
f = Where criteria are not available, RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
g = RBC and PRG values for Endosulfan (CAS# 115297) utilized.
h = RC and PRG values for Endrin (CAS# 72208) utilized.
i = RBC and PRG values for 4-methylphenol (CAS# 106445) utilized.

Definitions:
ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstracts Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals; USEPA, 2004
RBC: Risk Based Concentration; USEPA, October 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.2b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-RISB-02	12/13/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	18.458	18.458	ng/kg	J	0.01	0.185
HB-RISB-02	12/13/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	13.331	13.331	ng/kg	J	0.01	0.133
HB-RISB-02	12/13/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.801	2.801	ng/kg	J	0.01	0.028
HB-RISB-02	12/13/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.596	0.596	ng/kg	EMPC	0.1	0.060
HB-RISB-02	12/13/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	9.566	9.566	ng/kg	J	0.1	0.957
HB-RISB-02	12/13/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.966	1.966	ng/kg	J	0.1	0.197
HB-RISB-02	12/13/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.385	2.385	ng/kg	J	0.1	0.239
HB-RISB-02	12/13/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	0.875	0.875	ng/kg	EMPC	0.1	0.088
HB-RISB-02	12/13/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.958	1.958	ng/kg	EMPC	0.1	0.196
HB-RISB-02	12/13/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.68	0.68	ng/kg	J	1	0.680
HB-RISB-02	12/13/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	5.239	5.239	ng/kg	J	0.03	0.157
HB-RISB-02	12/13/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-RISB-02	12/13/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	12.75	12.75	ng/kg	J	0.1	1.275
HB-RISB-02	12/13/2002	0	0.5	3268-87-9	OCDD	Y	96.811	96.811	ng/kg	J	0.0003	0.029
HB-RISB-02	12/13/2002	0	0.5	39001-02-0	OCDF	Y	46.831	46.831	ng/kg	J	0.0003	0.014
Sample Location TEQ =												4.7
HB-RISB-02	12/13/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	9.541	9.541	ng/kg		0.01	0.095
HB-RISB-02	12/13/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	7.187	7.187	ng/kg		0.01	0.072
HB-RISB-02	12/13/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.445	1.445	ng/kg	J	0.01	0.014
HB-RISB-02	12/13/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-02	12/13/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	6.501	6.501	ng/kg		0.1	0.650
HB-RISB-02	12/13/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.852	0.852	ng/kg	J	0.1	0.085
HB-RISB-02	12/13/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.59	1.59	ng/kg	J	0.1	0.159
HB-RISB-02	12/13/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-02	12/13/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-02	12/13/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-RISB-02	12/13/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	4.574	4.574	ng/kg		0.03	0.137
HB-RISB-02	12/13/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-RISB-02	12/13/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	12.846	12.846	ng/kg		0.1	1.285
HB-RISB-02	12/13/2002	0.5	1	3268-87-9	OCDD	Y	66.844	66.844	ng/kg	J	0.0003	0.020
HB-RISB-02	12/13/2002	0.5	1	39001-02-0	OCDF	Y	20.584	20.584	ng/kg	J	0.0003	0.006
Sample Location TEQ =												4.6

TABLE 2.2b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SEEP-2	9/9/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.504	8.504	ng/kg	J	0.01	0.085
HB-SEEP-2	9/9/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	UJ	0.01	0.013
HB-SEEP-2	9/9/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.025	2.025	ng/kg	J	0.1	0.203
HB-SEEP-2	9/9/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.688	3.688	ng/kg	J	0.1	0.369
HB-SEEP-2	9/9/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.628	0.628	ng/kg	J	0.1	0.063
HB-SEEP-2	9/9/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.442	1.442	ng/kg	J	0.1	0.144
HB-SEEP-2	9/9/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-SEEP-2	9/9/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.087	1.087	ng/kg	J	0.03	0.033
HB-SEEP-2	9/9/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-SEEP-2	9/9/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.859	2.859	ng/kg	EMPC	0.1	0.286
HB-SEEP-2	9/9/2003	0	0.5	3268-87-9	OCDD	Y	260.17	260.17	ng/kg	J	0.0003	0.078
HB-SEEP-2	9/9/2003	0	0.5	39001-02-0	OCDF	Y	19.606	19.606	ng/kg	J	0.0003	0.006
Sample Location TEQ =												3.3
HB-SEEP-2	9/9/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	3.127	3.127	ng/kg	J	0.01	0.031
HB-SEEP-2	9/9/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	UJ	0.01	0.013
HB-SEEP-2	9/9/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.531	1.531	ng/kg	J	0.1	0.153
HB-SEEP-2	9/9/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-SEEP-2	9/9/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	UJ	0.03	0.038
HB-SEEP-2	9/9/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-SEEP-2	9/9/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	7.27	7.27	ng/kg	J	0.1	0.727
HB-SEEP-2	9/9/2003	0.5	1	3268-87-9	OCDD	Y	66.684	66.684	ng/kg	J	0.0003	0.020
HB-SEEP-2	9/9/2003	0.5	1	39001-02-0	OCDF	Y	4.583	4.583	ng/kg	EMPC	0.0003	0.001
Sample Location TEQ =												3.4

TABLE 2.2b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 ft)

	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
Sample Location												
HB-SS-04	12/3/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	1186.223	1186.223	ng/kg		0.01	11.862
HB-SS-04	12/3/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	274.059	274.059	ng/kg		0.01	2.741
HB-SS-04	12/3/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	64.442	64.442	ng/kg		0.01	0.644
HB-SS-04	12/3/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	210.432	210.432	ng/kg		0.1	21.043
HB-SS-04	12/3/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	437.121	437.121	ng/kg		0.1	43.712
HB-SS-04	12/3/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	446.742	446.742	ng/kg		0.1	44.674
HB-SS-04	12/3/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	125.718	125.718	ng/kg	J	0.1	12.572
HB-SS-04	12/3/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	282.125	282.125	ng/kg		0.1	28.213
HB-SS-04	12/3/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	69.159	69.159	ng/kg	J	0.1	6.916
HB-SS-04	12/3/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	275.231	275.231	ng/kg		1	275.231
HB-SS-04	12/3/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	352.347	352.347	ng/kg		0.03	10.570
HB-SS-04	12/3/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	31.176	31.176	ng/kg		1	31.176
HB-SS-04	12/3/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	855.5	855.5	ng/kg	J	0.1	85.550
HB-SS-04	12/3/2002	0	0.5	3268-87-9	OCDD	Y	1354.982	1354.982	ng/kg	J	0.0003	0.406
HB-SS-04	12/3/2002	0	0.5	39001-02-0	OCDF	Y	496.581	496.581	ng/kg	J	0.0003	0.149
Sample Location TEQ =												575.5
HB-SS-04	12/3/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	1083.071	1083.071	ng/kg		0.01	10.831
HB-SS-04	12/3/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	331.513	331.513	ng/kg		0.01	3.315
HB-SS-04	12/3/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	81.67	81.67	ng/kg		0.01	0.817
HB-SS-04	12/3/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	167.275	167.275	ng/kg		0.1	16.728
HB-SS-04	12/3/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	598.716	598.716	ng/kg		0.1	59.872
HB-SS-04	12/3/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	390.763	390.763	ng/kg		0.1	39.076
HB-SS-04	12/3/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	153.225	153.225	ng/kg		0.1	15.323
HB-SS-04	12/3/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	269.866	269.866	ng/kg		0.1	26.987
HB-SS-04	12/3/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	107.172	107.172	ng/kg		0.1	10.717
HB-SS-04	12/3/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	237.276	237.276	ng/kg		1	237.276
HB-SS-04	12/3/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	374.758	374.758	ng/kg		0.03	11.243
HB-SS-04	12/3/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	25.347	25.347	ng/kg		1	25.347
HB-SS-04	12/3/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	793	793	ng/kg	J	0.1	79.300
HB-SS-04	12/3/2002	0.5	1	3268-87-9	OCDD	Y	1447.067	1447.067	ng/kg	J	0.0003	0.434
HB-SS-04	12/3/2002	0.5	1	39001-02-0	OCDF	Y	485.628	485.628	ng/kg	J	0.0003	0.146
Sample Location TEQ =												537.4

TABLE 2.2b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SS-11	12/4/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	528.876	528.876	ng/kg		0.01	5.289
HB-SS-11	12/4/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	201.137	201.137	ng/kg		0.01	2.011
HB-SS-11	12/4/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	57.162	57.162	ng/kg		0.01	0.572
HB-SS-11	12/4/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	90.395	90.395	ng/kg		0.1	9.040
HB-SS-11	12/4/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	348.307	348.307	ng/kg		0.1	34.831
HB-SS-11	12/4/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	218.606	218.606	ng/kg		0.1	21.861
HB-SS-11	12/4/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	99.834	99.834	ng/kg		0.1	9.983
HB-SS-11	12/4/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	129.133	129.133	ng/kg		0.1	12.913
HB-SS-11	12/4/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	65.808	65.808	ng/kg		0.1	6.581
HB-SS-11	12/4/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	129.77	129.77	ng/kg		1	129.770
HB-SS-11	12/4/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	214.534	214.534	ng/kg		0.03	6.436
HB-SS-11	12/4/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	16.073	16.073	ng/kg		1	16.073
HB-SS-11	12/4/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	299.1	299.1	ng/kg		0.1	29.910
HB-SS-11	12/4/2002	0	0.5	3268-87-9	OCDD	Y	647.321	647.321	ng/kg	J	0.0003	0.194
Sample Location TEQ =												285.5
HB-SS-11	12/4/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	62.134	62.134	ng/kg	EMPC	0.01	0.621
HB-SS-11	12/4/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	63.752	63.752	ng/kg		0.01	0.638
HB-SS-11	12/4/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	16.645	16.645	ng/kg		0.01	0.166
HB-SS-11	12/4/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.298	6.298	ng/kg		0.1	0.630
HB-SS-11	12/4/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	108.398	108.398	ng/kg	EMPC	0.1	10.840
HB-SS-11	12/4/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	19.388	19.388	ng/kg		0.1	1.939
HB-SS-11	12/4/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	34.666	34.666	ng/kg		0.1	3.467
HB-SS-11	12/4/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	11.339	11.339	ng/kg		0.1	1.134
HB-SS-11	12/4/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	17.594	17.594	ng/kg	EMPC	0.1	1.759
HB-SS-11	12/4/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	12.855	12.855	ng/kg		1	12.855
HB-SS-11	12/4/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	63.962	63.962	ng/kg		0.03	1.919
HB-SS-11	12/4/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.975	1.975	ng/kg		1	1.975
HB-SS-11	12/4/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	80.2	80.2	ng/kg	J	0.1	8.020
HB-SS-11	12/4/2002	0.5	1	3268-87-9	OCDD	Y	133.435	133.435	ng/kg		0.0003	0.040
HB-SS-11	12/4/2002	0.5	1	39001-02-0	OCDF	Y	75.28	75.28	ng/kg		0.0003	0.023
Sample Location TEQ =												46.0

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PECDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HXCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HPCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable

EMPC = Estimated Maximum Possible Concentration

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.2c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-GP-01	0	0.17	7/14/2000	0.2	mg/kg
Highly Chlorinated PCBs	HB-GP-02	0	0.17	7/7/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-03	0	0.17	7/7/2000	3	mg/kg
Highly Chlorinated PCBs	HB-GP-04	0	0.17	7/10/2000	0.5	mg/kg
Highly Chlorinated PCBs	HB-GP-05	0	0.17	7/10/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-06	0	0.17	7/10/2000	6	mg/kg
Highly Chlorinated PCBs	HB-GP-07	0	0.17	7/10/2000	4	mg/kg
Highly Chlorinated PCBs	HB-GP-08	0	0.17	7/10/2000	0.4	mg/kg
Highly Chlorinated PCBs	HB-GP-09	0	0.17	7/12/2000	1	mg/kg
Highly Chlorinated PCBs	HB-GP-10	0	0.17	7/12/2000	1	mg/kg
Highly Chlorinated PCBs	HB-GP-11	0	0.17	7/13/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-12	0	0.17	7/13/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-13	0	0.17	7/13/2000	0.3	mg/kg
Highly Chlorinated PCBs	HB-GP-14	0	0.17	7/14/2000	0.2	mg/kg
Highly Chlorinated PCBs	HB-GP-15	0	0.17	7/14/2000	1	mg/kg
Highly Chlorinated PCBs	HB-GP-16	0	0.17	7/18/2000	0.1	mg/kg
Highly Chlorinated PCBs	HB-GP-17	0	0.17	7/18/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-18	0	0.17	7/17/2000	0.9	mg/kg
Highly Chlorinated PCBs	HB-GP-19	0	0.17	7/17/2000	0.03	mg/kg
Highly Chlorinated PCBs	HB-GP-20	0	0.17	7/17/2000	0.02	mg/kg
Highly Chlorinated PCBs	HB-HB-02I	0	0.17	7/19/2000	0.9	mg/kg
Highly Chlorinated PCBs	HB-HB-03S	0	0.17	7/26/2000	0.02	mg/kg
Highly Chlorinated PCBs	HB-HB-04S	0	0.17	7/27/2000	0.1	mg/kg
Highly Chlorinated PCBs	HB-HB-05I	0	0.17	7/27/2000	3	mg/kg
Highly Chlorinated PCBs	HB-HB-06S	0	0.17	8/2/2000	0.5	mg/kg
Highly Chlorinated PCBs	HB-HB-16D	0	0.5	1/7/2003	0.29	mg/kg
Highly Chlorinated PCBs	HB-HB-16D	0.5	1	1/7/2003	1.1	mg/kg
Highly Chlorinated PCBs	HB-HBW-01	0	0.17	8/4/2000	0.6	mg/kg
Highly Chlorinated PCBs	HB-HBW-02	0	0.17	8/4/2000	1	mg/kg
Highly Chlorinated PCBs	HB-HBW-03	0	0.17	8/7/2000	0.06	mg/kg
Highly Chlorinated PCBs	HB-HBW-04	0	0.17	8/7/2000	0.08	mg/kg
Highly Chlorinated PCBs	HB-HBW-05	0	0.17	8/8/2000	0.3	mg/kg
Highly Chlorinated PCBs	HB-HBW-06	0	0.17	8/8/2000	0.08	mg/kg
Highly Chlorinated PCBs	HB-RISB-01	0	0.5	12/13/2002	0.2	mg/kg
Highly Chlorinated PCBs	HB-RISB-02	0	0.5	12/13/2002	0.15	mg/kg
Highly Chlorinated PCBs	HB-SB-65	0	2	11/3/2006	1.55	mg/kg
Highly Chlorinated PCBs	HB-SS-04	0	0.5	12/3/2002	0.49	mg/kg
Highly Chlorinated PCBs	HB-SS-04	0.5	1	12/3/2002	0.43	mg/kg
Highly Chlorinated PCBs	HB-SS-05	0	0.5	12/3/2002	3.7	mg/kg
Highly Chlorinated PCBs	HB-SS-05	0.5	1	12/3/2002	3	mg/kg
Highly Chlorinated PCBs	HB-SS-06	0	0.5	12/3/2002	3.6	mg/kg
Highly Chlorinated PCBs	HB-SS-06	0.5	1	12/3/2002	2.5	mg/kg
Highly Chlorinated PCBs	HB-SS-07	0	0.5	12/5/2002	0.75	mg/kg
Highly Chlorinated PCBs	HB-SS-09	0.5	1	12/4/2002	2.7	mg/kg

TABLE 2.2c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-SS-11	0.5	1	12/4/2002	2.2	mg/kg
Highly Chlorinated PCBs	HB-TP-09	2	2	7/5/2000	0.2	mg/kg
Less Chlorinated PCBs	HB-GP-08	0	0.17	7/10/2000	0.4	mg/kg
Less Chlorinated PCBs	HB-GP-09	0	0.17	7/12/2000	0.03	mg/kg
Less Chlorinated PCBs	HB-GP-11	0	0.17	7/13/2000	0.09	mg/kg
Less Chlorinated PCBs	HB-GP-12	0	0.17	7/13/2000	0.08	mg/kg
Less Chlorinated PCBs	HB-GP-13	0	0.17	7/13/2000	0.03	mg/kg
Less Chlorinated PCBs	HB-GP-14	0	0.17	7/14/2000	0.03	mg/kg
Less Chlorinated PCBs	HB-GP-15	0	0.17	7/14/2000	0.04	mg/kg
Less Chlorinated PCBs	HB-HB-02I	0	0.17	7/19/2000	0.1	mg/kg
Total PCBs	HB-GP-01	0	0.17	7/14/2000	0.2	mg/kg
Total PCBs	HB-GP-02	0	0.17	7/7/2000	2	mg/kg
Total PCBs	HB-GP-03	0	0.17	7/7/2000	3	mg/kg
Total PCBs	HB-GP-04	0	0.17	7/10/2000	0.5	mg/kg
Total PCBs	HB-GP-05	0	0.17	7/10/2000	2	mg/kg
Total PCBs	HB-GP-06	0	0.17	7/10/2000	6	mg/kg
Total PCBs	HB-GP-07	0	0.17	7/10/2000	4	mg/kg
Total PCBs	HB-GP-08	0	0.17	7/10/2000	0.8	mg/kg
Total PCBs	HB-GP-09	0	0.17	7/12/2000	1.03	mg/kg
Total PCBs	HB-GP-10	0	0.17	7/12/2000	1	mg/kg
Total PCBs	HB-GP-11	0	0.17	7/13/2000	2.09	mg/kg
Total PCBs	HB-GP-12	0	0.17	7/13/2000	2.08	mg/kg
Total PCBs	HB-GP-13	0	0.17	7/13/2000	0.33	mg/kg
Total PCBs	HB-GP-14	0	0.17	7/14/2000	0.23	mg/kg
Total PCBs	HB-GP-15	0	0.17	7/14/2000	1.04	mg/kg
Total PCBs	HB-GP-16	0	0.17	7/18/2000	0.1	mg/kg
Total PCBs	HB-GP-17	0	0.17	7/18/2000	2	mg/kg
Total PCBs	HB-GP-18	0	0.17	7/17/2000	0.9	mg/kg
Total PCBs	HB-GP-19	0	0.17	7/17/2000	0.03	mg/kg
Total PCBs	HB-GP-20	0	0.17	7/17/2000	0.02	mg/kg
Total PCBs	HB-HB-02I	0	0.17	7/19/2000	1	mg/kg
Total PCBs	HB-HB-03S	0	0.17	7/26/2000	0.02	mg/kg
Total PCBs	HB-HB-04S	0	0.17	7/27/2000	0.1	mg/kg
Total PCBs	HB-HB-05I	0	0.17	7/27/2000	3	mg/kg
Total PCBs	HB-HB-06S	0	0.17	8/2/2000	0.5	mg/kg
Total PCBs	HB-HB-16D	0	0.5	1/7/2003	0.29	mg/kg
Total PCBs	HB-HB-16D	0.5	1	1/7/2003	1.1	mg/kg
Total PCBs	HB-HBW-01	0	0.17	8/4/2000	0.6	mg/kg
Total PCBs	HB-HBW-02	0	0.17	8/4/2000	1	mg/kg
Total PCBs	HB-HBW-03	0	0.17	8/7/2000	0.06	mg/kg
Total PCBs	HB-HBW-04	0	0.17	8/7/2000	0.08	mg/kg
Total PCBs	HB-HBW-05	0	0.17	8/8/2000	0.3	mg/kg

TABLE 2.2c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-HBW-06	0	0.17	8/8/2000	0.08	mg/kg
Total PCBs	HB-RISB-01	0	0.5	12/13/2002	0.2	mg/kg
Total PCBs	HB-RISB-02	0	0.5	12/13/2002	0.15	mg/kg
Total PCBs	HB-SB-65	0	2	11/3/2006	1.55	mg/kg
Total PCBs	HB-SS-04	0	0.5	12/3/2002	0.49	mg/kg
Total PCBs	HB-SS-04	0.5	1	12/3/2002	0.43	mg/kg
Total PCBs	HB-SS-05	0	0.5	12/3/2002	3.7	mg/kg
Total PCBs	HB-SS-05	0.5	1	12/3/2002	3	mg/kg
Total PCBs	HB-SS-06	0	0.5	12/3/2002	3.6	mg/kg
Total PCBs	HB-SS-06	0.5	1	12/3/2002	2.5	mg/kg
Total PCBs	HB-SS-07	0	0.5	12/5/2002	0.75	mg/kg
Total PCBs	HB-SS-09	0.5	1	12/4/2002	2.7	mg/kg
Total PCBs	HB-SS-11	0.5	1	12/4/2002	2.2	mg/kg
Total PCBs	HB-TP-09	2	2	7/5/2000	0.2	mg/kg

Notes:

* Less chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher, if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.2d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-GP-01	7/14/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-01	7/14/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-02	7/7/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.1
HB-GP-02	7/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.1
Total Chlordane =									0.1
HB-GP-03	7/7/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.1
HB-GP-03	7/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.1
Total Chlordane =									0.1
HB-GP-04	7/10/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.02
HB-GP-04	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.01
Total Chlordane =									0.01
HB-GP-05	7/10/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.01
HB-GP-05	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.05
Total Chlordane =									0.06
HB-GP-06	7/10/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.08
HB-GP-06	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.3
Total Chlordane =									0.38
HB-GP-07	7/10/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-GP-07	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.2
Total Chlordane =									0.25
HB-GP-08	7/10/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.04
HB-GP-08	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.04
Total Chlordane =									ND
HB-GP-09	7/12/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-GP-09	7/12/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									0.05
HB-GP-10	7/12/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-GP-10	7/12/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND
HB-GP-11	7/13/2000	0	0.17	57-74-9	CHLORDANE	Y		mg/kg	0.07
HB-GP-11	7/13/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.07
Total Chlordane =									0.07
HB-GP-12	7/13/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-GP-12	7/13/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									0.05
HB-GP-13	7/13/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-13	7/13/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-14	7/14/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-14	7/14/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND

TABLE 2.2d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-GP-15	7/14/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-GP-15	7/14/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND
HB-GP-16	7/18/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-16	7/18/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-17	7/18/2000	0	0.17	57-74-9	CHLORDANE	N	UJ	mg/kg	0.07
HB-GP-17	7/18/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.08
Total Chlordane =									0.08
HB-GP-18	7/17/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-GP-18	7/17/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.04
Total Chlordane =									0.04
HB-GP-19	7/17/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-19	7/17/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-20	7/17/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-20	7/17/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-HB-02I	7/19/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-HB-02I	7/19/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.09
Total Chlordane =									0.14
HB-HB-03S	7/26/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-HB-03S	7/26/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.005
Total Chlordane =									0.005
HB-HB-04S	7/27/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.04
HB-HB-04S	7/27/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.04
Total Chlordane =									ND
HB-HB-05I	7/27/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.04
HB-HB-05I	7/27/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.1
Total Chlordane =									0.14
HB-HB-06S	8/2/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-HB-06S	8/2/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND
HB-HB-16D	1/7/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0059
HB-HB-16D	1/7/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0059
Total Chlordane =									ND
HB-HB-16D	1/7/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0073
HB-HB-16D	1/7/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.049
Total Chlordane =									0.049
HB-HBW-01	8/4/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-HBW-01	8/4/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND

TABLE 2.2d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HBW-02	8/4/2000	0	0.17	57-74-9	CHLORDANE	N	UJ	mg/kg	0.07
HB-HBW-02	8/4/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.07
Total Chlordane =									ND
HB-HBW-03	8/7/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-HBW-03	8/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-HBW-04	8/7/2000	0	0.17	57-74-9	CHLORDANE	N	UJ	mg/kg	0.08
HB-HBW-04	8/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.08
Total Chlordane =									ND
HB-HBW-05	8/8/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-HBW-05	8/8/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-HBW-06	8/8/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.02
HB-HBW-06	8/8/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.02
Total Chlordane =									ND
HB-RISB-01	12/13/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0071
HB-RISB-01	12/13/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0071
Total Chlordane =									ND
HB-RISB-01	12/13/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0079
HB-RISB-01	12/13/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0079
Total Chlordane =									ND
HB-RISB-02	12/13/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0065
HB-RISB-02	12/13/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0065
Total Chlordane =									ND
HB-RISB-02	12/13/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.007
HB-RISB-02	12/13/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.007
Total Chlordane =									ND
HB-SB-65	11/3/2006	0	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.028
HB-SB-65	11/3/2006	0	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-SEEP-2	9/9/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.037
HB-SEEP-2	9/9/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.037
Total Chlordane =									ND
HB-SEEP-2	9/9/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.04
HB-SEEP-2	9/9/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.04
Total Chlordane =									ND
HB-SS-02	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0041
HB-SS-02	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0041
Total Chlordane =									ND
HB-SS-02	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0058
HB-SS-02	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0058
Total Chlordane =									ND

TABLE 2.2d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-SS-04	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.006
HB-SS-04	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.006
Total Chlordane =									ND
HB-SS-04	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0059
HB-SS-04	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0059
Total Chlordane =									ND
HB-SS-05	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.082
HB-SS-05	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.082
Total Chlordane =									ND
HB-SS-05	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.08
HB-SS-05	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.08
Total Chlordane =									ND
HB-SS-06	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.09
HB-SS-06	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.09
Total Chlordane =									ND
HB-SS-06	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.077
HB-SS-06	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.077
Total Chlordane =									ND
HB-SS-07	12/5/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0061
HB-SS-07	12/5/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0061
Total Chlordane =									ND
HB-SS-08	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.036
HB-SS-08	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.036
Total Chlordane =									ND
HB-SS-08	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.056
HB-SS-08	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.056
Total Chlordane =									ND
HB-SS-09	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.083
HB-SS-09	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.083
Total Chlordane =									ND
HB-SS-09	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.063
HB-SS-09	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.063
Total Chlordane =									ND
HB-SS-10	12/4/2002	0	0.5	57-74-9	CHLORDANE	Y	J	mg/kg	0.0063
HB-SS-10	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0059
Total Chlordane =									0.0122
HB-SS-10	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-SS-10	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-SS-11	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-SS-11	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND

TABLE 2.2d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-SS-11	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-SS-11	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-TP-09	7/5/2000	2	2	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-TP-09	7/5/2000	2	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.002
Total Chlordane =									0.002

TABLE 2.2e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-GP-01	7/14/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-02	7/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-03	7/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-04	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.003	0.0015
HB-GP-05	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.005	0.0025
HB-GP-06	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.006	0.003
HB-GP-07	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.006	0.003
HB-GP-08	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.003	0.0015
HB-GP-09	7/12/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-10	7/12/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-GP-11	7/13/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-GP-12	7/13/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-GP-13	7/13/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-14	7/14/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-GP-15	7/14/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-16	7/18/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-17	7/18/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-GP-18	7/17/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-19	7/17/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-20	7/17/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-02I	7/19/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-HB-03S	7/26/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-04S	7/27/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.003	0.0015
HB-HB-05I	7/27/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-06S	8/2/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.004	0.004
HB-HB-16D	1/7/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.015	
HB-HB-16D	1/7/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.015	
HB-HB-16D	1/7/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.015
HB-HB-16D	1/7/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.017	
HB-HB-16D	1/7/2003	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.017	
HB-HB-16D	1/7/2003	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.017
HB-HBW-01	8/4/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.003	0.003
HB-HBW-02	8/4/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.013	0.013
HB-HBW-03	8/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HBW-04	8/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.002	0.002
HB-HBW-05	8/8/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HBW-06	8/8/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.001	0.001
HB-RISB-01	12/13/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.32	
HB-RISB-01	12/13/2002	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.21	
HB-RISB-01	12/13/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.53
HB-RISB-01	12/13/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.42	
HB-RISB-01	12/13/2002	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.21	
HB-RISB-01	12/13/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.63
HB-RISB-02	12/13/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0096	
HB-RISB-02	12/13/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0096	
HB-RISB-02	12/13/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0096
HB-RISB-02	12/13/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.078	
HB-RISB-02	12/13/2002	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.033	
HB-RISB-02	12/13/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.111
HB-SB-65	11/3/2006	0	2	1330-20-7	XYLENES, TOTAL	Y		mg/kg	2.1	2.1
HB-SEEP-2	9/9/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	5.9	
HB-SEEP-2	9/9/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	2.1	
HB-SEEP-2	9/9/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		8

TABLE 2.2e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-SEEP-2	9/9/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	9.6	
HB-SEEP-2	9/9/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	2.8	
HB-SEEP-2	9/9/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		12.4
HB-SS-02	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.006	
HB-SS-02	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.006	
HB-SS-02	12/3/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.006
HB-SS-02	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0086	
HB-SS-02	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0086	
HB-SS-02	12/3/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0086
HB-SS-04	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.014	
HB-SS-04	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.014	
HB-SS-04	12/3/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.014
HB-SS-04	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.13	
HB-SS-04	12/3/2002	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.011	
HB-SS-04	12/3/2002	0.5	1	CALCULATED	TOTAL	Y		mg/kg		0.141
HB-SS-05	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.019	
HB-SS-05	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.019	
HB-SS-05	12/3/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.019
HB-SS-05	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.022	
HB-SS-05	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.022	
HB-SS-05	12/3/2002	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.022
HB-SS-06	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.009	
HB-SS-06	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.02	
HB-SS-06	12/3/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.009
HB-SS-06	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0091	
HB-SS-06	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.016	
HB-SS-06	12/3/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0091
HB-SS-07	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0089	
HB-SS-07	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0089	
HB-SS-07	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0089
HB-SS-08	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.012	
HB-SS-08	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.012	
HB-SS-08	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.012
HB-SS-08	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0083	
HB-SS-08	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0083	
HB-SS-08	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0083
HB-SS-09	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.012	
HB-SS-09	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.012	
HB-SS-09	12/4/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.012
HB-SS-09	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0046	
HB-SS-09	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0093	
HB-SS-09	12/4/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0046
HB-SS-10	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0082	
HB-SS-10	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0082	
HB-SS-10	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0082
HB-SS-10	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0078	
HB-SS-10	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0078	
HB-SS-10	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0078
HB-SS-11	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0074	
HB-SS-11	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0074	
HB-SS-11	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0074
HB-SS-11	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0053	
HB-SS-11	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0081	
HB-SS-11	12/4/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0053
HB-TP-09	7/5/2000	2	2	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.3a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
Lakeshore Area - Subsurface Soil	DIOXIN/FURAN (9)																		
	1746-01-6	2,3,7,8-TCDD Equivalent	0.000003	0.001	mg/kg	HB-SS-04	8/8		5.75E-04			4.26E-06	C	3.90E-06	ca	3.90E-06	Y	ASL	
	METALS																		
	7429-90-5	ALUMINUM	1870	24400	mg/kg	HB-HBW-03	77/77	-	2.44E+04			7.82E+03	N	7.61E+03	nc	7.61E+03	Y	ASL	
	7440-36-0	ANTIMONY	0.31 J	1.6 J	mg/kg	HB-TP-23, HB-SB-64	28/77	0.19-15.37	1.60E+00			3.13E+00	N	3.13E+00	nc	3.13E+00	N	BSL	
	7440-38-2	ARSENIC	2.5 J	33	mg/kg	HB-SB-63	77/77	-	3.30E+01		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX	
	7440-39-3	BARIUM	12.2 J	4880 J	mg/kg	HB-SEEP-2	77/77	-	4.88E+03		4.00E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	Y	ASL	
	7440-41-7	BERYLLIUM	0.26 J	1.4	mg/kg	HB-HBW-01	52/77	0.6-1.28	1.40E+00		7.20E+01	1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL	
	7440-43-9	CADMIUM	0.055 J	110 J	mg/kg	HB-SS-06	65/77	0.13-1.17	1.10E+02		4.30E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	Y	ASL	
	7440-70-2	CALCIUM	59100	383000	mg/kg	HB-TP-23	77/77	-	3.83E+05			NV	NV	NV	NV	N	NUT		
	7440-47-3	CHROMIUM ⁶	6.7	391 J	mg/kg	HB-SS-06	77/77	-	3.91E+02		1.10E+02	2.35E+01	N	3.01E+01	ca	2.35E+01	Y	TOX	
	7440-48-4	COBALT	2.1 J	13.3 J	mg/kg	HB-GP-20	55/77	7.21-12.81	1.33E+01			NV	NV	9.03E+02	ca	9.03E+02	N	BSL	
	7440-50-8	COPPER	13.4	744 J	mg/kg	HB-SS-06	77/77	-	7.44E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	Y	ASL	
	57-12-5	CYANIDE	0.76	9.5 J	mg/kg	HB-SB-84	36/77	0.51-2.64	9.50E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL	
	7439-89-6	IRON	2690	24400 J	mg/kg	HB-HBW-04	77/77	-	2.44E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	6.8 J	1800 J	mg/kg	HB-GP-06	77/77	-	1.80E+03			NV	NV	4.00E+02	nc	4.00E+02	Y	ASL	
	7439-95-4	MAGNESIUM	3600	38300	mg/kg	HB-GP-20	77/77	-	3.83E+04			NV	NV	NV	NV	N	NUT		
	7439-96-5	MANGANESE	119 J	722	mg/kg	HB-GP-05	77/77	-	7.22E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL	
	7439-97-6	MERCURY ⁶	0.09	64.3	mg/kg	HB-SS-08	76/77	0.059-0.059	6.43E+01			2.35E+00	N	2.35E+00	nc	2.35E+00	Y	ASL	
	22967-92-6	METHYL MERCURY	0.864	96.1	mg/kg	HB-SS-08	12/12	-	9.61E+01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL	
	7440-02-0	NICKEL	9.8	98.6 J	mg/kg	HB-TP-18	77/77	-	9.86E+01		3.10E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL	
	7440-09-7	POTASSIUM	260 J	8170 J	mg/kg	HB-HBW-03	69/77	8.2-448	8.17E+03			NV	NV	NV	NV	N	NUT		
	7782-49-2	SELENIUM	0.35 J	3.4 J	mg/kg	HB-TP-18	53/77	0.49-5.95	3.40E+00		1.80E+02	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-22-4	SILVER	0.2 J	102 J	mg/kg	HB-TP-18	46/77	0.076-2.33	1.02E+02		1.80E+02	3.91E+01	N	3.91E+01	nc	3.91E+01	Y	ASL	
	7440-23-5	SODIUM	326 J	6090	mg/kg	HB-TP-23	76/77	568-568	6.09E+03			NV	NV	NV	NV	N	NUT		
	7440-28-0	THALLIUM	0.57 J	2.3	mg/kg	HB-HBW-05	17/77	0.5-3.8	2.30E+00			5.48E-01	N	5.16E-01	nc	5.16E-01	Y	ASL	
	7440-62-2	VANADIUM	5.8 J	49.1	mg/kg	HB-HBW-03	73/77	8.09-11.66	4.91E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	14.8	2310 J	mg/kg	HB-TP-18	77/77	-	2.31E+03		1.00E+04	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL	
	PCBs																		
		LESS CHLORINATED PCBs ^c	0.03	0.4	mg/kg	HB-GP-08	8/77	0.02-4	4.00E-01			5.48E-01	N	3.93E-01	nc	3.93E-01	Y	ASL	
		HIGHLY CHLORINATED PCBs ^d	0.02	6	mg/kg	HB-TP-18, HB-GP-06	61/77	0.02-2	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL	
		TOTAL PCBs ^e	0.02	6	mg/kg	HB-TP-18, HB-GP-06	61/77	0.02-2	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL	
	PESTICIDES																		
	72-54-8	4,4'-DDD	0.1 J	0.3 J	mg/kg	HB-TP-18	2/77	0.003-0.5	3.00E-01			1.30E+01	2.66E+00	C	2.44E+00	ca	2.44E+00	N	BSL
	50-29-3	4,4'-DDT	0.015 J	0.03 J	mg/kg	HB-SB-63	3/77	0.003-0.8	3.00E-02			7.90E+00	1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL
	57-74-9	TOTAL CHLORDANE ^f	0.002 J	0.4 J	mg/kg	HB-TP-18	21/77	0.002-0.4	4.00E-01				1.82E+00	C	1.62E+00	ca	1.60E+00	N	BSL
	60-57-1	DIELDRIN	0.017 J	0.2 J	mg/kg	HB-GP-07	3/77	0.003-0.8	2.00E-01			2.00E-01	3.99E-02	C	3.04E-02	ca	3.04E-02	Y	ASL
	1031-07-8	ENDOSULFAN SULFATE ^g	0.2	0.2	mg/kg	HB-HB-05i	1/77	0.003-0.8	2.00E-01		2.40E+01	4.69E+01	N	3.67E+01	nc	3.67E+01	N	BSL	
	7421-93-4	ENDRIN ALDEHYDE ^h	0.012 J	0.043 J	mg/kg	HB-HB-16D	2/77	0.003-0.8	4.30E-02			2.35E+00	N	1.83E+00	nc	1.83E+00	N	BSL	
	53494-70-5	ENDRIN KETONE ⁱ	0.39	0.56	mg/kg	HB-SS-11	2/77	0.003-0.8	5.60E-01			2.35E+00	N	1.83E+00	nc	1.83E+00	N	BSL	

TABLE 2.3a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	SVOCs																	
	120-83-2	2,4-DICHLOROPHENOL	0.29 J	0.29 J	mg/kg	HB-TP-01B	1/74	0.34-920	2.90E-01			2.35E+01	N	1.83E+01	nc	1.83E+01	N	BSL
	105-67-9	2,4-DIMETHYLPHENOL	0.076 J	7.3 J	mg/kg	HB-SEEP-2	5/76	0.34-920	7.30E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL
	51-28-5	2,4-DINITROPHENOL	0.067 J	0.067 J	mg/kg	HB-GP-05	1/74	1.7-4600	6.70E-02			1.56E+01	N	1.22E+01	nc	1.22E+01	N	BSL
	91-57-6	2-METHYLNAPHTHALENE	0.06 J	3800 J	mg/kg	HB-TP-22	49/76	0.34-180	3.80E+03			3.13E+01	N	NV		3.13E+01	Y	ASL
	95-48-7	2-METHYLPHENOL	2 J	9.2 J	mg/kg	HB-SEEP-2	4/76	0.34-920	9.20E+00		1.00E+02	3.91E+02	N	3.06E+02	nc	3.06E+02	N	BSL
	34METPH	3&4-METHYLPHENOL ¹	0.091 J	72 J	mg/kg	HB-TP-18	9/70	0.34-920	7.20E+01			3.91E+01	N	3.06E+01	nc	3.06E+01	Y	ASL
	106-47-8	4-CHLOROANILINE	0.12 J	3.5 J	mg/kg	HB-TP-18	19/76	0.34-920	3.50E+00			3.13E+01	N	2.44E+01	nc	2.44E+01	N	BSL
	83-32-9	ACENAPHTHENE	0.064 J	940 J	mg/kg	HB-TP-22	32/76	0.34-180	9.40E+02		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02	Y	ASL
	208-96-8	ACENAPHTHYLENE	0.06 J	850 J	mg/kg	HB-TP-22	54/76	0.34-180	8.50E+02		1.00E+02	NV	NV	NV		NV	Y	NTX
	120-12-7	ANTHRACENE	0.08 J	810 J	mg/kg	HB-TP-22	57/76	0.34-180	8.10E+02		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	0.063 J	350 J	mg/kg	HB-TP-22	65/76	0.53-180	3.50E+02		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	50-32-8	BENZO(A)PYRENE	0.06 J	150 J	mg/kg	HB-TP-22	64/77	0.57-180	1.50E+02		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.071 J	210 J	mg/kg	HB-TP-22	65/77	0.57-180	2.10E+02		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.042 J	4.7	mg/kg	HB-SS-10	56/76	0.53-920	4.70E+00		1.00E+02	NV	NV	NV		NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.061 J	13 J	mg/kg	HB-SB-63	61/76	0.53-920	1.30E+01		3.90E+00	2.20E+00	C	6.21E+00	ca	2.20E+00	Y	ASL
	65-85-0	BENZOIC ACID	0.074 J	7.4 J	mg/kg	HB-TP-23	24/42	1.7-4600	7.40E+00			3.13E+04	N	1.00E+04	nc	1.00E+04	N	BSL
	100-51-6	BENZYL ALCOHOL	0.066 J	0.13 J	mg/kg	HB-GP-14	2/70	0.34-920	1.30E-01			3.91E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	111-44-4	BIS(2-CHLOROETHYL)ETHER	0.05 J	0.05 J	mg/kg	HB-HBW-06	1/76	0.34-920	5.00E-02			5.81E-01	C	2.18E-01	ca	2.18E-01	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.054 J	9.5 J	mg/kg	HB-TP-18	12/76	0.069-920	9.50E+00			4.56E+01	C	3.47E+01	ca	3.47E+01	N	BSL
	85-68-7	BUTYLBENZYL PHTHALATE	0.11 J	0.2 J	mg/kg	HB-HBW-03	3/76	0.34-920	2.00E-01			1.56E+03	N	1.22E+03	nc	1.22E+03	N	BSL
	86-74-8	CARBAZOLE	0.065 J	220 J	mg/kg	HB-TP-22	39/76	0.34-180	2.20E+02			3.19E+01	C	2.43E+01	ca	2.43E+01	Y	ASL
	218-01-9	CHRYSENE	0.07 J	290 J	mg/kg	HB-TP-22	66/77	0.53-180	2.90E+02		3.90E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.072 J	1.4 J	mg/kg	HB-SS-10	36/76	0.34-920	1.40E+00		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.051 J	1400 J	mg/kg	HB-TP-22	38/76	0.34-180	1.40E+03		5.90E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.047 J	0.22 J	mg/kg	HB-GP-07	9/76	0.34-920	2.20E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	117-84-0	DI-N-OCTYL PHTHALATE	0.3 J	0.3 J	mg/kg	HB-HBW-03	1/76	0.34-920	3.00E-01			NV	NV	2.44E+02	nc	2.44E+02	N	BSL
	206-44-0	FLUORANTHENE	0.076 J	1400 J	mg/kg	HB-TP-22	70/77	0.64-180	1.40E+03		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	Y	ASL
	86-73-7	FLUORENE	0.05 J	1800 J	mg/kg	HB-TP-22	31/76	0.34-180	1.80E+03		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	Y	ASL
	118-74-1	HEXACHLOROBENZENE	0.083 J	9 J	mg/kg	HB-TP-23	14/76	0.4-920	9.00E+00		1.20E+00	3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.047 J	4.1	mg/kg	HB-SS-10	57/76	0.53-920	4.10E+00		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.007 J	12000 J	mg/kg	HB-TP-22	72/116	0.004-160	1.20E+04		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	87-86-5	PENTACHLOROPHENOL	0.13 J	0.13 J	mg/kg	HB-GP-05	1/73	1.7-4600	1.30E-01		6.70E+00	5.32E+00	C	2.98E+00	ca	2.98E+00	N	BSL
	85-01-8	PHENANTHRENE	0.065 J	3500 J	mg/kg	HB-TP-22	64/77	0.53-180	3.50E+03		1.00E+02	NV	NV	NV		NV	Y	NTX
	108-95-2	PHENOL	0.055 J	26 J	mg/kg	HB-SEEP-2	16/76	0.34-920	2.60E+01		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.058 J	950 J	mg/kg	HB-TP-22	70/77	0.64-180	9.50E+02		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	Y	ASL

TABLE 2.3a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	VOCs																	
	71-55-6	1,1,1-TRICHLOROETHANE	0.004 J	0.004 J	mg/kg	HB-TP-19	1/76	0.003-38	4.00E-03		1.00E+02	1.56E+04	N	1.20E+02	nc	1.20E+02	N	BSL
	92-52-4	1,1'-BIPHENYL	12 J	12 J	mg/kg	HB-SB-63	1/6	0.55-57	1.20E+01			3.91E+02	N	3.01E+02	nc	3.01E+02	N	BSL
	87-61-6	1,2,3-TRICHLOROBENZENE	0.048	18 J	mg/kg	HB-TP-01	3/40	0.005-35	1.80E+01			NV	NV	NV	Y	NTX		
	120-82-1	1,2,4-TRICHLOROBENZENE	0.002 J	88 J	mg/kg	HB-TP-01D	40/117	0.005-920	8.80E+01			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.002 J	390 J	mg/kg	HB-TP-22	10/40	0.003-0.006	3.90E+02		5.20E+01	NV	5.16E+00	nc	5.16E+00	Y	ASL	
	95-50-1	1,2-DICHLOROBENZENE	0.001 J	360	mg/kg	HB-TP-01	58/118	0.003-920	3.60E+02		1.00E+02	7.04E+02	N	6.00E+01	nc	6.00E+01	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.001 J	160	mg/kg	HB-TP-22	10/40	0.003-0.006	1.60E+02		5.20E+01	NV	2.13E+00	nc	2.13E+00	Y	ASL	
	541-73-1	1,3-DICHLOROBENZENE	0.001 J	27 J	mg/kg	HB-TP-01D	20/117	0.003-920	2.70E+01		4.90E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	Y	ASL
	106-46-7	1,4-DICHLOROBENZENE	0.002 J	460 J	mg/kg	HB-TP-46A	66/118	0.000017-920	4.60E+02		1.30E+01	2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL
	78-93-3	2-BUTANONE	0.001 J	0.061 J	mg/kg	HB-TP-01B	8/76	0.00005-150	6.10E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	95-49-8	2-CHLOROTOLUENE	0.029	0.037	mg/kg	HB-TP-01B	2/40	0.003-38	3.70E-02			1.56E+02	N	1.58E+01	nc	1.58E+01	N	BSL
	106-43-4	4-CHLOROTOLUENE	0.022	0.024	mg/kg	HB-TP-01B	2/40	0.003-38	2.40E-02			5.48E+02	N	NV		5.48E+02	N	BSL
	67-64-1	ACETONE	0.007 J	0.17 J	mg/kg	HB-SS-09	8/75	0.01-150	1.70E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	98-86-2	ACETOPHENONE	0.22 J	0.22 J	mg/kg	HB-SB-85	1/6	28-57	2.20E-01			7.82E+02	N	NV		7.82E+02	N	BSL
	71-43-2	BENZENE	0.0000055 J	71	mg/kg	HB-TP-22	25/76	0.003-5.8	7.10E+01		4.80E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	108-86-1	BROMOBENZENE	0.004 J	0.004 J	mg/kg	HB-TP-01B	1/40	0.003-38	4.00E-03			NV	2.78E+00	nc	2.78E+00	N	BSL	
	75-25-2	BROMOFORM	4.2 J	4.2 J	mg/kg	HB-TP-22	1/76	0.003-38	4.20E+00			8.09E+01	C	6.16E+01	ca	6.16E+01	N	BSL
	104-51-8	BUTYLBENZENE	0.008 J	6.8 J	mg/kg	HB-TP-18	3/40	0.003-38	6.80E+00		1.00E+02	NV	2.40E+01	nc	2.40E+01	N	BSL	
	75-15-0	CARBON DISULFIDE	0.0026 J	0.11 J	mg/kg	HB-SB-65	2/30	0.012-2.9	1.10E-01			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL
	108-90-7	CHLOROBENZENE	0.000072 J	120	mg/kg	HB-TP-01	32/77	0.003-5.8	1.20E+02		1.00E+02	1.56E+02	N	1.51E+01	nc	1.51E+01	Y	ASL
	110-82-7	CYCLOHEXANE	0.25 J	0.25 J	mg/kg	HB-SB-63	1/5	0.42-0.95	2.50E-01			NV	1.40E+01	nc	1.40E+01	N	BSL	
	100-41-4	ETHYLBENZENE	0.005 J	35	mg/kg	HB-TP-22	16/76	0.003-38	3.50E+01		4.10E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.015 J	8.1 J	mg/kg	HB-TP-22	10/46	0.003-38	8.10E+00			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
	108-87-2	METHYLCYCLOHEXANE	0.2 J	1.2	mg/kg	HB-SB-63	4/6	0.43-0.95	1.20E+00			NV	2.59E+02	nc	2.59E+02	N	BSL	
	75-09-2	METHYLENE CHLORIDE	0.002 J	0.16	mg/kg	HB-HBW-01	4/76	0.001-77	1.60E-01		1.00E+02	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL
	103-65-1	N-PROPYLBENZENE	0.008 J	7.9 J	mg/kg	HB-TP-22	5/40	0.003-38	7.90E+00		1.00E+02	NV	2.40E+01	nc	2.40E+01	N	BSL	
	99-87-6	P-ISOPROPYLTOLUENE	0.002 J	8.7 J	mg/kg	HB-TP-18	6/41	0.003-38	8.70E+00			NV	NV	NV	Y	NTX		
	135-98-8	SEC-BUTYLBENZENE	0.002 J	2.8 J	mg/kg	HB-TP-01	5/40	0.003-17	2.80E+00		1.00E+02	NV	2.20E+01	nc	2.20E+01	N	BSL	
	100-42-5	STYRENE	0.004 J	98	mg/kg	HB-TP-22	7/76	0.003-38	9.80E+01			1.56E+03	N	1.70E+02	nc	1.70E+02	N	BSL
	98-06-6	TERT-BUTYLBENZENE	0.022	0.033	mg/kg	HB-TP-23	2/40	0.003-38	3.30E-02		1.00E+02	NV	3.90E+01	nc	3.90E+01	N	BSL	
	127-18-4	TETRACHLOROETHENE	0.001 J	0.004 J	mg/kg	HB-TP-05	3/76	0.000025-38	4.00E-03		1.90E+01	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL
	108-88-3	TOLUENE	0.001 J	240	mg/kg	HB-TP-22	26/77	0.003-38	2.40E+02		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	Y	ASL
	79-01-6	TRICHLOROETHENE	0.002 J	0.002 J	mg/kg	HB-HBW-04	1/76	0.000025-38	2.00E-03		2.10E+01	1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL
	1330-20-7	XYLENES, TOTAL	0.001 J	490	mg/kg	HB-TP-22	29/77	0.0015-0.022	4.90E+02		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
 - (9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.3b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for mercury compounds utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
f = Where criteria are not available, RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
g = RBC and PRG values for Endosulfan (CAS# 115297) utilized.
h = RBC and PRG values for Endrin (CAS# 72208) utilized.
i = RBC and PRG values for 4-methylphenol (CAS# 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals; USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.3b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-RISB-02	12/13/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	18.458	18.458	ng/kg	J	0.01	0.185
HB-RISB-02	12/13/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	13.331	13.331	ng/kg	J	0.01	0.133
HB-RISB-02	12/13/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.801	2.801	ng/kg	J	0.01	0.028
HB-RISB-02	12/13/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.596	0.596	ng/kg	EMPC	0.1	0.060
HB-RISB-02	12/13/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	9.566	9.566	ng/kg	J	0.1	0.957
HB-RISB-02	12/13/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.966	1.966	ng/kg	J	0.1	0.197
HB-RISB-02	12/13/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.385	2.385	ng/kg	J	0.1	0.239
HB-RISB-02	12/13/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	0.875	0.875	ng/kg	EMPC	0.1	0.088
HB-RISB-02	12/13/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.958	1.958	ng/kg	EMPC	0.1	0.196
HB-RISB-02	12/13/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.68	0.68	ng/kg	J	1	0.680
HB-RISB-02	12/13/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	5.239	5.239	ng/kg	J	0.03	0.157
HB-RISB-02	12/13/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-RISB-02	12/13/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	12.75	12.75	ng/kg	J	0.1	1.275
HB-RISB-02	12/13/2002	0	0.5	3268-87-9	OCDD	Y	96.811	96.811	ng/kg	J	0.0003	0.029
HB-RISB-02	12/13/2002	0	0.5	39001-02-0	OCDF	Y	46.831	46.831	ng/kg	J	0.0003	0.014
Sample Location TEQ =											4.7	
HB-RISB-02	12/13/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	9.541	9.541	ng/kg		0.01	0.095
HB-RISB-02	12/13/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	7.187	7.187	ng/kg		0.01	0.072
HB-RISB-02	12/13/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.445	1.445	ng/kg	J	0.01	0.014
HB-RISB-02	12/13/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-02	12/13/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	6.501	6.501	ng/kg		0.1	0.650
HB-RISB-02	12/13/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.852	0.852	ng/kg	J	0.1	0.085
HB-RISB-02	12/13/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.59	1.59	ng/kg	J	0.1	0.159
HB-RISB-02	12/13/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	416	208	ng/kg	U	0.1	20.800
HB-RISB-02	12/13/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-02	12/13/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-RISB-02	12/13/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	4.574	4.574	ng/kg		0.03	0.137
HB-RISB-02	12/13/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-RISB-02	12/13/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	12.846	12.846	ng/kg		0.1	1.285
HB-RISB-02	12/13/2002	0.5	1	3268-87-9	OCDD	Y	66.844	66.844	ng/kg	J	0.0003	0.020
HB-RISB-02	12/13/2002	0.5	1	39001-02-0	OCDF	Y	20.584	20.584	ng/kg	J	0.0003	0.006
Sample Location TEQ =											25.3	

TABLE 2.3b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SEEP-2	9/9/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.504	8.504	ng/kg	J	0.01	0.085
HB-SEEP-2	9/9/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	UJ	0.01	0.013
HB-SEEP-2	9/9/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.025	2.025	ng/kg	J	0.1	0.203
HB-SEEP-2	9/9/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.688	3.688	ng/kg	J	0.1	0.369
HB-SEEP-2	9/9/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.628	0.628	ng/kg	J	0.1	0.063
HB-SEEP-2	9/9/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.442	1.442	ng/kg	J	0.1	0.144
HB-SEEP-2	9/9/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-SEEP-2	9/9/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.087	1.087	ng/kg	J	0.03	0.033
HB-SEEP-2	9/9/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-SEEP-2	9/9/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.859	2.859	ng/kg	EMPC	0.1	0.286
HB-SEEP-2	9/9/2003	0	0.5	3268-87-9	OCDD	Y	260.17	260.17	ng/kg	J	0.0003	0.078
HB-SEEP-2	9/9/2003	0	0.5	39001-02-0	OCDF	Y	19.606	19.606	ng/kg	J	0.0003	0.006
Sample Location TEQ =												3.3
HB-SEEP-2	9/9/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	3.127	3.127	ng/kg	J	0.01	0.031
HB-SEEP-2	9/9/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	UJ	0.01	0.013
HB-SEEP-2	9/9/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.531	1.531	ng/kg	J	0.1	0.153
HB-SEEP-2	9/9/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-SEEP-2	9/9/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-SEEP-2	9/9/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	UJ	0.03	0.038
HB-SEEP-2	9/9/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-SEEP-2	9/9/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	7.27	7.27	ng/kg	J	0.1	0.727
HB-SEEP-2	9/9/2003	0.5	1	3268-87-9	OCDD	Y	66.684	66.684	ng/kg	J	0.0003	0.020
HB-SEEP-2	9/9/2003	0.5	1	39001-02-0	OCDF	Y	4.583	4.583	ng/kg	EMPC	0.0003	0.001
Sample Location TEQ =												3.4

TABLE 2.3b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SS-04	12/3/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	1186.223	1186.223	ng/kg		0.01	11.862
HB-SS-04	12/3/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	274.059	274.059	ng/kg		0.01	2.741
HB-SS-04	12/3/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	64.442	64.442	ng/kg		0.01	0.644
HB-SS-04	12/3/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	210.432	210.432	ng/kg		0.1	21.043
HB-SS-04	12/3/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	437.121	437.121	ng/kg		0.1	43.712
HB-SS-04	12/3/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	446.742	446.742	ng/kg		0.1	44.674
HB-SS-04	12/3/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	125.718	125.718	ng/kg	J	0.1	12.572
HB-SS-04	12/3/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	282.125	282.125	ng/kg		0.1	28.213
HB-SS-04	12/3/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	69.159	69.159	ng/kg	J	0.1	6.916
HB-SS-04	12/3/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	275.231	275.231	ng/kg		1	275.231
HB-SS-04	12/3/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	352.347	352.347	ng/kg		0.03	10.570
HB-SS-04	12/3/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	31.176	31.176	ng/kg		1	31.176
HB-SS-04	12/3/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	855.5	855.5	ng/kg	J	0.1	85.550
HB-SS-04	12/3/2002	0	0.5	3268-87-9	OCDD	Y	1354.982	1354.982	ng/kg	J	0.0003	0.406
HB-SS-04	12/3/2002	0	0.5	39001-02-0	OCDF	Y	496.581	496.581	ng/kg	J	0.0003	0.149
Sample Location TEQ =												575.5
HB-SS-04	12/3/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	1083.071	1083.071	ng/kg		0.01	10.831
HB-SS-04	12/3/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	331.513	331.513	ng/kg		0.01	3.315
HB-SS-04	12/3/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	81.67	81.67	ng/kg		0.01	0.817
HB-SS-04	12/3/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	167.275	167.275	ng/kg		0.1	16.728
HB-SS-04	12/3/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	598.716	598.716	ng/kg		0.1	59.872
HB-SS-04	12/3/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	390.763	390.763	ng/kg		0.1	39.076
HB-SS-04	12/3/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	153.225	153.225	ng/kg		0.1	15.323
HB-SS-04	12/3/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	269.866	269.866	ng/kg		0.1	26.987
HB-SS-04	12/3/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	107.172	107.172	ng/kg		0.1	10.717
HB-SS-04	12/3/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	237.276	237.276	ng/kg		1	237.276
HB-SS-04	12/3/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	374.758	374.758	ng/kg		0.03	11.243
HB-SS-04	12/3/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	25.347	25.347	ng/kg		1	25.347
HB-SS-04	12/3/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	793	793	ng/kg	J	0.1	79.300
HB-SS-04	12/3/2002	0.5	1	3268-87-9	OCDD	Y	1447.067	1447.067	ng/kg	J	0.0003	0.434
HB-SS-04	12/3/2002	0.5	1	39001-02-0	OCDF	Y	485.628	485.628	ng/kg	J	0.0003	0.146
Sample Location TEQ =												537.4

TABLE 2.3b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)	
HB-SS-11	12/4/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	528.876	528.876	ng/kg		0.01	5.289	
HB-SS-11	12/4/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	201.137	201.137	ng/kg		0.01	2.011	
HB-SS-11	12/4/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	57.162	57.162	ng/kg		0.01	0.572	
HB-SS-11	12/4/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	90.395	90.395	ng/kg		0.1	9.040	
HB-SS-11	12/4/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	348.307	348.307	ng/kg		0.1	34.831	
HB-SS-11	12/4/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	218.606	218.606	ng/kg		0.1	21.861	
HB-SS-11	12/4/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	99.834	99.834	ng/kg		0.1	9.983	
HB-SS-11	12/4/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	129.133	129.133	ng/kg		0.1	12.913	
HB-SS-11	12/4/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	65.808	65.808	ng/kg		0.1	6.581	
HB-SS-11	12/4/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	129.77	129.77	ng/kg		1	129.770	
HB-SS-11	12/4/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	214.534	214.534	ng/kg		0.03	6.436	
HB-SS-11	12/4/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	16.073	16.073	ng/kg		1	16.073	
HB-SS-11	12/4/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	299.1	299.1	ng/kg		0.1	29.910	
HB-SS-11	12/4/2002	0	0.5	3268-87-9	OCDD	Y	647.321	647.321	ng/kg	J	0.0003	0.194	
Sample Location TEQ =												285.5	
HB-SS-11	12/4/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	62.134	62.134	ng/kg	EMPC	0.01	0.621	
HB-SS-11	12/4/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	63.752	63.752	ng/kg		0.01	0.638	
HB-SS-11	12/4/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	16.645	16.645	ng/kg		0.01	0.166	
HB-SS-11	12/4/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.298	6.298	ng/kg		0.1	0.630	
HB-SS-11	12/4/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	108.398	108.398	ng/kg		0.1	10.840	
HB-SS-11	12/4/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	19.388	19.388	ng/kg		0.1	1.939	
HB-SS-11	12/4/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	34.666	34.666	ng/kg		0.1	3.467	
HB-SS-11	12/4/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	11.339	11.339	ng/kg		0.1	1.134	
HB-SS-11	12/4/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	17.594	17.594	ng/kg	EMPC	0.1	1.759	
HB-SS-11	12/4/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	12.855	12.855	ng/kg		1	12.855	
HB-SS-11	12/4/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	63.962	63.962	ng/kg		0.03	1.919	
HB-SS-11	12/4/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.975	1.975	ng/kg		1	1.975	
HB-SS-11	12/4/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	80.2	80.2	ng/kg		0.1	8.020	
HB-SS-11	12/4/2002	0.5	1	3268-87-9	OCDD	Y	133.435	133.435	ng/kg		J	0.0003	0.040
HB-SS-11	12/4/2002	0.5	1	39001-02-0	OCDF	Y	75.28	75.28	ng/kg		J	0.0003	0.023
Sample Location TEQ =												46.0	

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable

EMPC = Estimated Maximum Possible Concentration

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223-241.

TABLE 2.3c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0 - 10 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-GP-01	0	0.17	7/14/2000	0.2	mg/kg
Highly Chlorinated PCBs	HB-GP-02	0	0.17	7/7/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-03	0	0.17	7/7/2000	3	mg/kg
Highly Chlorinated PCBs	HB-GP-04	0	0.17	7/10/2000	0.5	mg/kg
Highly Chlorinated PCBs	HB-GP-05	0	0.17	7/10/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-06	0	0.17	7/10/2000	6	mg/kg
Highly Chlorinated PCBs	HB-GP-07	0	0.17	7/10/2000	4	mg/kg
Highly Chlorinated PCBs	HB-GP-08	0	0.17	7/10/2000	0.4	mg/kg
Highly Chlorinated PCBs	HB-GP-09	0	0.17	7/12/2000	1	mg/kg
Highly Chlorinated PCBs	HB-GP-10	0	0.17	7/12/2000	1	mg/kg
Highly Chlorinated PCBs	HB-GP-11	0	0.17	7/13/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-12	0	0.17	7/13/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-13	0	0.17	7/13/2000	0.3	mg/kg
Highly Chlorinated PCBs	HB-GP-14	0	0.17	7/14/2000	0.2	mg/kg
Highly Chlorinated PCBs	HB-GP-15	0	0.17	7/14/2000	1	mg/kg
Highly Chlorinated PCBs	HB-GP-16	0	0.17	7/18/2000	0.1	mg/kg
Highly Chlorinated PCBs	HB-GP-17	0	0.17	7/18/2000	2	mg/kg
Highly Chlorinated PCBs	HB-GP-18	0	0.17	7/17/2000	0.9	mg/kg
Highly Chlorinated PCBs	HB-GP-19	0	0.17	7/17/2000	0.03	mg/kg
Highly Chlorinated PCBs	HB-GP-20	0	0.17	7/17/2000	0.02	mg/kg
Highly Chlorinated PCBs	HB-HB-02I	0	0.17	7/19/2000	0.9	mg/kg
Highly Chlorinated PCBs	HB-HB-03S	0	0.17	7/26/2000	0.02	mg/kg
Highly Chlorinated PCBs	HB-HB-04S	0	0.17	7/27/2000	0.1	mg/kg
Highly Chlorinated PCBs	HB-HB-05I	0	0.17	7/27/2000	3	mg/kg
Highly Chlorinated PCBs	HB-HB-06S	0	0.17	8/2/2000	0.5	mg/kg
Highly Chlorinated PCBs	HB-HB-16D	0	0.5	1/7/2003	0.29	mg/kg
Highly Chlorinated PCBs	HB-HB-16D	0.5	1	1/7/2003	1.1	mg/kg
Highly Chlorinated PCBs	HB-HBW-01	0	0.17	8/4/2000	0.6	mg/kg
Highly Chlorinated PCBs	HB-HBW-02	0	0.17	8/4/2000	1	mg/kg
Highly Chlorinated PCBs	HB-HBW-03	0	0.17	8/7/2000	0.06	mg/kg
Highly Chlorinated PCBs	HB-HBW-04	0	0.17	8/7/2000	0.08	mg/kg
Highly Chlorinated PCBs	HB-HBW-05	0	0.17	8/8/2000	0.3	mg/kg
Highly Chlorinated PCBs	HB-HBW-06	0	0.17	8/8/2000	0.08	mg/kg
Highly Chlorinated PCBs	HB-RISB-01	0	0.5	12/13/2002	0.2	mg/kg
Highly Chlorinated PCBs	HB-RISB-02	0	0.5	12/13/2002	0.15	mg/kg
Highly Chlorinated PCBs	HB-SB-63	4	6	11/1/2006	1.497	mg/kg
Highly Chlorinated PCBs	HB-SB-65	0	2	11/3/2006	1.55	mg/kg
Highly Chlorinated PCBs	HB-SB-85	4	6	10/26/2006	0.1968	mg/kg
Highly Chlorinated PCBs	HB-SS-04	0	0.5	12/3/2002	0.49	mg/kg
Highly Chlorinated PCBs	HB-SS-04	0.5	1	12/3/2002	0.43	mg/kg
Highly Chlorinated PCBs	HB-SS-05	0	0.5	12/3/2002	3.7	mg/kg
Highly Chlorinated PCBs	HB-SS-05	0.5	1	12/3/2002	3	mg/kg
Highly Chlorinated PCBs	HB-SS-06	0	0.5	12/3/2002	3.6	mg/kg
Highly Chlorinated PCBs	HB-SS-06	0.5	1	12/3/2002	2.5	mg/kg
Highly Chlorinated PCBs	HB-SS-07	0	0.5	12/5/2002	0.75	mg/kg

TABLE 2.3c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0 - 10 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-SS-09	0.5	1	12/4/2002	2.7	mg/kg
Highly Chlorinated PCBs	HB-SS-11	0.5	1	12/4/2002	2.2	mg/kg
Highly Chlorinated PCBs	HB-TP-01	6	6	7/5/2000	0.07	mg/kg
Highly Chlorinated PCBs	HB-TP-01B	8	8	7/19/2000	0.1	mg/kg
Highly Chlorinated PCBs	HB-TP-01D	3	3	7/19/2000	0.9	mg/kg
Highly Chlorinated PCBs	HB-TP-05	3	3	7/5/2000	4	mg/kg
Highly Chlorinated PCBs	HB-TP-07	2.5	2.5	7/5/2000	0.06	mg/kg
Highly Chlorinated PCBs	HB-TP-09	2	2	7/5/2000	0.2	mg/kg
Highly Chlorinated PCBs	HB-TP-15	3	3	7/6/2000	3	mg/kg
Highly Chlorinated PCBs	HB-TP-18	4	4	7/6/2000	6	mg/kg
Highly Chlorinated PCBs	HB-TP-20	6.5	6.5	7/7/2000	0.02	mg/kg
Highly Chlorinated PCBs	HB-TP-20A	5	5	7/18/2000	0.04	mg/kg
Highly Chlorinated PCBs	HB-TP-21	3.5	3.5	7/18/2000	0.7	mg/kg
Highly Chlorinated PCBs	HB-TP-22	4	4	7/18/2000	0.09	mg/kg
Highly Chlorinated PCBs	HB-TP-23	4.5	4.5	7/18/2000	0.5	mg/kg
Highly Chlorinated PCBs	HB-TP-46A	3	4	11/16/2006	1.187	mg/kg
Less Chlorinated PCBs	HB-GP-08	0	0.17	7/10/2000	0.4	mg/kg
Less Chlorinated PCBs	HB-GP-09	0	0.17	7/12/2000	0.03	mg/kg
Less Chlorinated PCBs	HB-GP-11	0	0.17	7/13/2000	0.09	mg/kg
Less Chlorinated PCBs	HB-GP-12	0	0.17	7/13/2000	0.08	mg/kg
Less Chlorinated PCBs	HB-GP-13	0	0.17	7/13/2000	0.03	mg/kg
Less Chlorinated PCBs	HB-GP-14	0	0.17	7/14/2000	0.03	mg/kg
Less Chlorinated PCBs	HB-GP-15	0	0.17	7/14/2000	0.04	mg/kg
Less Chlorinated PCBs	HB-HB-02I	0	0.17	7/19/2000	0.1	mg/kg
Total PCBs	HB-GP-01	0	0.17	7/14/2000	0.2	mg/kg
Total PCBs	HB-GP-02	0	0.17	7/7/2000	2	mg/kg
Total PCBs	HB-GP-03	0	0.17	7/7/2000	3	mg/kg
Total PCBs	HB-GP-04	0	0.17	7/10/2000	0.5	mg/kg
Total PCBs	HB-GP-05	0	0.17	7/10/2000	2	mg/kg
Total PCBs	HB-GP-06	0	0.17	7/10/2000	6	mg/kg
Total PCBs	HB-GP-07	0	0.17	7/10/2000	4	mg/kg
Total PCBs	HB-GP-08	0	0.17	7/10/2000	0.8	mg/kg
Total PCBs	HB-GP-09	0	0.17	7/12/2000	1.03	mg/kg
Total PCBs	HB-GP-10	0	0.17	7/12/2000	1	mg/kg
Total PCBs	HB-GP-11	0	0.17	7/13/2000	2.09	mg/kg
Total PCBs	HB-GP-12	0	0.17	7/13/2000	2.08	mg/kg
Total PCBs	HB-GP-13	0	0.17	7/13/2000	0.33	mg/kg
Total PCBs	HB-GP-14	0	0.17	7/14/2000	0.23	mg/kg
Total PCBs	HB-GP-15	0	0.17	7/14/2000	1.04	mg/kg
Total PCBs	HB-GP-16	0	0.17	7/18/2000	0.1	mg/kg
Total PCBs	HB-GP-17	0	0.17	7/18/2000	2	mg/kg
Total PCBs	HB-GP-18	0	0.17	7/17/2000	0.9	mg/kg
Total PCBs	HB-GP-19	0	0.17	7/17/2000	0.03	mg/kg

TABLE 2.3c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0 - 10 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-GP-20	0	0.17	7/17/2000	0.02	mg/kg
Total PCBs	HB-HB-02I	0	0.17	7/19/2000	1	mg/kg
Total PCBs	HB-HB-03S	0	0.17	7/26/2000	0.02	mg/kg
Total PCBs	HB-HB-04S	0	0.17	7/27/2000	0.1	mg/kg
Total PCBs	HB-HB-05I	0	0.17	7/27/2000	3	mg/kg
Total PCBs	HB-HB-06S	0	0.17	8/2/2000	0.5	mg/kg
Total PCBs	HB-HB-16D	0	0.5	1/7/2003	0.29	mg/kg
Total PCBs	HB-HB-16D	0.5	1	1/7/2003	1.1	mg/kg
Total PCBs	HB-HBW-01	0	0.17	8/4/2000	0.6	mg/kg
Total PCBs	HB-HBW-02	0	0.17	8/4/2000	1	mg/kg
Total PCBs	HB-HBW-03	0	0.17	8/7/2000	0.06	mg/kg
Total PCBs	HB-HBW-04	0	0.17	8/7/2000	0.08	mg/kg
Total PCBs	HB-HBW-05	0	0.17	8/8/2000	0.3	mg/kg
Total PCBs	HB-HBW-06	0	0.17	8/8/2000	0.08	mg/kg
Total PCBs	HB-RISB-01	0	0.5	12/13/2002	0.2	mg/kg
Total PCBs	HB-RISB-02	0	0.5	12/13/2002	0.15	mg/kg
Total PCBs	HB-SB-63	4	6	11/1/2006	1.497	mg/kg
Total PCBs	HB-SB-65	0	2	11/3/2006	1.55	mg/kg
Total PCBs	HB-SB-85	4	6	10/26/2006	0.1968	mg/kg
Total PCBs	HB-SS-04	0	0.5	12/3/2002	0.49	mg/kg
Total PCBs	HB-SS-04	0.5	1	12/3/2002	0.43	mg/kg
Total PCBs	HB-SS-05	0	0.5	12/3/2002	3.7	mg/kg
Total PCBs	HB-SS-05	0.5	1	12/3/2002	3	mg/kg
Total PCBs	HB-SS-06	0	0.5	12/3/2002	3.6	mg/kg
Total PCBs	HB-SS-06	0.5	1	12/3/2002	2.5	mg/kg
Total PCBs	HB-SS-07	0	0.5	12/5/2002	0.75	mg/kg
Total PCBs	HB-SS-09	0.5	1	12/4/2002	2.7	mg/kg
Total PCBs	HB-SS-11	0.5	1	12/4/2002	2.2	mg/kg
Total PCBs	HB-TP-01	6	6	7/5/2000	0.07	mg/kg
Total PCBs	HB-TP-01B	8	8	7/19/2000	0.1	mg/kg
Total PCBs	HB-TP-01D	3	3	7/19/2000	0.9	mg/kg
Total PCBs	HB-TP-05	3	3	7/5/2000	4	mg/kg
Total PCBs	HB-TP-07	2.5	2.5	7/5/2000	0.06	mg/kg
Total PCBs	HB-TP-09	2	2	7/5/2000	0.2	mg/kg
Total PCBs	HB-TP-15	3	3	7/6/2000	3	mg/kg
Total PCBs	HB-TP-18	4	4	7/6/2000	6	mg/kg
Total PCBs	HB-TP-20	6.5	6.5	7/7/2000	0.02	mg/kg
Total PCBs	HB-TP-20A	5	5	7/18/2000	0.04	mg/kg
Total PCBs	HB-TP-21	3.5	3.5	7/18/2000	0.7	mg/kg
Total PCBs	HB-TP-22	4	4	7/18/2000	0.09	mg/kg
Total PCBs	HB-TP-23	4.5	4.5	7/18/2000	0.5	mg/kg
Total PCBs	HB-TP-46A	3	4	11/16/2006	1.187	mg/kg

Notes:

* Less Chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.3d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-GP-01	7/14/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-01	7/14/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-02	7/7/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.1
HB-GP-02	7/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.1
Total Chlordane =									0.1
HB-GP-03	7/7/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.1
HB-GP-03	7/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.1
Total Chlordane =									0.1
HB-GP-04	7/10/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.02
HB-GP-04	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.01
Total Chlordane =									0.01
HB-GP-05	7/10/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.01
HB-GP-05	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.05
Total Chlordane =									0.06
HB-GP-06	7/10/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.08
HB-GP-06	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.3
Total Chlordane =									0.38
HB-GP-07	7/10/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-GP-07	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.2
Total Chlordane =									0.25
HB-GP-08	7/10/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.04
HB-GP-08	7/10/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.04
Total Chlordane =									ND
HB-GP-09	7/12/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-GP-09	7/12/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									0.05
HB-GP-10	7/12/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-GP-10	7/12/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND
HB-GP-11	7/13/2000	0	0.17	57-74-9	CHLORDANE	Y		mg/kg	0.07
HB-GP-11	7/13/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.07
Total Chlordane =									0.07
HB-GP-12	7/13/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-GP-12	7/13/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									0.05
HB-GP-13	7/13/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-13	7/13/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-14	7/14/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-14	7/14/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND

TABLE 2.3d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-GP-15	7/14/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-GP-15	7/14/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND
HB-GP-16	7/18/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-16	7/18/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-17	7/18/2000	0	0.17	57-74-9	CHLORDANE	N	UJ	mg/kg	0.07
HB-GP-17	7/18/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.08
Total Chlordane =									0.08
HB-GP-18	7/17/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-GP-18	7/17/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.04
Total Chlordane =									0.04
HB-GP-19	7/17/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-GP-19	7/17/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-GP-20	7/17/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-20	7/17/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-HB-02I	7/19/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.05
HB-HB-02I	7/19/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.09
Total Chlordane =									0.14
HB-HB-03S	7/26/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-HB-03S	7/26/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.005
Total Chlordane =									0.005
HB-HB-04S	7/27/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.04
HB-HB-04S	7/27/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.04
Total Chlordane =									ND
HB-HB-05I	7/27/2000	0	0.17	57-74-9	CHLORDANE	Y	J	mg/kg	0.04
HB-HB-05I	7/27/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.1
Total Chlordane =									0.14
HB-HB-06S	8/2/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-HB-06S	8/2/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND
HB-HB-16D	1/7/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0059
HB-HB-16D	1/7/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0059
Total Chlordane =									ND
HB-HB-16D	1/7/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0073
HB-HB-16D	1/7/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.049
Total Chlordane =									0.049
HB-HBW-01	8/4/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.06
HB-HBW-01	8/4/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.06
Total Chlordane =									ND

TABLE 2.3d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HBW-02	8/4/2000	0	0.17	57-74-9	CHLORDANE	N	UJ	mg/kg	0.07
HB-HBW-02	8/4/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.07
Total Chlordane =									ND
HB-HBW-03	8/7/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.05
HB-HBW-03	8/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									ND
HB-HBW-04	8/7/2000	0	0.17	57-74-9	CHLORDANE	N	UJ	mg/kg	0.08
HB-HBW-04	8/7/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.08
Total Chlordane =									ND
HB-HBW-05	8/8/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-HBW-05	8/8/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-HBW-06	8/8/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.02
HB-HBW-06	8/8/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.02
Total Chlordane =									ND
HB-RISB-01	12/13/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0071
HB-RISB-01	12/13/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0071
Total Chlordane =									ND
HB-RISB-01	12/13/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0079
HB-RISB-01	12/13/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0079
Total Chlordane =									ND
HB-RISB-02	12/13/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.0065
HB-RISB-02	12/13/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0065
Total Chlordane =									ND
HB-RISB-02	12/13/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.007
HB-RISB-02	12/13/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.007
Total Chlordane =									ND
HB-SB-63	11/1/2006	4	6	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.028
HB-SB-63	11/1/2006	4	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-SB-64	11/2/2006	2	4	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.03
HB-SB-64	11/2/2006	2	4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-SB-65	11/3/2006	0	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.028
HB-SB-65	11/3/2006	0	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-SB-84	10/27/2006	6	8	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.032
HB-SB-84	10/27/2006	6	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.032
Total Chlordane =									ND
HB-SB-85	10/26/2006	4	6	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.014
HB-SB-85	10/26/2006	4	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.014
Total Chlordane =									ND

TABLE 2.3d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-SEEP-2	9/9/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.037
HB-SEEP-2	9/9/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.037
Total Chlordane =									ND
HB-SEEP-2	9/9/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.04
HB-SEEP-2	9/9/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.04
Total Chlordane =									ND
HB-SS-02	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0041
HB-SS-02	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0041
Total Chlordane =									ND
HB-SS-02	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0058
HB-SS-02	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0058
Total Chlordane =									ND
HB-SS-04	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.006
HB-SS-04	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.006
Total Chlordane =									ND
HB-SS-04	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0059
HB-SS-04	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0059
Total Chlordane =									ND
HB-SS-05	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.082
HB-SS-05	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.082
Total Chlordane =									ND
HB-SS-05	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.08
HB-SS-05	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.08
Total Chlordane =									ND
HB-SS-06	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.09
HB-SS-06	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.09
Total Chlordane =									ND
HB-SS-06	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.077
HB-SS-06	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.077
Total Chlordane =									ND
HB-SS-07	12/5/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0061
HB-SS-07	12/5/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0061
Total Chlordane =									ND
HB-SS-08	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.036
HB-SS-08	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.036
Total Chlordane =									ND
HB-SS-08	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.056
HB-SS-08	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.056
Total Chlordane =									ND
HB-SS-09	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.083
HB-SS-09	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.083
Total Chlordane =									ND

TABLE 2.3d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-SS-09	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.063
HB-SS-09	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.063
Total Chlordane =									ND
HB-SS-10	12/4/2002	0	0.5	57-74-9	CHLORDANE	Y	J	mg/kg	0.0063
HB-SS-10	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0059
Total Chlordane =									0.0122
HB-SS-10	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-SS-10	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-SS-11	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-SS-11	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-SS-11	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-SS-11	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-TP-01	7/5/2000	6	6	57-74-9	CHLORDANE	N	U	mg/kg	0.005
HB-TP-01	7/5/2000	6	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.005
Total Chlordane =									ND
HB-TP-01B	7/19/2000	8	8	57-74-9	CHLORDANE	N	UJ	mg/kg	0.1
HB-TP-01B	7/19/2000	8	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.1
Total Chlordane =									ND
HB-TP-01D	7/19/2000	3	3	57-74-9	CHLORDANE	N	UJ	mg/kg	0.05
HB-TP-01D	7/19/2000	3	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.05
Total Chlordane =									ND
HB-TP-05	7/5/2000	3	3	57-74-9	CHLORDANE	Y	J	mg/kg	0.1
HB-TP-05	7/5/2000	3	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.3
Total Chlordane =									0.4
HB-TP-07	7/5/2000	2.5	2.5	57-74-9	CHLORDANE	N	U	mg/kg	0.003
HB-TP-07	7/5/2000	2.5	2.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.003
Total Chlordane =									ND
HB-TP-09	7/5/2000	2	2	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-TP-09	7/5/2000	2	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.002
Total Chlordane =									0.002
HB-TP-12	7/6/2000	3	3	57-74-9	CHLORDANE	N	U	mg/kg	0.003
HB-TP-12	7/6/2000	3	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.003
Total Chlordane =									ND
HB-TP-15	7/6/2000	3	3	57-74-9	CHLORDANE	Y	J	mg/kg	0.1
HB-TP-15	7/6/2000	3	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.08
Total Chlordane =									0.18
HB-TP-18	7/6/2000	4	4	57-74-9	CHLORDANE	N	UJ	mg/kg	0.4
HB-TP-18	7/6/2000	4	4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.4
Total Chlordane =									ND

TABLE 2.3d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-TP-19	7/7/2000	6	6	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-TP-19	7/7/2000	6	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-TP-20	7/7/2000	6.5	6.5	57-74-9	CHLORDANE	N	U	mg/kg	0.01
HB-TP-20	7/7/2000	6.5	6.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.01
Total Chlordane =									ND
HB-TP-20A	7/18/2000	5	5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.03
HB-TP-20A	7/18/2000	5	5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.02
Total Chlordane =									0.02
HB-TP-21	7/18/2000	3.5	3.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.06
HB-TP-21	7/18/2000	3.5	3.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.03
Total Chlordane =									0.03
HB-TP-22	7/18/2000	4	4	57-74-9	CHLORDANE	N	UJ	mg/kg	0.2
HB-TP-22	7/18/2000	4	4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.2
Total Chlordane =									ND
HB-TP-23	7/18/2000	4.5	4.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.05
HB-TP-23	7/18/2000	4.5	4.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.05
Total Chlordane =									ND
HB-TP-46A	11/16/2006	3	4	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-TP-46A	11/16/2006	3	4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND

TABLE 2.3e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-GP-01	7/14/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-02	7/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-03	7/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-04	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.003	0.0015
HB-GP-05	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.005	0.0025
HB-GP-06	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.006	0.003
HB-GP-07	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.006	0.003
HB-GP-08	7/10/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.003	0.0015
HB-GP-09	7/12/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-10	7/12/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-GP-11	7/13/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-GP-12	7/13/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-GP-13	7/13/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-14	7/14/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-GP-15	7/14/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-16	7/18/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-17	7/18/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-GP-18	7/17/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-19	7/17/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-GP-20	7/17/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-02I	7/19/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-HB-03S	7/26/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-04S	7/27/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.003	0.0015
HB-HB-05I	7/27/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-06S	8/2/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.004	0.004
HB-HB-16D	1/7/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.015	
HB-HB-16D	1/7/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.015	
HB-HB-16D	1/7/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.015
HB-HB-16D	1/7/2003	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.017	
HB-HB-16D	1/7/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.017	
HB-HB-16D	1/7/2003	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.017
HB-HBW-01	8/4/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.003	0.003
HB-HBW-02	8/4/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.013	0.013
HB-HBW-03	8/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HBW-04	8/7/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.002	0.002
HB-HBW-05	8/8/2000	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HBW-06	8/8/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.001	0.001
HB-RISB-01	12/13/2002	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.21	
HB-RISB-01	12/13/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.32	
HB-RISB-01	12/13/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.53
HB-RISB-01	12/13/2002	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.21	
HB-RISB-01	12/13/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.42	
HB-RISB-01	12/13/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.63
HB-RISB-02	12/13/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0096	
HB-RISB-02	12/13/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0096	
HB-RISB-02	12/13/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0096
HB-RISB-02	12/13/2002	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.033	
HB-RISB-02	12/13/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.078	
HB-RISB-02	12/13/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.111
HB-SB-63	11/1/2006	4	6	1330-20-7	XYLENES, TOTAL	Y		mg/kg	65	65
HB-SB-64	11/2/2006	2	4	1330-20-7	XYLENES, TOTAL	Y		mg/kg	7.4	7.4
HB-SB-65	11/3/2006	0	2	1330-20-7	XYLENES, TOTAL	Y		mg/kg	2.1	2.1
HB-SB-84	10/27/2006	6	8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	3.6	3.6
HB-SB-85	10/26/2006	4	6	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	4.7	4.7
HB-SEEP-2	9/9/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	2.1	
HB-SEEP-2	9/9/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	5.9	
HB-SEEP-2	9/9/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		8
HB-SEEP-2	9/9/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	2.8	
HB-SEEP-2	9/9/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	9.6	
HB-SEEP-2	9/9/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		12.4
HB-SS-02	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.006	
HB-SS-02	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.006	
HB-SS-02	12/3/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.006
HB-SS-02	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0086	
HB-SS-02	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0086	
HB-SS-02	12/3/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0086

TABLE 2.3e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-SS-04	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.014	
HB-SS-04	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.014	
HB-SS-04	12/3/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.014
HB-SS-04	12/3/2002	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.011	
HB-SS-04	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.13	
HB-SS-04	12/3/2002	0.5	1	CALCULATED	TOTAL	Y		mg/kg		0.141
HB-SS-05	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.019	
HB-SS-05	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.019	
HB-SS-05	12/3/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.019
HB-SS-05	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.022	
HB-SS-05	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.022	
HB-SS-05	12/3/2002	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.022
HB-SS-06	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.02	
HB-SS-06	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.009	
HB-SS-06	12/3/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.009
HB-SS-06	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.016	
HB-SS-06	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0091	
HB-SS-06	12/3/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0091
HB-SS-07	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0089	
HB-SS-07	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0089	
HB-SS-07	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0089
HB-SS-08	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.012	
HB-SS-08	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.012	
HB-SS-08	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.012
HB-SS-08	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0083	
HB-SS-08	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0083	
HB-SS-08	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0083
HB-SS-09	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.012	
HB-SS-09	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.012	
HB-SS-09	12/4/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.012
HB-SS-09	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0093	
HB-SS-09	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0046	
HB-SS-09	12/4/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0046
HB-SS-10	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0082	
HB-SS-10	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0082	
HB-SS-10	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0082
HB-SS-10	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0078	
HB-SS-10	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0078	
HB-SS-10	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0078
HB-SS-11	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0074	
HB-SS-11	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0074	
HB-SS-11	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0074
HB-SS-11	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0081	
HB-SS-11	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0053	
HB-SS-11	12/4/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0053
HB-TP-01	7/5/2000	6	6	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	6.2	6.2
HB-TP-01B	7/19/2000	8	8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.84	0.84
HB-TP-01D	7/19/2000	3	3	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.87	0.87
HB-TP-05	7/5/2000	3	3	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.006	0.003
HB-TP-07	7/5/2000	2.5	2.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-TP-09	7/5/2000	2	2	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.004	0.002
HB-TP-12	7/6/2000	3	3	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.005	0.0025
HB-TP-15	7/6/2000	3	3	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.006	0.003
HB-TP-18	7/6/2000	4	4	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	4.9	4.9
HB-TP-19	7/7/2000	6	6	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.035	0.035
HB-TP-20	7/7/2000	6.5	6.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-TP-20A	7/18/2000	5	5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-TP-21	7/18/2000	3.5	3.5	1330-20-7	XYLENES, TOTAL	Y		mg/kg	8	8
HB-TP-22	7/18/2000	4	4	1330-20-7	XYLENES, TOTAL	Y		mg/kg	490	490
HB-TP-23	7/18/2000	4.5	4.5	1330-20-7	XYLENES, TOTAL	Y		mg/kg	3.3	3.3
HB-TP-46A	11/16/2006	3	4	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	1.2	1.2

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.4a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SHALLOW GROUND WATER : VAPOR INTRUSION
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs*)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	Target Groundwater Concentration Corresponding to Target Indoor Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor = 0.001 and Partitioning Across the Water Table Obeys Henry's Law (5)	Screening Toxicity Value	COPC Flag (Y/N)	Rationale for Selection or Deletion (6)		
Lakeshore Area - Shallow Ground Water	SVOCs																
	92-52-4	1,1'-BIPHENYL	4 J	83 J	ug/l	HB-HB-04S	4/6	10-10	8.30E+01			**	nc	**	N	INC	
	120-83-2	2,4-DICHLOROPHENOL	18 J	75	ug/l	HB-WA-03S	5/26	9.6-1000	7.50E+01					NV	Y	NTX	
	105-67-9	2,4-DIMETHYLPHENOL	14 J	7500	ug/l	HB-HB-02S	11/26	9.6-97	7.50E+03					NV	Y	NTX	
	95-57-8	2-CHLOROPHENOL	2 J	2 J	ug/l	HB-WA-03S	1/26	9.6-1000	2.00E+00			1.10 E+02	nc	1.10E+02	N	BSL	
	91-57-6	2-METHYLNAPHTHALENE	1 J	9800	ug/l	HB-HB-04S	19/26	9.6-10	9.80E+03			3.30 E+02	nc	3.30E+02	Y	ASL	
	95-48-7	2-METHYLPHENOL	3.8 J	8000	ug/l	HB-HB-02S	12/26	9.6-97	8.00E+03					NV	Y	NTX	
	88-75-5	2-NITROPHENOL	2.6 J	3 J	ug/l	HB-WA-08S	2/26	9.6-1000	3.00E+00					NV	Y	NTX	
	34METPH	3&4-METHYLPHENOL	2 J	16000	ug/l	HB-HB-02S	11/20	9.6-97	1.60E+04					NV	Y	NTX	
	106-44-5	4-METHYLPHENOL	8.9 J	12000	ug/l	HB-HB-02S	4/6	10-10	1.20E+04					NV	Y	NTX	
	100-02-7	4-NITROPHENOL	3 J	8 J	ug/l	HB-WA-08S	2/26	48-5100	8.00E+00					NV	Y	NTX	
	83-32-9	ACENAPHTHENE	17	2200	ug/l	HB-HB-04S	13/26	9.6-1000	2.20E+03			**	nc	**	N	INC	
	208-96-8	ACENAPHTHYLENE	1.2 J	2700	ug/l	HB-HB-04S	12/26	9.6-970	2.70E+03					NV	Y	NTX	
	120-12-7	ANTHRACENE	2.8 J	2000	ug/l	HB-HB-04S	6/26	9.6-1000	2.00E+03					NV	Y	NTX	
	100-52-7	BENZALDEHYDE	2.3 J	13 J	ug/l	HB-HB-02S	2/6	10-100	1.30E+01			3.60 E+04	nc	3.60E+04	N	BSL	
	56-55-3	BENZ(A)ANTHRACENE	20 J	690 J	ug/l	HB-HB-04S	4/26	9.6-1000	6.90E+02			2.00 E-01		NV	Y	NTX	
	50-32-8	BENZO(A)PYRENE	16 J	310 J	ug/l	HB-HB-04S	2/26	9.6-1000	3.10E+02					NV	Y	NTX	
	205-99-2	BENZO(B)FLUORANTHENE	10 UJ	240 J	ug/l	HB-HB-04S	3/26	9.6-1000	2.40E+02			**	c	**	N	INC	
	207-08-9	BENZO(K)FLUORANTHENE	340 J	340 J	ug/l	HB-HB-04S	1/26	9.6-1000	3.40E+02					NV	Y	NTX	
	65-85-0	BENZOIC ACID	3 J	2300 J	ug/l	HB-HB-02S	5/7	510-5000	2.30E+03					NV	Y	NTX	
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1 J	65	ug/l	HB-WA-08S	4/26	9.6-1000	6.50E+01					NV	Y	NTX	
	86-74-8	CARBAZOLE	6.8 J	840 J	ug/l	HB-HB-04S	16/25	9.6-10	8.40E+02					NV	Y	NTX	
	218-01-9	CHRYSENE	16 J	590 J	ug/l	HB-HB-04S	4/26	9.6-1000	5.90E+02			**	c	**	N	INC	
	132-64-9	DIBENZOFURAN	5.9 J	3400	ug/l	HB-HB-04S	14/26	9.6-970	3.40E+03			**	nc	**	N	INC	
	84-74-2	DI-N-BUTYL PHTHALATE	2 J	2 J	ug/l	HB-WA-08S	1/26	9.6-1000	2.00E+00					NV	Y	NTX	
	206-44-0	FLUORANTHENE	1.6 J	3200	ug/l	HB-HB-04S	8/26	9.6-1000	3.20E+03					NV	Y	NTX	
	86-73-7	FLUORENE	3 J	4200	ug/l	HB-HB-04S	15/26	9.6-970	4.20E+03			**	nc	**	N	INC	
	193-39-5	INDENO(1,2,3-CD)PYRENE	110 J	110 J	ug/l	HB-HB-04S	1/26	9.6-1000	1.10E+02					NV	Y	NTX	
	91-20-3	NAPHTHALENE	1.5 J	35000	ug/l	HB-HB-04S	24/29	1-10	3.50E+04			1.50 E+01	nc	1.50E+01	Y	ASL	
	85-01-8	PHENANTHRENE	4.7 J	8300	ug/l	HB-HB-04S	15/26	9.6-730	8.30E+03					NV	Y	NTX	
	108-95-2	PHENOL	2.8 J	18000	ug/l	HB-HB-02S	21/26	10-10	1.80E+04			**	nc	**	Y	NTX	
	129-00-0	PYRENE	1.2 J	1900	ug/l	HB-HB-04S	8/26	9.6-1000	1.90E+03					nc	**	N	INC
	VOCs																
	120-82-1	1,2,4-TRICHLOROBENZENE	230	230	ug/l	HB-WA-03S	1/31	1-1000	2.30E+02			7.00 E+01	**	nc	**	N	INC
	95-63-6	1,2,4-TRIMETHYLBENZENE	2 J	420	ug/l	HB-HB-02S	5/6	0.5-0.5	4.20E+02				2.40 E+00	nc	2.40E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	0.19 J	3800	ug/l	HB-WA-03S	12/33	0.5-1000	3.80E+03			6.00 E+02		nc	2.60E+02	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.6 J	150	ug/l	HB-HB-02S	5/6	0.5-0.5	1.50E+02				2.50 E+00	nc	2.50E+00	Y	ASL
	541-73-1	1,3-DICHLOROBENZENE	3.6	62 J	ug/l	HB-WA-03S	4/33	0.5-1000	6.20E+01				8.30 E+01	nc	8.30E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.11 J	4500	ug/l	HB-WA-03S	13/33	0.5-1000	4.50E+03				8.20 E+02	nc	8.20E+02	Y	ASL
	78-93-3	2-BUTANONE	2 J	21	ug/l	HB-HB-03S	9/27	10-2500	2.10E+01				4.40 E+04	nc	4.40E+04	N	BSL
	591-78-6	2-HEXANONE	0.3 J	2.6 J	ug/l	HB-HB-03S	3/27	5-1250	2.60E+00						NV	Y	NTX
	106-43-4	4-CHLOROTOLUENE	2 J	2 J	ug/l	HB-HB-06S	1/6	0.5-50	2.00E+00						NV	Y	NTX
	108-10-1	4-METHYL-2-PENTANONE	0.5 J	0.5 J	ug/l	HB-WA-08S	2/27	5-1250	5.00E-01						NV	Y	NTX
	67-64-1	ACETONE	3.06 J	460 J	ug/l	HB-HB-04S	16/27	10-2500	4.60E+02				2.20 E+04	nc	2.20E+04	N	BSL
	98-86-2	ACETOPHENONE	6.7 J	6.7 J	ug/l	HB-HB-03S	1/6	10-110	6.70E+00				8.00 E+04	nc	8.00E+04	N	BSL
	71-43-2	BENZENE	0.3 J	3900	ug/l	HB-HB-02S	22/27	0.5-50	3.90E+03			5.00 E+00		c	1.37E+01	Y	TOX
	75-15-0	CARBON DISULFIDE	4.7 J	4.7 J	ug/l	HB-HB-03S	2/21	0.5-500	4.70E+00				5.60 E+01	nc	5.60E+01	N	BSL
	108-90-7	CHLOROGENZENE	8.7	580	ug/l	HB-WA-03S	10/27	0.5-250	5.80E+02			1.00 E+02		nc	3.90E+01	Y	ASL
	67-66-3	CHLOROFORM	0.4 J	0.4 J	ug/l	HB-WA-08S	1/27	0.5-250	4.00E-01				7.33 E+00	c	7.33E+00	N	BSL
	156-59-2	CIS-1,2-DICHLOROETHENE	1 J	1 J	ug/l	HB-HB-06S	1/23	0.5-250	1.00E+00			7.00 E+01			NV	Y	NTX
	100-41-4	ETHYLBENZENE	0.7 J	350	ug/l	HB-HB-04S	16/27	0.5-100	3.50E+02			7.00 E+02		c	3.01E+01	Y	ASL
	98-82-8	ISOPROPYLBENZENE	0.1 J	3	ug/l	HB-HB-06S	3/12	0.5-125	3.00E+00						NV	Y	NTX
	75-09-2	METHYLENE CHLORIDE	5.5 J	5.5 J	ug/l	HB-HB-03S	1/27	2-500	5.50E+00				5.80 E+01	c	5.80E+01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.8 J	0.8 J	ug/l	HB-HB-06S	1/6	0.5-50	8.00E-01						NV	Y	NTX
	135-98-8	SEC-BUTYLBENZENE	1 J	1 J	ug/l	HB-HB-06S	1/6	0.5-50	1.00E+00				2.50 E+01	nc	2.50E+01	N	BSL

TABLE 2.4a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SHALLOW GROUND WATER : VAPOR INTRUSION
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs*)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	Target Groundwater Concentration Corresponding to Target Indoor Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor = 0.001 and Partitioning Across the Water Table Obeys Henry's Law (5)	Screening Toxicity Value	COPC Flag (Y/N)	Rationale for Selection or Deletion (6)	
	100-42-5	STYRENE	0.3 J	850	ug/l	HB-HB-02S	11/27	0.5-50	8.50E+02		1.00 E+02	8.89 E+03	nc	8.89E+03	N	BSL
	127-18-4	TETRACHLOROETHENE	0.2 J	0.2 J	ug/l	HB-WA-08S	1/27	0.5-250	2.00E-01		5.00 E+00	1.33 E+01	c	1.33E+01	N	BSL
	108-88-3	TOLUENE	0.39 J	5740	ug/l	HB-HB-02S	19/27	0.5-100	5.74E+03		1.00 E+03	1.50 E+02	nc	1.50E+02	Y	ASL
	75-01-4	VINYL CHLORIDE	0.7 J	4.1 J	ug/l	HB-HB-03S	4/27	1-250	4.10E+00		2.00 E+00	1.45 E+00	c	1.45E+00	Y	TOX
	1330-20-7	XYLENES, TOTAL ^a	0.29 J	3380	ug/l	HB-HB-02S	19/27	0.25-150	3.38E+03		1.00 E+04	2.20 E+03	nc	2.20E+03	Y	ASL

Footnotes:

* Sample start depth less than or equal to 10 ft bgs.

** Target soil gas concentration exceeds maximum possible vapor concentration (pathway incomplete)

(1) J - estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) Primary and Secondary Drinking Water Regulations

(5) USEPA - OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils (Subsurface Vapor Intrusion Guidance) Tables. November 2002. ca = Cancer; nc = Noncancer. Screening criteria correspond to a cancer risk of 10⁻⁶ and a noncancer hazard of 0.1. For USEPA (2002) criteria that defaulted to MCLs, criteria were derived (in italics) from USEPA (2009) RSL residential air concentration based on an attenuation factor of 10 and the Henry's Law constant for each compound at 25 deg C.

(6) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level; INC - Pathway Incomplete

a =Target groundwater concentration for p-xylene (CAS #106-42-3) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NV: No Value

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.4b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SHALLOW GROUND WATER : VAPOR INTRUSION

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-02S	5/17/2001	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y		ug/l	2800	2800
HB-HB-02S	5/20/2003	4.01	14.01	95-47-6	O-XYLENE	Y		ug/l	810	
HB-HB-02S	5/20/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	Y		ug/l	2100	
HB-HB-02S	5/20/2003	4.01	14.01	CALCULATED	TOTAL	Y		ug/l		2910
HB-HB-02S	8/22/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	Y		ug/l	2000	
HB-HB-02S	8/22/2003	4.01	14.01	95-47-6	O-XYLENE	Y		ug/l	770	
HB-HB-02S	8/22/2003	4.01	14.01	CALCULATED	TOTAL	Y		ug/l		2770
HB-HB-02S	3/15/2007	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y		ug/l	3380	3380
HB-WA-03S	4/6/1992	3	13	1330-20-7	XYLENES, TOTAL	Y		ug/l	98	98
HB-WA-03S	10/14/1992	3	13	1330-20-7	XYLENES, TOTAL	N	U	ug/l	300	150
HB-HB-03S	5/22/2001	4.96	14.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	23	23
HB-HB-03S	5/14/2003	4.96	14.96	XYLENES1314	XYLENES, M & P	Y		ug/l	32	
HB-HB-03S	5/14/2003	4.96	14.96	95-47-6	O-XYLENE	Y		ug/l	11	
HB-HB-03S	5/14/2003	4.96	14.96	CALCULATED	TOTAL	Y		ug/l		43
HB-HB-03S	8/19/2003	4.96	14.96	XYLENES1314	XYLENES, M & P	Y		ug/l	51	
HB-HB-03S	8/19/2003	4.96	14.96	95-47-6	O-XYLENE	Y		ug/l	15	
HB-HB-03S	8/19/2003	4.96	14.96	CALCULATED	TOTAL	Y		ug/l		43
HB-HB-03S	3/8/2007	4.96	14.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	33	33
HB-HB-04S	5/17/2001	8.59	18.59	1330-20-7	XYLENES, TOTAL	Y		ug/l	800	800
HB-HB-04S	5/16/2003	8.59	18.59	XYLENES1314	XYLENES, M & P	Y		ug/l	1400	
HB-HB-04S	5/16/2003	8.59	18.59	95-47-6	O-XYLENE	Y		ug/l	540	
HB-HB-04S	5/16/2003	8.59	18.59	CALCULATED	TOTAL	Y		ug/l		1940
HB-HB-04S	8/20/2003	8.59	18.59	XYLENES1314	XYLENES, M & P	Y		ug/l	2300	
HB-HB-04S	8/20/2003	8.59	18.59	95-47-6	O-XYLENE	Y		ug/l	870	
HB-HB-04S	8/20/2003	8.59	18.59	CALCULATED	TOTAL	Y		ug/l		3170
HB-HB-04S	3/14/2007	8.59	18.59	1330-20-7	XYLENES, TOTAL	Y		ug/l	3220	3220
HB-HB-05S	5/23/2001	7.03	17.03	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-05S	5/20/2003	7.03	17.03	XYLENES1314	XYLENES, M & P	N	U	ug/l	50	
HB-HB-05S	5/20/2003	7.03	17.03	95-47-6	O-XYLENE	N	U	ug/l	50	
HB-HB-05S	5/20/2003	7.03	17.03	CALCULATED	TOTAL	N	U	ug/l		50
HB-HB-05S	8/19/2003	7.03	17.03	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-05S	8/19/2003	7.03	17.03	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-05S	8/19/2003	7.03	17.03	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-05S	3/13/2007	7.03	17.03	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-06S	5/23/2001	3	13	1330-20-7	XYLENES, TOTAL	Y		ug/l	55	55
HB-HB-06S	5/22/2003	3	13	XYLENES1314	XYLENES, M & P	Y		ug/l	35	
HB-HB-06S	5/22/2003	3	13	95-47-6	O-XYLENE	Y		ug/l	34	
HB-HB-06S	5/22/2003	3	13	CALCULATED	TOTAL	Y		ug/l		69
HB-HB-06S	8/25/2003	3	13	XYLENES1314	XYLENES, M & P	Y		ug/l	11	
HB-HB-06S	8/25/2003	3	13	95-47-6	O-XYLENE	Y		ug/l	12	
HB-HB-06S	8/25/2003	3	13	CALCULATED	TOTAL	Y		ug/l		23
HB-HB-06S	3/20/2007	3	13	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2.2	2.2
HB-WA-08S	1/5/1995	8.95	18.95	1330-20-7	XYLENES, TOTAL	N	U	ug/l	10	5
HB-WA-08S	5/21/2001	8.95	18.95	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	5	5
HB-WA-08S	5/15/2003	8.95	18.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-WA-08S	5/15/2003	8.95	18.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-WA-08S	5/15/2003	8.95	18.95	CALCULATED	TOTAL	N	U	ug/l		5
HB-WA-08S	8/14/2003	8.95	18.95	XYLENES1314	XYLENES, M & P	Y		ug/l	6.4	
HB-WA-08S	8/14/2003	8.95	18.95	95-47-6	O-XYLENE	Y	J	ug/l	3.3	
HB-WA-08S	8/14/2003	8.95	18.95	CALCULATED	TOTAL	Y		ug/l		9.7
HB-WA-08S	3/12/2007	8.95	18.95	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.29	0.29

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.5a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs*)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
Lakeshore Area - Shallow Ground Water	METALS																		
	7429-90-5	ALUMINUM	0.11	12.7 J	mg/L	HB-HB-05S	24/27	0.055-0.3	1.27E+01		2.00 E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	Y	ASL	
	7440-36-0	ANTIMONY	0.0029 J	0.0029 J	mg/L	HB-HB-05S	1/28	0.0014-0.3	2.90E-03		6.00 E-03	1.46E-03	N	1.46E-03	nc	1.46E-03	Y	ASL	
	7440-38-2	ARSENIC	0.0037 J	0.0144	mg/L	HB-WA-03S	7/28	0.0016-0.05	1.44E-02		1.00 E-02	4.46E-05	C	4.48E-05	ca	4.46E-05	Y	TOX	
	7440-39-3	BARIUM	0.0015 J	20.3	mg/L	HB-HB-06S	27/28	0.02-0.02	2.03E+01		2.00 E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	Y	ASL	
	7440-41-7	BERYLLIUM	0.00022 J	0.0007 J	mg/L	HB-HB-02S	4/28	0.000076-0.05	7.00E-04		4.00 E-03	7.30E-03	N	7.30E-03	nc	7.30E-03	N	BSL	
	7440-43-9	CADMIUM	0.0012 J	0.0116	mg/L	HB-WA-03S	3/27	0.00024-0.05	1.16E-02		5.00 E-03	1.83E-03	N	1.82E-03	nc	1.82E-03	Y	ASL	
	7440-70-2	CALCIUM	158	7970	mg/L	HB-HB-06S	28/28	-	7.97E+03			NV	NV	NV	N	NUT			
	7440-47-3	CHROMIUM ^a	0.0026 J	0.0512	mg/L	HB-HB-05S	20/28	0.002-0.03	5.12E-02		1.00 E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX	
	7440-48-4	COBALT	0.0029 J	0.0035	mg/L	HB-WA-03S	3/28	0.00093-0.25	3.50E-03			NV	7.30E-02	nc	7.30E-02	N	BSL		
	7440-50-8	COPPER	0.0026	0.0866	mg/L	HB-HB-02S	16/28	0.01-0.06	8.66E-02		1.30 E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	N	BSL	
	57-12-5	CYANIDE	0.0108	0.12	mg/L	HB-HB-05S	15/26	0.01-0.01	1.20E-01		2.00 E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	Y	ASL	
	7439-89-6	IRON	0.03 J	29	mg/L	HB-HB-06S	26/28	0.0879-0.3	2.90E+01		3.00 E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL	
	7439-92-1	LEAD	0.0079 J	0.103 J	mg/L	HB-HB-02S	16/28	0.005-0.0524	1.03E-01		1.50 E-02	NV	NV	NV	N	Y	ASL		
	7439-95-4	MAGNESIUM	0.1 J	513	mg/L	HB-HB-05S	24/28	0.08-1.5	5.13E+02			NV	NV	NV	N	NUT			
	7439-96-5	MANGANESE	0.0023 J	1.9	mg/L	HB-HB-06S	23/28	0.0069-0.05	1.90E+00		5.00 E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	Y	ASL	
	7439-97-6	MERCURY ^b	0.00011 J	0.0088	mg/L	HB-HB-03S	16/28	0.00018-0.0026	8.80E-03		2.00 E-03	3.65E-04	N	3.65E-04	nc	3.65E-04	Y	ASL	
	7440-02-0	NICKEL	0.0012 J	0.0328 J	mg/L	HB-HB-02S	14/28	0.04-0.25	3.28E-02			7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL	
	7440-09-7	POTASSIUM	1.99 J	182 J	mg/L	HB-HB-06S	26/29	2-110	1.82E+02			NV	NV	NV	N	NUT			
	7782-49-2	SELENIUM	0.0026 J	0.0086 J	mg/L	HB-HB-05S	5/28	0.0018-0.05	8.60E-03		5.00 E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL	
	7440-22-4	SILVER	0.00085 J	0.00099 J	mg/L	HB-HB-02S	2/28	0.00073-0.05	9.90E-04		1.00 E-01	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL	
	7440-23-5	SODIUM	62 J	10560	mg/L	HB-HB-06S	28/28	-	1.06E+04			NV	NV	NV	N	NUT			
	7440-62-2	VANADIUM	0.00074 J	0.0276 J	mg/L	HB-HB-02S	13/28	0.015-0.25	2.76E-02			3.65E-03	N	3.65E-03	nc	3.65E-03	Y	ASL	
	7440-66-6	ZINC	0.004	0.159	mg/L	HB-HB-02S	12/28	0.015-0.1	1.59E-01		5.00 E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL	
	PCBs																		
		HIGHLY CHLORINATED PCBs ^c	0.07	0.07	ug/l	HB-HB-06S	1/24	0.5-0.9	7.00E-02				3.35E-02	C	NV		3.35E-02	Y	ASL
		TOTAL PCBs ^d	0.07	0.07	ug/l	HB-HB-06S	1/24	0.5-0.9	7.00E-02				3.35E-02	C	3.36E-02	ca	3.35E-02	Y	ASL
	PESTICIDES																		
	50-29-3	4,4'-DDT	20 J	20 J	ug/l	HB-HB-04S	1/24	0.093-2.1	2.00E+01				1.97E-01	C	1.98E-01	ca	1.97E-01	Y	ASL
	SVOCs																		
	92-52-4	1,1'-BIPHENYL	4 J	83 J	ug/l	HB-HB-04S	4/6	10-10	8.30E+01				3.04E+01	N	3.04E+01	nc	3.04E+01	Y	ASL
	120-83-2	2,4-DICHLOROPHENOL	18 J	75	ug/l	HB-WA-03S	5/26	9.6-1000	7.50E+01				1.10E+01	N	1.09E+01	nc	1.09E+01	Y	ASL
	105-67-9	2,4-DIMETHYLPHENOL	14 J	7500	ug/l	HB-HB-02S	11/26	9.6-97	7.50E+03				7.30E+01	N	7.30E+01	nc	7.30E+01	Y	ASL
	95-57-8	2-CHLOROPHENOL	2 J	2 J	ug/l	HB-WA-03S	1/26	9.6-1000	2.00E+00				3.04E+00	N	3.04E+00	nc	3.04E+00	N	BSL
	91-57-6	2-METHYLNAPHTHALENE	1 J	9800	ug/l	HB-HB-04S	19/26	9.6-10	9.80E+03				2.43E+00	N	NV		2.43E+00	Y	ASL
	95-48-7	2-METHYLPHENOL	3.8 J	8000	ug/l	HB-HB-02S	12/26	9.6-97	8.00E+03				1.83E+02	N	1.82E+02	nc	1.82E+02	Y	ASL
	88-75-5	2-NITROPHENOL	2.6 J	3 J	ug/l	HB-WA-08S	2/26	9.6-1000	3.00E+00				NV	NV	NV	Y	NTX		
	34METPH	3&4-METHYLPHENOL ^e	2 J	16000	ug/l	HB-HB-02S	11/20	9.6-97	1.60E+04				1.83E+01	N	1.82E+01	nc	1.82E+01	Y	ASL
	106-44-5	4-METHYLPHENOL	8.9 J	12000	ug/l	HB-HB-02S	4/6	10-10	1.20E+04				1.83E+01	N	1.82E+01	nc	1.82E+01	Y	ASL
	100-02-7	4-NITROPHENOL	3 J	8 J	ug/l	HB-WA-08S	2/26	48-5100	8.00E+00				NV	NV	NV	Y	NTX		
	83-32-9	ACENAPHTHENE	17	2200	ug/l	HB-HB-04S	13/26	9.6-1000	2.20E+03				3.65E+01	N	3.65E+01	nc	3.65E+01	Y	ASL
	208-96-8	ACENAPHTHYLENE	1.2 J	2700	ug/l	HB-HB-04S	12/26	9.6-970	2.70E+03				NV	NV	NV	Y	NTX		
	120-12-7	ANTHRACENE	2.8 J	2000	ug/l	HB-HB-04S	6/26	9.6-1000	2.00E+03				1.83E+02	N	1.83E+02	nc	1.83E+02	Y	ASL
	100-52-7	BENZALDEHYDE	2.3 J	13 J	ug/l	HB-HB-02S	2/6	10-100	1.30E+01				3.65E+02	N	3.65E+02	nc	3.65E+02	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	20 J	690 J	ug/l	HB-HB-04S	4/26	9.6-1000	6.90E+02				3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	50-32-8	BENZO(A)PYRENE	16 J	310 J	ug/l	HB-HB-04S	2/26	9.6-1000	3.10E+02		2.00 E-01		3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	10 UJ	240 J	ug/l	HB-HB-04S	3/26	9.6-1000	2.40E+02				3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	207-08-9	BENZO(K)FLUORANTHENE	340 J	340 J	ug/l	HB-HB-04S	1/26	9.6-1000	3.40E+02				3.00E-01	C	9.21E-01	ca	3.00E-01	Y	ASL
	65-85-0	BENZOIC ACID	3 J	2300 J	ug/l	HB-HB-02S	5/7	510-5000	2.30E+03				1.46E+04	N	1.46E+04	nc	1.46E+04	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1 J	65	ug/l	HB-WA-08S	4/26	9.6-1000	6.50E+01		6.00 E+00		4.78E+00	C	4.80E+00	ca	4.78E+00	Y	ASL
	86-74-8	CARBAZOLE	6.8 J	840 J	ug/l	HB-HB-04S	16/25	9.6-10	8.40E+02				3.35E+00	C	3.36E+00	ca	3.35E+00	Y	ASL
	218-01-9	CHRYSENE	16 J	590 J	ug/l	HB-HB-04S	4/26	9.6-1000	5.90E+02				3.00E+00	C	9.21E+00	ca	3.00E+00	Y	ASL
	132-64-9	DIBENZOFURAN	5.9 J	3400	ug/l	HB-HB-04S	14/26	9.6-970	3.40E+03				3.65E+00	N	1.22E+00	nc	1.22E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	2 J	2 J	ug/l	HB-WA-08S	1/26	9.6-1000	2.00E+00				3.65E+02	N	3.65E+02	nc	3.65E+02	N	BSL
	206-44-0	FLUORANTHENE	1.6 J	3200	ug/l	HB-HB-04S	8/26	9.6-1000	3.20E+03				1.46E+02	N	1.46E+02	nc	1.46E+02	Y	ASL

TABLE 2.5a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs*)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
	86-73-7	FLUORENE	3 J	4200	ug/l	HB-HB-04S	15/26	9.6-970	4.20E+03			2.43E+01	N	2.43E+01	nc	2.43E+01	Y	ASL	
	193-39-5	INDENO(1,2,3-CD)PYRENE	110 J	110 J	ug/l	HB-HB-04S	1/26	9.6-1000	1.10E+02			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL	
	91-20-3	NAPHTHALENE	1.5 J	35000	ug/l	HB-HB-04S	24/29	1-10	3.50E+04			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL	
	85-01-8	PHENANTHRENE	4.7 J	8300	ug/l	HB-HB-04S	15/26	9.6-730	8.30E+03			NV	NV	NV		NV	Y	NTX	
	108-95-2	PHENOL	2.8 J	18000	ug/l	HB-HB-02S	21/26	10-10	1.80E+04			1.10E+03	N	1.09E+03	nc	1.09E+03	Y	ASL	
	129-00-0	PYRENE	1.2 J	1900	ug/l	HB-HB-04S	8/26	9.6-1000	1.90E+03			1.83E+01	N	1.83E+01	nc	1.83E+01	Y	ASL	
	VOCs																		
	120-82-1	1,2,4-TRICHLOROBENZENE	230	230	ug/l	HB-WA-03S	1/31	1-1000	2.30E+02			7.00 E+01	6.08E+00	N	7.16E-01	nc	7.16E-01	Y	ASL
	95-63-6	1,2,4-TRIMETHYLBENZENE	2 J	420	ug/l	HB-HB-02S	5/6	0.5-0.5	4.20E+02				1.46E+00	N	1.23E+00	nc	1.23E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	0.19 J	3800	ug/l	HB-WA-03S	12/33	0.5-1000	3.80E+03			6.00 E+02	2.68E+01	N	3.70E+01	nc	2.68E+01	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.6 J	150	ug/l	HB-HB-02S	5/6	0.5-0.5	1.50E+02				NV	NV	1.23E+00	nc	1.23E+00	Y	ASL
	541-73-1	1,3-DICHLOROBENZENE	3.6	62 J	ug/l	HB-WA-03S	4/33	0.5-1000	6.20E+01				1.83E+00	N	1.83E+01	nc	1.83E+00	Y	ASL
	106-46-7	1,4-DICHLOROBENZENE	0.11 J	4500	ug/l	HB-WA-03S	13/33	0.5-1000	4.50E+03			7.50 E+01	2.81E-01	C	5.02E-01	ca	2.81E-01	Y	ASL
	78-93-3	2-BUTANONE	2 J	21	ug/l	HB-HB-03S	9/27	10-2500	2.10E+01				6.97E+02	N	6.97E+02	nc	6.97E+02	N	BSL
	591-78-6	2-HEXANONE	0.3 J	2.6 J	ug/l	HB-HB-03S	3/27	5-1250	2.60E+00				NV	NV	NV		NV	Y	NTX
	106-43-4	4-CHLOROTOLUENE	2 J	2 J	ug/l	HB-HB-06S	1/6	0.5-50	2.00E+00				4.26E+01	N	NV	nc	4.26E+01	N	BSL
	108-10-1	4-METHYL-2-PENTANONE	0.5 J	0.5 J	ug/l	HB-WA-08S	2/27	5-1250	5.00E-01				6.28E+02	N	1.99E+02	nc	1.99E+02	N	BSL
	67-64-1	ACETONE	3.06 J	460 J	ug/l	HB-HB-04S	16/27	10-2500	4.60E+02				5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	98-86-2	ACETOPHENONE	6.7 J	6.7 J	ug/l	HB-HB-03S	1/6	10-110	6.70E+00				6.08E+01	N	NV	nc	6.08E+01	N	BSL
	71-43-2	BENZENE	0.3 J	3900	ug/l	HB-HB-02S	22/27	0.5-50	3.90E+03			5.00 E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	4.7 J	4.7 J	ug/l	HB-HB-03S	2/21	0.5-500	4.70E+00				1.04E+02	N	1.04E+02	nc	1.04E+02	N	BSL
	108-90-7	CHLOROBENZENE	8.7	580	ug/l	HB-WA-03S	10/27	0.5-250	5.80E+02			1.00 E+02	8.96E+00	N	1.06E+01	nc	8.96E+00	Y	ASL
	67-66-3	CHLOROFORM	0.4 J	0.4 J	ug/l	HB-WA-08S	1/27	0.5-250	4.00E-01				1.55E-01	C	1.66E-01	ca	1.55E-01	Y	ASL
	156-59-2	CIS-1,2-DICHLOROETHENE	1 J	1 J	ug/l	HB-HB-06S	1/23	0.5-250	1.00E+00			7.00 E+01	6.08E+00	N	6.08E+00	nc	6.08E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.7 J	350	ug/l	HB-HB-04S	16/27	0.5-100	3.50E+02			7.00 E+02	1.34E+02	N	1.34E+02	nc	1.34E+02	Y	ASL
	98-82-8	ISOPROPYLBENZENE	0.1 J	3	ug/l	HB-HB-06S	3/12	0.5-125	3.00E+00				6.58E+01	N	6.58E+01	nc	6.58E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	5.5 J	5.5 J	ug/l	HB-HB-03S	1/27	2-500	5.50E+00			5.00 E+00	4.10E+00	C	4.28E+00	ca	4.10E+00	Y	ASL
	99-87-6	P-ISOPROPYLTOLUENE	0.8 J	0.8 J	ug/l	HB-HB-06S	1/6	0.5-50	8.00E-01				NV	NV	NV		NV	Y	NTX
	135-98-8	SEC-BUTYLBENZENE	1 J	1 J	ug/l	HB-HB-06S	1/6	0.5-50	1.00E+00				NV	NV	2.43E+01	nc	2.43E+01	N	BSL
	100-42-5	STYRENE	0.3 J	850	ug/l	HB-HB-02S	11/27	0.5-50	8.50E+02			1.00 E+02	1.62E+02	N	1.64E+02	nc	1.62E+02	Y	ASL
	127-18-4	TETRACHLOROETHENE	0.2 J	0.2 J	ug/l	HB-WA-08S	1/27	0.5-250	2.00E-01			5.00 E+00	1.04E-01	C	1.04E-01	ca	1.04E-01	Y	ASL
	108-88-3	TOLUENE	0.39 J	5740	ug/l	HB-HB-02S	19/27	0.5-100	5.74E+03			1.00 E+03	2.27E+02	N	7.23E+01	nc	7.23E+01	Y	ASL
	75-01-4	VINYL CHLORIDE	0.7 J	4.1 J	ug/l	HB-HB-03S	4/27	1-250	4.10E+00			2.00 E+00	1.50E-02	C	1.98E-02	ca	1.50E-02	Y	TOX
	1330-20-7	XYLENES, TOTAL	0.29 J	3380	ug/l	HB-HB-02S	20/27	0.25-150	3.38E+03			1.00 E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	Y	ASL

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.

(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.

(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.

(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.

(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = RBC and PRG values for chromium VI utilized.

b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1254.

d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

e = RBC and PRG value for 4-methylphenol (CAS # 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NUT: Compound is an essential nutrient and not screened in

NV: No Value

PRG: Preliminary Remediation Goals; USEPA, 2004

RBC: Risk Based Concentration; USEPA, October, 2007

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.5b
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SHALLOW GROUND WATER

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-HB-06S	3	13	5/23/2001	0.07	ug/l
Total PCBs	HB-HB-06S	3	13	5/23/2001	0.07	ug/l

Notes:

*Highly chlorinated PCBs were defined as Arorclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Arorclors.

TABLE 2.5c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SHALLOW GROUND WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-02S	5/17/2001	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y		ug/l	2800	2800
HB-HB-02S	5/20/2003	4.01	14.01	95-47-6	O-XYLENE	Y		ug/l	810	
HB-HB-02S	5/20/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	Y		ug/l	2100	
HB-HB-02S	5/20/2003	4.01	14.01	CALCULATED	TOTAL	Y		ug/l		2910
HB-HB-02S	8/22/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	Y		ug/l	2000	
HB-HB-02S	8/22/2003	4.01	14.01	95-47-6	O-XYLENE	Y		ug/l	770	
HB-HB-02S	8/22/2003	4.01	14.01	CALCULATED	TOTAL	Y		ug/l		2770
HB-HB-02S	3/15/2007	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y		ug/l	3380	3380
HB-HB-03S	5/22/2001	4.96	14.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	23	23
HB-HB-03S	5/14/2003	4.96	14.96	XYLENES1314	XYLENES, M & P	Y		ug/l	32	
HB-HB-03S	5/14/2003	4.96	14.96	95-47-6	O-XYLENE	Y		ug/l	11	
HB-HB-03S	5/14/2003	4.96	14.96	CALCULATED	TOTAL	Y		ug/l		43
HB-HB-03S	8/19/2003	4.96	14.96	XYLENES1314	XYLENES, M & P	Y		ug/l	51	
HB-HB-03S	8/19/2003	4.96	14.96	95-47-6	O-XYLENE	Y		ug/l	15	
HB-HB-03S	8/19/2003	4.96	14.96	CALCULATED	TOTAL	Y		ug/l		66
HB-HB-03S	3/8/2007	4.96	14.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	33	33
HB-HB-04S	5/17/2001	8.59	18.59	1330-20-7	XYLENES, TOTAL	Y		ug/l	800	800
HB-HB-04S	5/16/2003	8.59	18.59	XYLENES1314	XYLENES, M & P	Y		ug/l	1400	
HB-HB-04S	5/16/2003	8.59	18.59	95-47-6	O-XYLENE	Y		ug/l	540	
HB-HB-04S	5/16/2003	8.59	18.59	CALCULATED	TOTAL	Y		ug/l		1940
HB-HB-04S	8/20/2003	8.59	18.59	XYLENES1314	XYLENES, M & P	Y		ug/l	2300	
HB-HB-04S	8/20/2003	8.59	18.59	95-47-6	O-XYLENE	Y		ug/l	870	
HB-HB-04S	8/20/2003	8.59	18.59	CALCULATED	TOTAL	Y		ug/l		3170
HB-HB-04S	3/14/2007	8.59	18.59	1330-20-7	XYLENES, TOTAL	Y		ug/l	3220	3220
HB-HB-05S	5/23/2001	7.03	17.03	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-05S	5/20/2003	7.03	17.03	XYLENES1314	XYLENES, M & P	N	U	ug/l	50	
HB-HB-05S	5/20/2003	7.03	17.03	95-47-6	O-XYLENE	N	U	ug/l	50	
HB-HB-05S	5/20/2003	7.03	17.03	CALCULATED	TOTAL	N	U	ug/l		50
HB-HB-05S	8/19/2003	7.03	17.03	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-05S	8/19/2003	7.03	17.03	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-05S	8/19/2003	7.03	17.03	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-05S	3/13/2007	7.03	17.03	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-06S	5/23/2001	3	13	1330-20-7	XYLENES, TOTAL	Y		ug/l	55	55
HB-HB-06S	5/22/2003	3	13	XYLENES1314	XYLENES, M & P	Y		ug/l	35	
HB-HB-06S	5/22/2003	3	13	95-47-6	O-XYLENE	Y		ug/l	34	
HB-HB-06S	5/22/2003	3	13	CALCULATED	TOTAL	Y		ug/l		69
HB-HB-06S	8/25/2003	3	13	XYLENES1314	XYLENES, M & P	Y		ug/l	11	
HB-HB-06S	8/25/2003	3	13	95-47-6	O-XYLENE	Y		ug/l	12	
HB-HB-06S	8/25/2003	3	13	CALCULATED	TOTAL	Y		ug/l		23
HB-HB-06S	3/20/2007	3	13	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2.2	2.2
HB-WA-03S	4/6/1992	3	13	1330-20-7	XYLENES, TOTAL	Y		ug/l	98	98
HB-WA-03S	10/14/1992	3	13	1330-20-7	XYLENES, TOTAL	N	U	ug/l	300	150
HB-WA-08S	1/5/1995	8.95	18.95	1330-20-7	XYLENES, TOTAL	N	U	ug/l	10	5
HB-WA-08S	5/21/2001	8.95	18.95	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	5	5
HB-WA-08S	5/15/2003	8.95	18.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-WA-08S	5/15/2003	8.95	18.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-WA-08S	5/15/2003	8.95	18.95	CALCULATED	TOTAL	N	U	ug/l		5
HB-WA-08S	8/14/2003	8.95	18.95	XYLENES1314	XYLENES, M & P	Y		ug/l	6.4	
HB-WA-08S	8/14/2003	8.95	18.95	95-47-6	O-XYLENE	Y	J	ug/l	3.3	
HB-WA-08S	8/14/2003	8.95	18.95	CALCULATED	TOTAL	Y		ug/l		9.7
HB-WA-08S	3/12/2007	8.95	18.95	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.29	0.29

Notes:

a - Total Xylene value utilized in the risk assessment.

Table 2.6a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTED B/HARBOR BROOK SITE- LAKESHORE AREA SURFACE WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
Harbor Brook Surface Water	METALS																	
	7429-90-5	ALUMINUM	0.0796 J	0.179 J	mg/L	HB-SEEP-1	2/3	0.1-0.1	1.79E-01		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	N	BSL
	7440-36-0	ANTIMONY	0.0028 J	0.0028 J	mg/L	HB-SEEP-1	1/3	0.06-0.06	2.80E-03		6.00E-03	1.46E-03	N	1.46E-03	nc	1.46E-03	Y	ASL
	7440-39-3	BARIUM	0.0842 J	0.108	mg/L	HB-SEEP-1	3/3	-	1.08E-01		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	N	BSL
	7440-41-7	BERYLLIUM	0.00082 J	0.00082 J	mg/L	HB-SEEP-1	1/3	0.005-0.005	8.20E-04		4.00E-03	7.30E-03	N	7.30E-03	nc	7.30E-03	N	BSL
	7440-43-9	CADMIUM	0.0016 J	0.0016 J	mg/L	HB-SEEP-1	1/3	0.005-0.005	1.60E-03		5.00E-03	1.83E-03	N	1.82E-03	nc	1.82E-03	N	BSL
	7440-70-2	CALCIUM	622	707	mg/L	HB-SEEP-1	3/3	-	7.07E+02			NV	NV	NV	N	NUT	N	NUT
	7440-50-8	COPPER	0.0063 J	0.0063 J	mg/L	HB-SEEP-1	1/3	0.02-0.02	6.30E-03		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	N	BSL
	7439-89-6	IRON	0.0741 J	0.231 J	mg/L	HB-SEEP-1	2/3	0.1-0.1	2.31E-01		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	7439-92-1	LEAD	0.0072	0.0279	mg/L	HB-SEEP-1	3/3	-	2.79E-02		1.50E-02	NV	NV	NV	Y	ASL	N	ASL
	7439-95-4	MAGNESIUM	0.271 J	0.271 J	mg/L	HB-SEEP-1	1/3	0.5-0.5	2.71E-01			NV	NV	NV	N	NUT	N	NUT
	7439-96-5	MANGANESE	0.0023 J	0.0023 J	mg/L	HB-SEEP-1	1/3	0.01-0.01	2.30E-03		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	N	BSL
	7439-97-6	MERCURY ^a	0.000334	0.00047	mg/L	HB-SEEP-1	3/4	0.00015 - 0.00015	4.70E-04		2.00E-03	3.65E-04	N	3.65E-04	nc	3.65E-04	Y	ASL
	7440-02-0	NICKEL	0.0045 J	0.0045 J	mg/L	HB-SEEP-1	1/3	0.04-0.04	4.50E-03			7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL
	7440-09-7	POTASSIUM	12.7	17.6	mg/L	HB-SEEP-1	3/3	-	1.76E+01			NV	NV	NV	N	NUT	N	NUT
	7782-49-2	SELENIUM	0.0045 J	0.0045 J	mg/L	HB-SEEP-1	1/3	0.005-0.025	4.50E-03		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL
	7440-23-5	SODIUM	261	357	mg/L	HB-SEEP-1	3/3	-	3.57E+02			NV	NV	NV	N	NUT	N	NUT
	7440-62-2	VANADIUM	0.0019 J	0.0019 J	mg/L	HB-SEEP-1	1/3	0.05-0.05	1.90E-03			3.65E-03	N	3.65E-03	nc	3.65E-03	N	BSL
	7440-66-6	ZINC	0.0067 J	0.0067 J	mg/L	HB-SEEP-1	1/3	0.02-0.02	6.70E-03		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	SVOCs																	
	105-67-9	2,4-DIMETHYLPHENOL	12 J	190	ug/l	HB-SEEP-1	2/3	230-230	1.90E+02			7.30E+01	N	7.30E+01	nc	7.30E+01	Y	ASL
	91-57-6	2-METHYLNAPHTHALENE	220	300	ug/l	HB-SEEP-1	3/3	-	3.00E+02			2.43E+00	N	NV	NV	2.43E+00	Y	ASL
	95-48-7	2-METHYLPHENOL	73 J	170	ug/l	HB-SEEP-1	3/3	-	1.70E+02			1.83E+02	N	1.82E+02	nc	1.82E+02	N	BSL
	34METPH	3&4-METHYLPHENOL ^b	180	280	ug/l	HB-SEEP-1	2/3	230-230	2.80E+02			1.83E+01	N	1.82E+01	nc	1.82E+01	Y	ASL
	83-32-9	ACENAPHTHENE	43 J	49 J	ug/l	HB-SEEP-1	3/3	-	4.90E+01			3.65E+01	N	3.65E+01	nc	3.65E+01	Y	ASL
	208-96-8	ACENAPHTHYLENE	45 J	55 J	ug/l	HB-SEEP-1	3/3	-	5.50E+01			NV	NV	NV	Y	NTX	Y	NTX
	120-12-7	ANTHRACENE	13 J	13 J	ug/l	HB-SEEP-1	1/3	93-230	1.30E+01			1.83E+02	N	1.83E+02	nc	1.83E+02	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	4 J	4 J	ug/l	HB-SEEP-1	1/3	93-230	4.00E+00			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	50-32-8	BENZO(A)PYRENE	2 J	2 J	ug/l	HB-SEEP-1	1/3	93-230	2.00E+00		2.00E-01	3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	3 J	3 J	ug/l	HB-SEEP-1	1/3	93-230	3.00E+00			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	86-74-8	CARBAZOLE	42	56 J	ug/l	HB-SEEP-1	3/3	-	5.60E+01			3.35E+00	C	3.36E+00	ca	3.35E+00	Y	ASL
	218-01-9	CHRYSENE	4 J	4 J	ug/l	HB-SEEP-1	1/3	93-230	4.00E+00			3.00E+00	C	9.21E+00	ca	3.00E+00	Y	ASL
	132-64-9	DIBENZOFURAN	54 J	73 J	ug/l	HB-SEEP-1	3/3	-	7.30E+01			3.65E+00	N	1.22E+00	nc	1.22E+00	Y	ASL
	206-44-0	FLUORANTHENE	25	25	ug/l	HB-SEEP-1	1/3	93-230	2.50E+01			1.46E+02	N	1.46E+02	nc	1.46E+02	N	BSL
	86-73-7	FLUORENE	37 J	42	ug/l	HB-SEEP-1	3/3	-	4.20E+01			2.43E+01	N	2.43E+01	nc	2.43E+01	Y	ASL
	91-20-3	NAPHTHALENE	1100	2100	ug/l	HB-SEEP-1	3/3	-	2.10E+03			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	85-01-8	PHENANTHRENE	65 J	83	ug/l	HB-SEEP-1	3/3	-	8.30E+01			NV	NV	NV	Y	NTX	Y	NTX
	108-95-2	PHENOL	110 J	280	ug/l	HB-SEEP-1	3/3	-	2.80E+02			1.10E+03	N	1.09E+03	nc	1.09E+03	N	BSL
	129-00-0	PYRENE	28	28	ug/l	HB-SEEP-1	1/3	93-230	2.80E+01			1.83E+01	N	1.83E+01	nc	1.83E+01	Y	ASL
	VOCs																	
	95-50-1	1,2-DICHLOROENZENE	2 J	2 J	ug/l	HB-SEEP-1	1/3	93-230	2.00E+00			6.00E+02	N	3.70E+01	nc	2.68E+01	N	BSL
	106-46-7	1,4-DICHLOROENZENE	8 J	8 J	ug/l	HB-SEEP-1	1/3	93-230	8.00E+00			7.50E+01	C	5.02E-01	ca	2.81E-01	Y	ASL
	78-93-3	2-BUTANONE	5 J	7.5 J	ug/l	HB-SEEP-1	2/3	50-50	7.50E+00			6.97E+02	N	6.97E+02	nc	6.97E+02	N	BSL
	67-64-1	ACETONE	32	68	ug/l	HB-SEEP-1	2/3	100-100	6.80E+01			5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	71-43-2	BENZENE	190	200	ug/l	HB-SEEP-1	3/3	-	2.00E+02		5.00E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	100-41-4	ETHYLBENZENE	34	45	ug/l	HB-SEEP-1	3/3	-	4.50E+01			7.00E+02	N	1.34E+02	nc	1.34E+02	N	BSL
	100-42-5	STYRENE	67	67	ug/l	HB-SEEP-1	1/3	10-25	6.70E+01			1.00E+02	N	1.64E+02	nc	1.62E+02	N	BSL
	108-88-3	TOLUENE	320	410	ug/l	HB-SEEP-1	2/2	-	4.10E+02			1.00E+03	N	7.23E+01	nc	7.23E+01	Y	ASL
	1330-20-7	XYLENES, TOTAL	450	550	ug/l	HB-SEEP-1	3/3	-	5.50E+02			1.00E+04	N	2.06E+01	nc	2.06E+01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) N/A - No background screening performed.
 - (4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
a = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
b = RBC and PRG values for 4-methylphenol (CAS # 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.6b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - LAKESHORE AREA SURFACE WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-SEEP-1	4/5/1999	---	---	1330-20-7	XYLENES, TOTAL	Y		ug/l	470	470
HB-SEEP-1	12/11/2002	---	---	95-47-6	O-XYLENE	Y		ug/l	150	
HB-SEEP-1	12/11/2002	---	---	XYLENES1314	XYLENES, M & P	Y		ug/l	400	
HB-SEEP-1	12/11/2002	---	---	CALCULATED	TOTAL	Y		ug/l		550
HB-SEEP-1	6/12/2003	---	---	95-47-6	O-XYLENE	Y		ug/l	130	
HB-SEEP-1	6/12/2003	---	---	XYLENES1314	XYLENES, M & P	Y		ug/l	320	
HB-SEEP-1	6/12/2003	---	---	CALCULATED	TOTAL	Y		ug/l		450

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.7a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
Penn-Can Property - Surface Soil	METALS																		
	7429-90-5	ALUMINUM	1080	9220	mg/Kg	HB-HB-12D	23/23	-	9.22E+03			7.82E+03	N	7.61E+03	nc	7.61E+03	Y	ASL	
	7440-36-0	ANTIMONY	0.19 J	4.9 J	mg/Kg	HB-HB-10	11/23	6.5-9.3	4.90E+00			3.13E+00	N	3.13E+00	nc	3.13E+00	Y	ASL	
	7440-38-2	ARSENIC	2.5	34.4	mg/Kg	HB-PCSS-1	23/23	-	3.44E+01		1.60E+01	4.26E-01	C	3.90E-02	nc	3.90E-02	Y	TOX	
	7440-39-3	BARIUM	11.9 J	147	mg/Kg	HB-GP-39	23/23	-	1.47E+02		4.00E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL	
	7440-41-7	BERYLLIUM	0.2 J	1.4	mg/Kg	HB-GP-39	11/23	0.54-0.77	1.40E+00		7.20E+01	1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL	
	7440-43-9	CADMIUM	0.059 J	1.4	mg/Kg	HB-HB-10	7/23	0.028-0.73	1.40E+00		4.30E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	N	BSL	
	7440-70-2	CALCIUM	5880	241000	mg/Kg	HB-GP-37	23/23	-	2.41E+05			NV	NV	NV	nc	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	4.3	93.4	mg/Kg	HB-GP-39	23/23	-	9.34E+01		1.10E+02	2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX	
	7440-48-4	COBALT	2.8 J	15.6	mg/Kg	HB-GP-39	19/23	5.6-7.7	1.56E+01			NV	9.03E+01	nc	9.03E+01	N	BSL		
	7440-50-8	COPPER	5.5 J	81.8 J	mg/Kg	HB-PCSS-1	23/23	-	8.18E+01		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL	
	57-12-5	CYANIDE	0.66	1.53	mg/Kg	HB-PSD-02	3/23	0.56-1.54	1.53E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL	
	7439-89-6	IRON	3300	30000	mg/Kg	HB-GP-39	23/23	-	3.00E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	11.1	263	mg/Kg	HB-HB-10	23/23	-	2.63E+02			NV	4.00E+02	nc	4.00E+02	N	BSL		
	7439-95-4	MAGNESIUM	811	44400	mg/Kg	HB-HB-17D	23/23	-	4.44E+04			NV	NV	NV	nc	NV	N	NUT	
	7439-96-5	MANGANESE	104	402	mg/Kg	HB-PSD-02	23/23	-	4.02E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL	
	7439-97-6	MERCURY ^b	0.04 J	7.9	mg/kg	HB-PSD-02	23/23	-	7.90E+00			7.82E-01	N	6.11E-01	nc	6.11E-01	Y	ASL	
	7440-02-0	NICKEL	9.5	51.2	mg/Kg	HB-GP-39	23/23	-	5.12E+01		3.10E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL	
	7440-09-7	POTASSIUM	378	1680	mg/Kg	HB-GP-35	23/23	-	1.68E+03			NV	NV	NV	nc	NV	N	NUT	
	7782-49-2	SELENIUM	0.34 J	3.6	mg/Kg	HB-GP-39	11/23	0.24-2.82	3.60E+00		1.80E+02	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-22-4	SILVER	0.15 J	5.3	mg/Kg	HB-HB-10	2/23	0.082-1.5	5.30E+00		1.80E+02	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-23-5	SODIUM	60.7	1630	mg/Kg	HB-PSD-01	23/23	-	1.63E+03			NV	NV	NV	nc	NV	N	NUT	
	7440-28-0	THALLIUM	1 J	1 J	mg/Kg	HB-HB-12D	1/23	0.41-1.5	1.00E+00			5.48E-01	N	5.16E-01	nc	5.16E-01	Y	ASL	
	7440-62-2	VANADIUM	10.4	44.1	mg/Kg	HB-GP-39	23/23	-	4.41E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	14.3	399	mg/Kg	HB-HB-10	23/23	-	3.99E+02		1.00E+04	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL	
	PCBs																		
			HIGHLY CHLORINATED PCBs ^c	0.02	6	mg/kg	HB-HB-10	11/23	0.02-0.39	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
			TOTAL PCBs ^d	0.02	6	mg/kg	HB-HB-10	11/23	0.02-0.39	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
	PESTICIDES																		
	72-54-8	4,4'-DDD	0.01 J	0.2 J	mg/kg	HB-HB-10	3/23	0.006-0.4	2.00E-01		1.30E+01	2.66E+00	C	2.44E+00	ca	2.44E+00	N	BSL	
	72-55-9	4,4'-DDE	0.01 J	0.01 J	mg/kg	HB-GP-35, HB-GP-39	2/23	0.0074-0.5	1.00E-02		8.90E+00	1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL	
	50-29-3	4,4'-DDT	0.04	0.7	mg/kg	HB-HB-10	2/23	0.006-0.4	7.00E-01		7.90E+00	1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL	
	1031-07-8	ENDOSULFAN SULFATE ^e	0.13 J	0.13 J	mg/kg	HB-PSD-02	2/23	0.006-0.5	1.30E-01		2.40E+01	4.69E+01	N	3.67E+01	nc	3.67E+01	N	BSL	
	7421-93-4	ENDRIN ALDEHYDE ^f	0.046	0.15 J	mg/kg	HB-HB-17D	2/23	0.006-0.5	1.50E-01			2.35E+00	N	1.83E+00	nc	1.83E+00	N	BSL	
	SVOCs																		
	105-67-9	2,4-DIMETHYLPHENOL	0.043 J	0.043 J	mg/kg	HB-HB-15	1/23	0.4-19	4.30E-02			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL	
	91-57-6	2-METHYLNAPHTHALENE	0.3 J	10 J	mg/kg	HB-HB-17D	17/23	0.4-7.7	1.00E+01			3.13E+01	N	NV	nc	3.13E+01	N	BSL	
	34METPH	3&4-METHYLPHENOL ^g	0.044 J	0.044 J	mg/kg	HB-HB-15	1/23	0.4-19	4.40E-02			3.91E+01	N	3.06E+01	nc	3.06E+01	N	BSL	
	83-32-9	ACENAPHTHENE	0.22 J	17 J	mg/kg	HB-HB-17D	16/23	0.4-19	1.70E+01		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL	
	208-96-8	ACENAPHTHYLENE	0.08 J	30	mg/kg	HB-PSD-02	19/23	1.9-4	3.00E+01		1.00E+02	NV	NV	NV	nc	NV	Y	NTX	
	120-12-7	ANTHRACENE	0.18 J	61	mg/kg	HB-HB-17D	21/23	1.9-3.8	6.10E+01		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL	
	56-55-3	BENZ(A)ANTHRACENE	0.44 J	120	mg/kg	HB-HB-17D	23/23	-	1.20E+02		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	50-32-8	BENZO(A)PYRENE	0.48 J	100	mg/kg	HB-HB-17D	23/23	-	1.00E+02		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL	
	205-99-2	BENZO(B)FLUORANTHENE	0.37 J	81	mg/kg	HB-HB-17D	23/23	-	8.10E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	191-24-2	BENZO(G,H,I)PERYLENE	0.08 J	69	mg/kg	HB-PSD-02	22/23	3.8-3.8	6.90E+01		1.00E+02	NV	NV	NV	nc	NV	Y	NTX	
	207-08-9	BENZO(K)FLUORANTHENE	0.33 J	94	mg/kg	HB-HB-17D	22/23	3.8-3.8	9.40E+01		3.90E+00	2.20E+00	C	6.21E+00	ca	2.20E+00	Y	ASL	
	86-74-8	CARBAZOLE	0.14 J	17 J	mg/kg	HB-HB-17D	16/23	0.4-19	1.70E+01			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL	

TABLE 2.7a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	218-01-9	CHRYSENE	0.49 J	110	mg/kg	HB-HB-17D	23/23	-	1.10E+02		3.90E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.11 J	22	mg/kg	HB-PSD-02	20/23	1.9-19	2.20E+01		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.22 J	19 J	mg/kg	HB-HB-17D	17/23	0.4-4	1.90E+01		5.90E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.044 J	0.044 J	mg/kg	HB-HB-15	1/23	0.4-19	4.40E-02			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	206-44-0	FLUORANTHENE	0.63 J	310	mg/kg	HB-HB-17D	23/23	-	3.10E+02		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	Y	ASL
	86-73-7	FLUORENE	0.12 J	34	mg/kg	HB-HB-17D	16/23	0.4-4	3.40E+01		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	87-68-3	HEXACHLOROBUTADIENE	0.001 J	0.001 J	mg/kg	HB-GP-35	1/34	0.006-19	1.00E-03			8.19E+00	C	6.24E-01	nc	6.24E-01	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.29 J	64	mg/kg	HB-PSD-02	22/23	3.8-3.8	6.40E+01		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.002 J	23	mg/kg	HB-PSD-02	24/34	0.006-3.8	2.30E+01		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	85-01-8	PHENANTHRENE	0.29 J	210	mg/kg	HB-HB-17D	23/23	-	2.10E+02		1.00E+02	NV	NV	NV	Y	NTX		
	108-95-2	PHENOL	0.84 J	0.84 J	mg/kg	HB-HB-12D	1/23	0.4-19	8.40E-01		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.64 J	180	mg/kg	HB-HB-17D	23/23	-	1.80E+02		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																	
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.0008 J	0.13 J	mg/kg	HB-GP-34	5/11	0.003-0.005	1.30E-01		5.20E+01	NV	5.16E+00	nc	5.16E+00	N	BSL	
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.001 J	0.002 J	mg/kg	HB-GP-36, HB-HB-12D	3/11	0.003-0.3	2.00E-03		5.20E+01	NV	2.13E+00	nc	2.13E+00	N	BSL	
	106-46-7	1,4-DICHLOROBENZENE	0.059 J	0.58 J	mg/kg	HB-GP-39	3/34	0.003-19	5.80E-01		1.30E+01	2.66E+01	C	3.45E+00	ca	3.45E+00	N	BSL
	78-93-3	2-BUTANONE	0.0077 J	0.011 J	mg/kg	HB-GP-38	2/23	0.01-1.2	1.10E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.14 J	0.14 J	mg/kg	HB-PCSS-1	1/23	0.011-1.2	1.40E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.0008 J	0.052	mg/kg	HB-GP-35	6/23	0.003-0.3	5.20E-02		4.80E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.0021 J	0.0021 J	mg/kg	HB-PCSS-1	1/12	0.01-0.018	2.10E-03			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL
	100-41-4	ETHYLBENZENE	0.001 J	0.0032 J	mg/kg	HB-HB-17D	2/23	0.003-0.3	3.20E-03		4.10E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.002 J	0.002 J	mg/kg	HB-GP-36	1/11	0.003-0.3	2.00E-03			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.023	0.09	mg/kg	HB-GP-38	3/23	0.005-0.6	9.00E-02		1.00E+02	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.0009 J	0.009 J	mg/kg	HB-GP-38	3/11	0.003-0.3	9.00E-03			NV	NV	NV	Y	NTX		
	127-18-4	TETRACHLOROETHENE	0.0007 J	0.001 J	mg/kg	HB-GP-35	4/23	0.003-0.3	1.00E-03		1.90E+01	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL
	108-88-3	TOLUENE	0.0008 J	0.014	mg/kg	HB-HB-17D	5/23	0.0028-0.3	1.40E-02		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	0.0006 J	0.036	mg/kg	HB-HB-17D	8/23	0.0015-0.15	3.60E-02		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = RBC and PRG values for Endosulfan (CAS# 115297) utilized.
f = RBC and PRG values for Endrin (CAS# 72208) utilized
g = RBC and PRG values for 4-methylphenol (CAS# 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals; USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.7b
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-GP-32	0	0.17	3/13/2001	0.02	mg/kg
Highly Chlorinated PCBs	HB-GP-33	0	0.17	3/12/2001	0.03	mg/kg
Highly Chlorinated PCBs	HB-GP-36	0	0.17	3/9/2001	0.03	mg/kg
Highly Chlorinated PCBs	HB-GP-38	0	0.17	3/14/2001	0.07	mg/kg
Highly Chlorinated PCBs	HB-GP-39	0	0.17	4/6/2001	0.1	mg/kg
Highly Chlorinated PCBs	HB-HB-10	0	0.17	3/6/2001	6	mg/kg
Highly Chlorinated PCBs	HB-HB-12D	0	0.17	3/7/2001	0.06	mg/kg
Highly Chlorinated PCBs	HB-HB-15	0	0.17	3/14/2001	0.05	mg/kg
Highly Chlorinated PCBs	HB-PCSS-1	0	0.5	12/5/2002	0.14	mg/kg
Highly Chlorinated PCBs	HB-PSD-01	0	0.5	10/9/2003	0.29	mg/kg
Highly Chlorinated PCBs	HB-PSD-01	0.5	1	10/9/2003	0.28	mg/kg
Total PCBs	HB-GP-32	0	0.17	3/13/2001	0.02	mg/kg
Total PCBs	HB-GP-33	0	0.17	3/12/2001	0.03	mg/kg
Total PCBs	HB-GP-36	0	0.17	3/9/2001	0.03	mg/kg
Total PCBs	HB-GP-38	0	0.17	3/14/2001	0.07	mg/kg
Total PCBs	HB-GP-39	0	0.17	4/6/2001	0.1	mg/kg
Total PCBs	HB-HB-10	0	0.17	3/6/2001	6	mg/kg
Total PCBs	HB-HB-12D	0	0.17	3/7/2001	0.06	mg/kg
Total PCBs	HB-HB-15	0	0.17	3/14/2001	0.05	mg/kg
Total PCBs	HB-PCSS-1	0	0.5	12/5/2002	0.14	mg/kg
Total PCBs	HB-PSD-01	0	0.5	10/9/2003	0.29	mg/kg
Total PCBs	HB-PSD-01	0.5	1	10/9/2003	0.28	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.7c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-GP-32	3/13/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-33	3/12/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0006	0.0006
HB-GP-34	3/12/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.3	0.15
HB-GP-35	3/12/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0007	0.0007
HB-GP-36	3/9/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.007	0.007
HB-GP-37	3/9/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-38	3/14/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0007	0.0007
HB-GP-39	4/6/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-HB-10	3/6/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-12D	3/7/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.005	0.005
HB-HB-15	3/14/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-17D	1/14/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0092	
HB-HB-17D	1/14/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0092	
HB-HB-17D	1/14/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0092
HB-HB-17D	1/14/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.023	
HB-HB-17D	1/14/2003	0.5	1	95-47-6	O-XYLENE	Y		mg/kg	0.013	
HB-HB-17D	1/14/2003	0.5	1	CALCULATED	TOTAL	Y		mg/kg		0.036
HB-PSD-02	10/9/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.0071	
HB-PSD-02	10/9/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.0071	
HB-PSD-02	10/9/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.0071
HB-PSD-02	10/9/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0073	
HB-PSD-02	10/9/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0073	
HB-PSD-02	10/9/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0073
HB-PSD-01	10/9/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0071	
HB-PSD-01	10/9/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0071	
HB-PSD-01	10/9/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0071
HB-PSD-01	10/9/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0032	
HB-PSD-01	10/9/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0077	
HB-PSD-01	10/9/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0032
HB-PCSS-3	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0057	
HB-PCSS-3	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0057	
HB-PCSS-3	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0057
HB-PCSS-3	12/5/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0056	
HB-PCSS-3	12/5/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0056	
HB-PCSS-3	12/5/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0056
HB-PCSS-2	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0051	
HB-PCSS-2	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0051	
HB-PCSS-2	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0051
HB-PCSS-2	12/5/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.005	
HB-PCSS-2	12/5/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.005	
HB-PCSS-2	12/5/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.005
HB-PCSS-1	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0074	
HB-PCSS-1	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0074	
HB-PCSS-1	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0074
HB-PCSS-1	12/5/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.004	
HB-PCSS-1	12/5/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.01	
HB-PCSS-1	12/5/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.004

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.8a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
Penn-Can Property - Subsurface Soil	METALS															
	7429-90-5	ALUMINUM	1080	12500	mg/Kg	HB-TP-35	29/29	-	9.22E+03			7.82E+03	N	7.61E+03	nc	7.61E+03 Y ASL
	7440-36-0	ANTIMONY	0.19 J	4.9 J	mg/Kg	HB-HB-10	14/29	0.19-9.3	4.90E+00			3.13E+00	N	3.13E+00	nc	3.13E+00 Y ASL
	7440-38-2	ARSENIC	2.5	103	mg/Kg	HB-HB-111	29/29	-	3.44E+01		1.60E+01	4.26E+01	C	3.90E+01	ca	3.90E+01 Y TOX
	7440-39-3	BARIUM	4.8 J	147	mg/Kg	HB-GP-39	29/29	-	1.47E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02 N BSL
	7440-41-7	BERYLLIUM	0.2 J	1.4	mg/Kg	HB-GP-39	17/29	0.54-0.77	1.40E+00		1.40E+01	1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL
	7440-43-9	CADMIUM	0.059 J	10.7	mg/Kg	HB-HB-111	11/29	0.028-0.73	1.40E+00		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00 N BSL
	7440-70-2	CALCIUM	5880	306000 J	mg/Kg	HB-TP-39	29/29	-	3.06E+05			NV	NV	NV	N	NUT
	7440-47-3	CHROMIUM ^a	4.3	93.4	mg/Kg	HB-GP-39	29/29	-	9.34E+01			2.35E+01	N	3.01E+00	nc	3.01E+00 Y TOX
	7440-48-4	COBALT	2.5 J	15.6	mg/Kg	HB-GP-39	25/29	5.6-7.7	1.56E+01			NV	N	9.03E+01	nc	9.03E+01 N BSL
	7440-50-8	COPPER	5.5 J	98.7 J	mg/Kg	HB-TP-36	29/29	-	9.87E+01		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02 N BSL
	57-12-5	CYANIDE	0.66	30.9 J	mg/Kg	HB-TP-39	8/29	0.56-1.54	3.09E+01			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	7439-89-6	IRON	3300	30000	mg/Kg	HB-GP-39	29/29	-	3.00E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL
	7439-92-1	LEAD	10.6 J	348	mg/Kg	HB-HB-111	29/29	-	3.48E+02			NV	N	4.00E+02	nc	4.00E+02 N BSL
	7439-95-4	MAGNESIUM	811	44400	mg/Kg	HB-HB-17D	29/29	-	4.44E+04			NV	N	NV	N	NUT
	7439-96-5	MANGANESE	104	470	mg/Kg	HB-TP-35	29/29	-	4.70E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL
	7439-97-6	MERCURY ^b	0.04 J	7.9	mg/Kg	HB-PSD-02	28/29	0.037-0.037	7.90E+00			7.82E-01	N	6.11E-01	nc	6.11E-01 Y ASL
	7440-02-0	NICKEL	9.5	51.2	mg/Kg	HB-GP-39	29/29	-	5.12E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL
	7440-09-7	POTASSIUM	378	2780	mg/Kg	HB-TP-35	28/29	16.7-16.7	2.78E+03			NV	N	NV	N	NUT
	7782-49-2	SELENIUM	0.34 J	6.2	mg/Kg	HB-HB-111	17/29	0.24-2.82	6.20E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-22-4	SILVER	0.095 J	5.3	mg/Kg	HB-HB-10	3/29	0.082-1.5	5.30E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-23-5	SODIUM	60.7	2930	mg/Kg	HB-HB-111	29/29	-	2.93E+03			NV	N	NV	N	NUT
	7440-28-0	THALLIUM	1 J	38.5	mg/Kg	HB-HB-111	2/29	0.41-1.5	3.85E+01			5.48E-01	N	5.16E-01	nc	5.16E-01 Y ASL
	7440-62-2	VANADIUM	10.4	44.1	mg/Kg	HB-GP-39	29/29	-	4.41E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL
	7440-66-6	ZINC	14.3	399	mg/Kg	HB-HB-10	29/29	-	3.99E+02		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL
	PCBs															
		HIGHLY CHLORINATED PCBs ^c	0.02	6	mg/kg	HB-HB-10	13/29	0.02-0.39	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL
		TOTAL PCBs ^d	0.02	6	mg/kg	HB-HB-10	13/29	0.02-0.39	6.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL
	PESTICIDES															
	72-54-8	4,4'-DDD	0.002 J	0.2 J	mg/kg	HB-HB-10	6/29	0.006-4	2.00E-01		2.60E+00	2.66E+00	C	2.44E+00	ca	2.44E+00 N BSL
	72-55-9	4,4'-DDE	0.001 J	0.01 J	mg/kg	HB-TP-37, HB-GP-39	4/29	0.006-4	1.00E-02		1.80E+00	1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	50-29-3	4,4'-DDT	0.002 J	0.7	mg/kg	HB-HB-10	3/29	0.006-4	7.00E-01		1.70E+00	1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	1031-07-8	ENDOSULFAN SULFATE ^e	0.13 J	0.13 J	mg/kg	HB-PSD-02	2/29	0.004-4	1.30E-01		4.80E+00	4.69E+01	N	3.67E+02	ca	4.69E+01 N BSL
	7421-93-4	ENDRIN ALDEHYDE ^f	0.046	0.15 J	mg/kg	HB-HB-17D	2/29	0.004-4	1.50E-01			2.35E+00	N	1.83E+01	ca	2.35E+00 N BSL
	SVOCs															
	105-67-9	2,4-DIMETHYLPHENOL	0.043 J	190 J	mg/kg	HB-HB-111	2/29	0.39-19	1.90E+02			1.56E+02	N	1.22E+02	nc	1.22E+02 Y ASL
	91-57-6	2-METHYLNAPHTHALENE	0.05 J	3000	mg/kg	HB-HB-111	22/29	0.4-7.7	3.00E+03			3.13E+01	N	NV	nc	3.13E+01 Y ASL
	34METPH	3&4-METHYLPHENOL ^g	0.044 J	500	mg/kg	HB-HB-111	3/29	0.4-19	5.00E+02			3.91E+01	N	3.06E+01	nc	3.06E+01 Y ASL
	83-32-9	ACENAPHTHENE	0.059 J	1400	mg/kg	HB-HB-111	19/29	0.4-19	1.40E+03		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02 Y ASL
	208-96-8	ACENAPHTHYLENE	0.044 J	100 J	mg/kg	HB-HB-111	23/29	0.41-4	1.00E+02		1.00E+02	NV	NV	NV	Y	NTX
	120-12-7	ANTHRACENE	0.082 J	3000	mg/kg	HB-HB-111	25/29	0.41-3.8	3.00E+03		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03 Y ASL
	56-55-3	BENZ(A)ANTHRACENE	0.073 J	2000	mg/kg	HB-HB-111	28/29	1.2-1.2	2.00E+03		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	50-32-8	BENZO(A)PYRENE	0.07 J	1400	mg/kg	HB-HB-111	28/29	1.2-1.2	1.40E+03		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL

TABLE 2.8a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	205-99-2	BENZO(B)FLUORANTHENE	0.11 J	1900	mg/kg	HB-HB-111	28/29	1.2-1.2	1.90E+03		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.05 J	380	mg/kg	HB-HB-111	27/29	1.2-3.8	3.80E+02		1.00E+02	NV	NV	NV	Y	NTX		
	207-08-9	BENZO(K)FLUORANTHENE	0.27 J	740	mg/kg	HB-HB-111	26/29	0.41-3.8	7.40E+02		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00	Y	ASL
	86-74-8	CARBAZOLE	0.093 J	1500	mg/kg	HB-HB-111	19/29	0.4-19	1.50E+03			3.19E+01	C	2.43E+01	ca	2.43E+01	Y	ASL
	218-01-9	CHRYSENE	0.088 J	1700	mg/kg	HB-HB-111	28/29	1.2-1.2	1.70E+03		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.09 J	130 J	mg/kg	HB-HB-111	24/29	0.41-19	1.30E+02		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.13 J	1800	mg/kg	HB-HB-111	20/29	0.39-4	1.80E+03		1.40E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.044 J	0.044 J	mg/kg	HB-HB-15	1/29	0.39-370	4.40E-02			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	206-44-0	FLUORANTHENE	0.14 J	5800	mg/kg	HB-HB-111	28/29	1.2-1.2	5.80E+03		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	Y	ASL
	86-73-7	FLUORENE	0.077 J	2700	mg/kg	HB-HB-111	19/29	0.4-4	2.70E+03		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	Y	ASL
	87-68-3	HEXACHLOROBUTADIENE	0.001 J	0.001 J	mg/kg	HB-GP-35	1/46	0.006-370	1.00E-03			8.19E+00	C	6.24E-01	nc	6.24E-01	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.045 J	410	mg/kg	HB-HB-111	27/29	1.2-3.8	4.10E+02		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.002 J	14000	mg/kg	HB-HB-111	32/46	0.006-3.8	1.40E+04		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	85-01-8	PHENANTHRENE	0.15 J	9300	mg/kg	HB-HB-111	28/29	1.2-1.2	9.30E+03		1.00E+02	NV	NV	NV	Y	NTX		
	108-95-2	PHENOL	0.84 J	360 J	mg/kg	HB-HB-111	2/29	0.39-19	3.60E+02		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.14 J	4700	mg/kg	HB-HB-111	28/29	1.2-1.2	4.70E+03		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	Y	ASL
	VOCs																	
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.0008 J	0.13 J	mg/kg	HB-GP-34	6/17	0.003-55	1.30E-01		4.70E+01	NV		5.16E+00	nc	5.16E+00	N	BSL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.001 J	0.002 J	mg/kg	HB-GP-36, HB-HB-12D	4/17	0.003-55	2.00E-03		4.70E+01	NV		2.13E+00	nc	2.13E+00	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.002 J	0.58 J	mg/kg	HB-GP-39	5/46	0.003-370	5.80E-01		9.80E+00	2.66E+01	C	3.45E+00	ca	3.45E+00	N	BSL
	78-93-3	2-BUTANONE	0.0077 J	0.011 J	mg/kg	HB-GP-38	2/29	0.01-220	1.10E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.022 J	0.14 J	mg/kg	HB-PCSS-1	6/29	0.011-220	1.40E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.0008 J	0.052	mg/kg	HB-GP-35	6/29	0.003-55	5.20E-02		2.90E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.0021 J	0.0021 J	mg/kg	HB-PCSS-1	1/12	0.01-0.018	2.10E-03			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL
	100-41-4	ETHYLBENZENE	0.001 J	0.0032 J	mg/kg	HB-HB-17D	3/29	0.003-55	3.20E-03		3.00E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.002 J	0.002 J	mg/kg	HB-GP-36	1/17	0.003-55	2.00E-03			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.012	0.09	mg/kg	HB-GP-38	5/29	0.005-110	9.00E-02		5.10E+01	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.0009 J	0.009 J	mg/kg	HB-GP-38	5/17	0.003-55	9.00E-03			NV	NV	NV	Y	NTX		
	127-18-4	TETRACHLOROETHENE	0.0007 J	0.001 J	mg/kg	HB-GP-35	4/29	0.003-55	1.00E-03		5.50E+00	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL
	108-88-3	TOLUENE	0.0007 J	0.014	mg/kg	HB-HB-17D	6/29	0.0028-55	1.40E-02		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	0.0006 J	0.036	mg/kg	HB-HB-17D	9/29	0.0015-27.5	3.60E-02		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value.
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = RBC and PRG values for Endosulfan (CAS # 1115297) utilized.
f = RBC and PRG values for Endrin (CAS # 72208) utilized.
g = RBC and PRG values for 4-methylphenol (CAS # 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals; USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.8b
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-GP-32	0	0.17	3/13/2001	0.02	mg/kg
Highly Chlorinated PCBs	HB-GP-33	0	0.17	3/12/2001	0.03	mg/kg
Highly Chlorinated PCBs	HB-GP-36	0	0.17	3/9/2001	0.03	mg/kg
Highly Chlorinated PCBs	HB-GP-38	0	0.17	3/14/2001	0.07	mg/kg
Highly Chlorinated PCBs	HB-GP-39	0	0.17	4/6/2001	0.1	mg/kg
Highly Chlorinated PCBs	HB-HB-10	0	0.17	3/6/2001	6	mg/kg
Highly Chlorinated PCBs	HB-HB-12D	0	0.17	3/7/2001	0.06	mg/kg
Highly Chlorinated PCBs	HB-HB-15	0	0.17	3/14/2001	0.05	mg/kg
Highly Chlorinated PCBs	HB-PCSS-1	0	0.5	12/5/2002	0.14	mg/kg
Highly Chlorinated PCBs	HB-PSD-01	0	0.5	10/9/2003	0.29	mg/kg
Highly Chlorinated PCBs	HB-PSD-01	0.5	1	10/9/2003	0.28	mg/kg
Highly Chlorinated PCBs	HB-TP-36	6	6	3/7/2001	0.1	mg/kg
Highly Chlorinated PCBs	HB-TP-37	7	7	3/7/2001	0.04	mg/kg
Total PCBs	HB-GP-32	0	0.17	3/13/2001	0.02	mg/kg
Total PCBs	HB-GP-36	0	0.17	3/9/2001	0.03	mg/kg
Total PCBs	HB-GP-33	0	0.17	3/12/2001	0.03	mg/kg
Total PCBs	HB-TP-37	7	7	3/7/2001	0.04	mg/kg
Total PCBs	HB-HB-15	0	0.17	3/14/2001	0.05	mg/kg
Total PCBs	HB-HB-12D	0	0.17	3/7/2001	0.06	mg/kg
Total PCBs	HB-GP-38	0	0.17	3/14/2001	0.07	mg/kg
Total PCBs	HB-TP-36	6	6	3/7/2001	0.1	mg/kg
Total PCBs	HB-GP-39	0	0.17	4/6/2001	0.1	mg/kg
Total PCBs	HB-PCSS-1	0	0.5	12/5/2002	0.14	mg/kg
Total PCBs	HB-PSD-01	0.5	1	10/9/2003	0.28	mg/kg
Total PCBs	HB-PSD-01	0	0.5	10/9/2003	0.29	mg/kg
Total PCBs	HB-HB-10	0	0.17	3/6/2001	6	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.8c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-GP-32	3/13/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-33	3/12/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0006	0.0006
HB-GP-34	3/12/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.3	0.15
HB-GP-35	3/12/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0007	0.0007
HB-GP-36	3/9/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.007	0.007
HB-GP-37	3/9/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-38	3/14/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0007	0.0007
HB-GP-39	4/6/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.005	0.0025
HB-HB-10	3/6/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-111	3/8/2001	4	6	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	55	27.5
HB-HB-12D	3/7/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.005	0.005
HB-HB-15	3/14/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-17D	1/14/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0092	
HB-HB-17D	1/14/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0092	
HB-HB-17D	1/14/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0092
HB-HB-17D	1/14/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.023	
HB-HB-17D	1/14/2003	0.5	1	95-47-6	O-XYLENE	Y		mg/kg	0.013	
HB-HB-17D	1/14/2003	0.5	1	CALCULATED	TOTAL	Y		mg/kg		0.036
HB-PSD-02	10/9/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.0071	
HB-PSD-02	10/9/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.0071	
HB-PSD-02	10/9/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.0071
HB-PSD-02	10/9/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0073	
HB-PSD-02	10/9/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0073	
HB-PSD-02	10/9/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0073
HB-PSD-01	10/9/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0071	
HB-PSD-01	10/9/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0071	
HB-PSD-01	10/9/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0071
HB-PSD-01	10/9/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0032	
HB-PSD-01	10/9/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0077	
HB-PSD-01	10/9/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0032
HB-PCSS-3	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0057	
HB-PCSS-3	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0057	
HB-PCSS-3	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0057
HB-PCSS-3	12/5/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0056	
HB-PCSS-3	12/5/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0056	
HB-PCSS-3	12/5/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0056
HB-PCSS-2	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0051	
HB-PCSS-2	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0051	
HB-PCSS-2	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0051
HB-PCSS-2	12/5/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.005	
HB-PCSS-2	12/5/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.005	
HB-PCSS-2	12/5/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.005
HB-PCSS-1	12/5/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0074	
HB-PCSS-1	12/5/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0074	
HB-PCSS-1	12/5/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0074
HB-PCSS-1	12/5/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.004	
HB-PCSS-1	12/5/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.01	
HB-PCSS-1	12/5/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.004
HB-TP-35	3/7/2001	9	10	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-TP-36	3/7/2001	6	6	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.003	0.003
HB-TP-37	3/7/2001	7	7	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-TP-38	3/8/2001	7	7	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.005	0.0025
HB-TP-39	3/8/2001	8	8	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.009	0.0045

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.9a
OCCURRENCE, DISTRIBUTION, AND ACTION DECISION FOR CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SUBSLAB VAPOR
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Ambient Air
Exposure Medium: Subslab Vapor

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Target Shallow Gas Concentration Corresponding to Target Residential Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor=0.1 (ug/m3) (4)	Screening Toxicity Value	Decision	Rationale for Decision (5)
Penn-Can Property - Subslab Vapor	SVOCs													
	91-20-3	NAPHTHALENE	62 J	62 J	ug/m ³	HB-HW-SS-04	1/4	10-10	6.20E+01		7.2E+00	c	7.2E+00	Sample Indoor Air ASL
	VOCs													
	71-55-6	1,1,1-TRICHLOROETHANE	13 J	13 J	ug/m ³	HB-HW-SS-02	1/4	0.15-5	1.30E+01		5.2E+04	n	5.2E+04	No Action BSL
	95-63-6	1,2,4-TRIMETHYLBENZENE	8.9	12 J	ug/m ³	HB-HW-SS-03	3/4	5-5	1.20E+01		7.3E+01	n	7.3E+01	No Action BSL
	78-93-3	2-BUTANONE	6.2	6.2	ug/m ³	HB-HW-SS-04	1/4	10-10	6.20E+00		5.2E+04	n	5.2E+04	No Action BSL
	107-06-2	1,2-DICHLOROETHANE	1.2	1.2	ug/m ³	HB-HW-SS-04	1/4	5-5	1.20E+00		9.4E+00	c	9.4E+00	No Action BSL
	108-67-8	1,3,5-TRIMETHYLBENZENE	3.1	5.3 J	ug/m ³	HB-HW-SS-03	2/4	5-5	5.30E+00		6.3E+01	n	6.3E+01	No Action BSL
	108-10-1	4-METHYL-2-PENTANONE	1 J	73	ug/m ³	HB-HW-SS-03	2/4	10-10	7.30E+01		3.1E+04	n	3.1E+04	No Action BSL
	67-64-1	ACETONE	11 J	770	ug/m ³	HB-HW-SS-03	4/4	-	7.70E+02		3.2E+05	n	3.2E+05	No Action BSL
	71-43-2	BENZENE	2.5	38	ug/m ³	HB-HW-SS-02	4/4	-	3.80E+01		3.1E+01	c	3.1E+01	Sample Indoor Air TOX
	123-91-1	1,4-DIOXANE	1.5	1.5	ug/m ³	HB-HW-SS-04	1/4	10-10	1.50E+00		3.2E+01	c	NV	Sample Indoor Air NTX
	75-15-0	CARBON DISULFIDE	1.5	9.2 J	ug/m ³	HB-HW-SS-02	3/4	5-5	9.20E+00		7.3E+03	n	7.3E+03	No Action BSL
	56-23-5	CARBON TETRACHLORIDE	2.6	2.6	ug/m ³	HB-HW-SS-04	1/4	5-5	2.60E+00		1.6E+01	c	1.6E+01	No Action BSL
	67-66-3	CHLOROFORM	35	100	ug/m ³	HB-HW-SS-01	2/4	5-5	1.00E+02		1.1E+01	c	1.1E+01	Sample Indoor Air ASL
	156-59-2	CIS-1,2-DICHLOROETHENE	1.6	1.6	ug/m ³	HB-HW-SS-04	1/4	5-5	1.60E+00		NV			Sample Indoor Air NTX
	75-71-8	DICHLORODIFLUOROMETHANE	2.6	2.6	ug/m ³	HB-HW-SS-04	1/4	5-5	2.60E+00		2.1E+03	n	2.1E+03	No Action BSL
	100-41-4	ETHYLBENZENE	5.3	7.1 J	ug/m ³	HB-HW-SS-03	2/4	5-5	7.10E+00		9.7E+01	c	9.7E+01	No Action BSL
	622-96-8	4-ETHYLTOLUENE	2.2	2.2	ug/m ³	HB-HW-SS-04	1/4	5-5	2.20E+00		NV			Sample Indoor Air NTX
	75-09-2	METHYLENE CHLORIDE	3.5 J	4.3 J	ug/m ³	HB-HW-SS-03	2/4	0.15-5	4.30E+00		5.2E+02	c	5.2E+02	No Action BSL
	100-42-5	STYRENE	1.8	1.8	ug/m ³	HB-HW-SS-04	1/4	5-5	1.80E+00		1.0E+04	n	1.0E+04	No Action BSL
	127-18-4	TETRACHLOROETHENE	1.2	23 J	ug/m ³	HB-HW-SS-01	4/4	-	2.30E+01		1.0E+02	c	1.0E+02	No Action BSL
	142-82-5	N-HEPTANE	2.7	45	ug/m ³	HB-HW-SS-03	3/4	5-5	4.50E+01		NV			Sample Indoor Air NTX
	110-54-3	N-HEXANE	4.6	30	ug/m ³	HB-HW-SS-02	3/4	5-5	3.00E+01		7.3E+03	n	7.3E+03	No Action BSL
	108-88-3	TOLUENE	14 J	120	ug/m ³	HB-HW-SS-03	4/4	-	1.20E+02		5.2E+04	n	5.2E+04	No Action BSL
	79-01-6	TRICHLOROETHENE	5.7	5.7	ug/m ³	HB-HW-SS-04	1/4	5-5	5.70E+00		5.0E+00	c	5.0E+00	Sample Indoor Air ASL
	75-69-4	TRICHLOROFLUOROMETHANE	0.91	9.5 J	ug/m ³	HB-HW-SS-02	2/4	5-5	9.50E+00		7.3E+03	n	7.3E+03	No Action BSL
	1330-20-7	XYLENES, TOTAL ^a	9.1 J	18	ug/m ³	HB-HW-SS-04	4/4	-	1.80E+01		1.0E+03	n	1.0E+03	No Action BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) N/A - No background screening performed.
(4) USEPA - Regional Screening Level Table, April 2009. c = Cancer; n = Noncancer. Cancer risk = 10⁻⁵ and Noncancer hazard = 1. Note for trichloroethene and tetrachlorethene, screening criteria are based on NYSDOH target indoor air concentration.
(5) Decision Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NV: No Value
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.9b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SUBSLAB VAPOR

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HW-SS-01	11/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	9.1	9.1
HB-HW-SS-02	11/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	9.5	9.5
HB-HW-SS-03	11/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	18	18
HB-HW-SS-04	11/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y		ug/l	18	18

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.9c
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SHALLOW GROUND WATER : VAPOR INTRUSION
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs*)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	Target Groundwater Concentration Corresponding to Target Indoor Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor = 0.001 and Partitioning Across the Water Table Obeys Henry's Law (5)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
Penn-Can Property - Shallow Ground Water	SVOCs														
	105-67-9	2,4-DIMETHYLPHENOL	1 J	6.3 J	ug/l	HB-HB-12S	2/9	9.5-11	6.30E+00				NV	Y	NTX
	91-57-6	2-METHYLNAPHTHALENE	3.1 J	10 J	ug/l	HB-HB-11S	2/9	9.5-11	1.00E+01			3.30E+02	nc	3.30E+02	BSL
	34METPH	3&4-METHYLPHENOLa	4 J	4 J	ug/l	HB-HB-12S	1/7	9.5-11	4.00E+00				NV	Y	NTX
	106-44-5	4-METHYLPHENOL	1.9 J	1.9 J	ug/l	HB-HB-12S	1/2	10-10	1.90E+00				NV	Y	NTX
	83-32-9	ACENAPHTHENE	29	29	ug/l	HB-HB-11S	1/9	9.5-11	2.90E+01			**	nc	**	INC
	208-96-8	ACENAPHTHYLENE	2 J	2 J	ug/l	HB-HB-11S	1/9	9.5-11	2.00E+00				NV	Y	NTX
	120-12-7	ANTHRACENE	39	39	ug/l	HB-HB-11S	1/9	9.5-11	3.90E+01				NV	Y	NTX
	56-55-3	BENZ(A)ANTHRACENE	1 J	69	ug/l	HB-HB-11S	2/9	9.5-11	6.90E+01		2 E-01		NV	Y	NTX
	50-32-8	BENZO(A)PYRENE	62	62	ug/l	HB-HB-11S	1/9	9.5-11	6.20E+01			**	c	**	INC
	205-99-2	BENZO(B)FLUORANTHENE	78	78	ug/l	HB-HB-11S	1/9	9.5-11	7.80E+01				NV	Y	NTX
	191-24-2	BENZO(G,H,I)PERYLENE	27	27	ug/l	HB-HB-11S	1/9	9.5-11	2.70E+01				NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	27	27	ug/l	HB-HB-11S	1/9	9.5-11	2.70E+01				NV	Y	NTX
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1.2 J	71 J	ug/l	HB-HB-14S	7/9	10-10	7.10E+01		6 E+00		NV	Y	NTX
	86-74-8	CARBAZOLE	24	24	ug/l	HB-HB-11S	1/9	9.5-11	2.40E+01				NV	Y	NTX
	218-01-9	CHRYSENE	1 J	62	ug/l	HB-HB-11S	3/9	9.5-11	6.20E+01			**	c	**	INC
	53-70-3	DIBENZ(A,H)ANTHRACENE	10 J	10 J	ug/l	HB-HB-11S	1/9	9.5-11	1.00E+01				NV	Y	NTX
	132-64-9	DIBENZOFURAN	14	14	ug/l	HB-HB-11S	1/9	9.5-11	1.40E+01			**	nc	**	INC
	206-44-0	FLUORANTHENE	1.2 J	120	ug/l	HB-HB-11S	5/9	9.5-11	1.20E+02				NV	Y	NTX
	86-73-7	FLUORENE	23	23	ug/l	HB-HB-11S	1/9	9.5-11	2.30E+01			**	nc	**	INC
	193-39-5	INDENO(1,2,3-CD)PYRENE	27	27	ug/l	HB-HB-11S	1/9	9.5-11	2.70E+01				NV	Y	NTX
	91-20-3	NAPHTHALENE	1 J	35	ug/l	HB-HB-12S	4/12	1-11	3.50E+01			1.50E+01	nc	1.50E+01	ASL
	85-01-8	PHENANTHRENE	1 J	120	ug/l	HB-HB-11S	3/9	9.5-11	1.20E+02				NV	Y	NTX
	108-95-2	PHENOL	34	34	ug/l	HB-HB-12S	1/9	9.5-11	3.40E+01				NV	Y	NTX
	129-00-0	PYRENE	1.1 J	99	ug/l	HB-HB-11S	5/9	9.5-11	9.90E+01			**	nc	**	INC
	VOCs														
	71-55-6	1,1,1-TRICHLOROETHANE	6	32 J	ug/l	HB-HB-12S	4/9	0.5-5	3.20E+01		2 E+02	3.10E+02	nc	3.10E+02	BSL
	75-34-3	1,1-DICHLOROETHANE	0.3 J	0.3 J	ug/l	HB-HB-12S	1/9	0.5-5	3.00E-01			2.20E+02	nc	2.20E+02	BSL
	75-35-4	1,1-DICHLOROETHENE	0.1 J	0.1 J	ug/l	HB-HB-12S	1/9	0.5-5	1.00E-01		7 E+00	1.90E+01	nc	1.90E+01	BSL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.3 J	0.3 J	ug/l	HB-HB-12S	1/3	0.5-0.5	3.00E-01			2.40E+00	nc	2.40E+00	BSL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.2 J	0.2 J	ug/l	HB-HB-12S	1/3	0.5-0.5	2.00E-01			2.50E+00	nc	2.50E+00	BSL
	67-64-1	ACETONE	6.9 J	6.9 J	ug/l	HB-HB-12S	1/9	10-20	6.90E+00			2.20E+04	nc	2.20E+04	BSL
	71-43-2	BENZENE	1.7 J	32.5	ug/l	HB-HB-12S	3/9	0.5-5	3.25E+01		5 E+00	1.37E+01	c	1.37E+01	Y
	75-27-4	BROMODICHLOROMETHANE	0.6	0.6	ug/l	HB-HB-11S	1/9	0.5-5	6.00E-01			2.10E+00	c	2.10E+00	N
	75-00-3	CHLOROETHANE	0.6 J	0.6 J	ug/l	HB-HB-14S	1/9	1-5	6.00E-01			2.80E+03	nc	2.80E+03	N
	67-66-3	CHLOROFORM	5.56	27	ug/l	HB-HB-11S	2/9	0.5-5	2.70E+01			7.33E+00	c	7.33E+00	Y
	156-59-2	CIS-1,2-DICHLOROETHENE	0.1 J	0.1 J	ug/l	HB-HB-12S	1/9	0.5-5	1.00E-01		7 E+01		NV	Y	NTX
	100-41-4	ETHYLBENZENE	0.5 J	1.56	ug/l	HB-HB-12S	4/9	0.5-5	1.56E+00		7 E+02	3.01E+01	c	3.01E+01	N
	100-42-5	STYRENE	1.2 J	1.2 J	ug/l	HB-HB-12S	1/9	0.5-5	1.20E+00		1 E+02	8.90E+02	nc	8.90E+02	N
	127-18-4	TETRACHLOROETHENE	0.14 J	1.7 J	ug/l	HB-HB-12S	4/9	0.5-5	1.70E+00		5 E+00	1.33E+01	c	1.33E+01	N
	108-88-3	TOLUENE	2.7 J	16.9	ug/l	HB-HB-12S	4/9	0.5-5	1.69E+01		1 E+03	1.50E+02	nc	1.50E+02	N
	1330-20-7	XYLENES, TOTAL ^a	2 J	11.7	ug/l	HB-HB-12S	4/9	0.25-5	1.17E+01		1 E+04	2.20 E+03	nc	2.20E+03	N

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

** Target soil gas concentration exceeds maximum possible vapor concentration (pathway incomplete)

(1) J - estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.

(5) USEPA - OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils (Subsurface Vapor Intrusion Guidance) Tables. November 2002. ca = Cancer; nc = Noncancer. Screening criteria correspond to a cancer risk of 10-6 and a noncancer hazard of 0.1. For USEPA (2002) criteria that defaulted to MCLs, criteria were derived (in italics) from USEPA (2009) RSL residential air concentration based on an attenuation factor of 10 and the Henry's Law constant for each compound at 25 deg C.

(6) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; INC - Pathway Incomplete

a = Target groundwater concentration for p-xylene (CAS #106-42-3) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NV: No Value

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.9d
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
ONEYWELL, WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SHALLOW GROUND WATER : VAPOR INTRUSIC

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-11S	3/15/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-11S	5/11/2001	3.98	13.98	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-12S	5/14/2001	5.96	15.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2	2
HB-HB-12S	5/12/2003	5.96	15.96	XYLENES1314	XYLENES, M & P	Y		ug/l	8	
HB-HB-12S	5/12/2003	5.96	15.96	95-47-6	O-XYLENE	Y	J	ug/l	3.7	
HB-HB-12S	5/12/2003	5.96	15.96	CALCULATED	TOTAL	Y		ug/l		11.7
HB-HB-12S	8/13/2003	5.96	15.96	XYLENES1314	XYLENES, M & P	Y	J	ug/l	4.4	
HB-HB-12S	8/13/2003	5.96	15.96	95-47-6	O-XYLENE	Y	J	ug/l	1.8	
HB-HB-12S	8/13/2003	5.96	15.96	CALCULATED	TOTAL	Y	J	ug/l		6.2
HB-HB-12S	3/16/2007	5.96	15.96	1330-20-7	XYLENES, TOTAL	Y		ug/l	5.06	5.06
HB-HB-14S	5/16/2001	6.95	11.95	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-14S	5/13/2003	6.95	11.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-14S	5/13/2003	6.95	11.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-14S	5/13/2003	6.95	11.95	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-14S	8/18/2003	6.95	11.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-14S	8/18/2003	6.95	11.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-14S	8/18/2003	6.95	11.95	CALCULATED	TOTAL	N	U	ug/l		5

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.10a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs*)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
Penn-Can Property - Shallow Ground Water	METALS															
	7429-90-5	ALUMINUM	0.051 J	8.46 J	mg/L	HB-HB-14S	7/8	0.1-0.1	8.46E+00		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00 Y ASL
	7440-36-0	ANTIMONY	0.0049 J	0.0049 J	mg/L	HB-HB-11S	1/8	0.0014-0.06	4.90E-03		6.00E-03	1.46E-03	N	1.46E-03	nc	1.46E-03 Y ASL
	7440-38-2	ARSENIC	0.0041 J	0.0181	mg/L	HB-HB-11S	3/8	0.01-0.01	1.81E-02		1.00E-02	4.46E-05	C	4.48E-05	ca	4.46E-05 Y TOX
	7440-39-3	BARIIUM	0.0357	0.191	mg/L	HB-HB-14S	8/8	-	1.91E-01		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01 N BSL
	7440-41-7	BERYLLIUM	0.0002 J	0.0002 J	mg/L	HB-HB-12S	1/8	0.000076-0.01	2.00E-04		4.00E-03	7.30E-03	N	7.30E-03	nc	7.30E-03 N BSL
	7440-70-2	CALCIUM	73	760	mg/L	HB-HB-12S	8/8	-	7.60E+02			NV	NV	NV	N	NUT
	7440-47-3	CHROMIUM ^a	0.0041 J	0.0521	mg/L	HB-HB-12S	5/8	0.01-0.0108	5.21E-02		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02 Y TOX
	7440-50-8	COPPER	0.0019 J	0.0162 J	mg/L	HB-HB-12S	3/8	0.01-0.02	1.62E-02		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01 N BSL
	57-12-5	CYANIDE	0.017	0.035	mg/L	HB-HB-12S	2/8	0.01-0.01	3.50E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02 N BSL
	7439-89-6	IRON	0.059	9.84 J	mg/L	HB-HB-14S	7/8	0.05-0.05	9.84E+00		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00 Y ASL
	7439-92-1	LEAD	0.007	0.0254	mg/L	HB-HB-11S	4/8	0.005-0.01	2.54E-02		1.50E-02	NV	NV	NV	Y	ASL
	7439-95-4	MAGNESIUM	2.15 J	23.3	mg/L	HB-HB-14S	7/8	1-1	2.33E+01			NV	NV	NV	N	NUT
	7439-96-5	MANGANESE	0.0203 J	0.36	mg/L	HB-HB-12S	6/8	0.05-0.05	3.60E-01		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02 Y ASL
	7439-97-6	MERCURY ^b	0.0009	0.0009	mg/L	HB-HB-11S	1/8	0.00018-0.0002	9.00E-04		2.00E-03	3.65E-04	N	3.65E-04	nc	3.65E-04 Y ASL
	7440-02-0	NICKEL	0.0064 J	0.0288 J	mg/L	HB-HB-12S	3/8	0.04-0.05	2.88E-02			7.30E-02	N	7.30E-02	nc	7.30E-02 N BSL
	7440-09-7	POTASSIUM	1.88 J	13 J	mg/L	HB-HB-12S	8/8	-	1.30E+01			NV	NV	NV	N	NUT
	7782-49-2	SELENIUM	0.0044 J	0.0044 J	mg/L	HB-HB-12S	1/8	0.0018-0.01	4.40E-03		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02 N BSL
	7440-22-4	SILVER	0.0012 J	0.0245	mg/L	HB-HB-12S	2/8	0.01-0.01	2.45E-02		1.00E-01	1.83E-02	N	1.82E-02	nc	1.82E-02 Y ASL
	7440-23-5	SODIUM	16	83	mg/L	HB-HB-11S	8/8	-	8.30E+01			NV	NV	NV	N	NUT
	7440-62-2	VANADIUM	0.0013 J	0.0151 J	mg/L	HB-HB-12S	2/8	0.05-0.05	1.51E-02			3.65E-03	N	3.65E-03	nc	3.65E-03 Y ASL
	7440-66-6	ZINC	0.0172 J	0.0726	mg/L	HB-HB-14S	5/8	0.02-0.02	7.26E-02		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00 N BSL
	PCBs															
		HIGHLY CHLORINATED PCBs ^c	0.3	0.3	ug/l	HB-HB-11S	1/8	0.5-0.99	3.00E-01		5.00E-01	3.35E-02	C	3.36E-02	ca	3.35E-02 Y ASL
		TOTAL PCBs ^d	0.3	0.3	ug/l	HB-HB-11S	1/8	0.50.99	3.00E-01		5.00E-01	3.35E-02	C	3.36E-02	ca	3.35E-02 Y ASL
	SVOCs															
	105-67-9	2,4-DIMETHYLPHENOL	1 J	6.3 J	ug/l	HB-HB-12S	2/9	9.5-11	6.30E+00			7.30E+01	N	7.30E+01	nc	7.30E+01 N BSL
	91-57-6	2-METHYLNAPHTHALENE	3.1 J	10 J	ug/l	HB-HB-11S	2/9	9.5-11	1.00E+01			2.43E+00	N	NV	nc	2.43E+00 Y ASL
	34METPH	3&4-METHYLPHENOL ^e	4 J	4 J	ug/l	HB-HB-12S	1/7	9.5-11	4.00E+00			1.83E+01	N	1.82E+01	nc	1.82E+01 N BSL
	106-44-5	4-METHYLPHENOL	1.9 J	1.9 J	ug/l	HB-HB-12S	1/2	10-10	1.90E+00			1.83E+01	N	1.82E+01	nc	1.82E+01 N BSL
	83-32-9	ACENAPHTHENE	29	29	ug/l	HB-HB-11S	1/9	9.5-11	2.90E+01			3.65E+01	N	3.65E+01	nc	3.65E+01 N BSL
	208-96-8	ACENAPHTHYLENE	2 J	2 J	ug/l	HB-HB-11S	1/9	9.5-11	2.00E+00			NV	NV	NV	Y	NTX
	120-12-7	ANTHRACENE	39	39	ug/l	HB-HB-11S	1/9	9.5-11	3.90E+01			1.83E+02	N	1.83E+02	nc	1.83E+02 N BSL
	56-55-3	BENZ(A)ANTHRACENE	1 J	69	ug/l	HB-HB-11S	2/9	9.5-11	6.90E+01		2.00E-01	3.00E-02	C	9.21E-02	ca	3.00E-02 Y ASL
	50-32-8	BENZO(A)PYRENE	62	62	ug/l	HB-HB-11S	1/9	9.5-11	6.20E+01			3.00E-03	C	9.21E-03	ca	3.00E-03 Y ASL
	205-99-2	BENZO(B)FLUORANTHENE	78	78	ug/l	HB-HB-11S	1/9	9.5-11	7.80E+01			3.00E-02	C	9.21E-02	ca	3.00E-02 Y ASL
	191-24-2	BENZO(G,H,I)PERYLENE	27	27	ug/l	HB-HB-11S	1/9	9.5-11	2.70E+01			NV	NV	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	27	27	ug/l	HB-HB-11S	1/9	9.5-11	2.70E+01			3.00E-01	C	9.21E-01	ca	3.00E-01 Y ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1.2 J	71 J	ug/l	HB-HB-14S	7/9	10-10	7.10E+01		6.00E+00	4.78E+00	C	4.80E+00	ca	4.78E+00 Y ASL
	86-74-8	CARBAZOLE	24	24	ug/l	HB-HB-11S	1/9	9.5-11	2.40E+01			3.35E+00	C	3.36E+00	ca	3.35E+00 Y ASL
	218-01-9	CHRYSENE	1 J	62	ug/l	HB-HB-11S	3/9	9.5-11	6.20E+01			3.00E+00	C	9.21E+00	ca	3.00E+00 Y ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	10 J	10 J	ug/l	HB-HB-11S	1/9	9.5-11	1.00E+01			3.00E-03	C	9.21E-03	ca	3.00E-03 Y ASL
	132-64-9	DIBENZOFURAN	14	14	ug/l	HB-HB-11S	1/9	9.5-11	1.40E+01			3.65E+00	N	1.22E+00	nc	1.22E+00 Y ASL
	206-44-0	FLUORANTHENE	1.2 J	120	ug/l	HB-HB-11S	5/9	9.5-11	1.20E+02			1.46E+02	N	1.46E+02	nc	1.46E+02 N BSL

TABLE 2.10a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs*)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	86-73-7	FLUORENE	23	23	ug/l	HB-HB-11S	1/9	9.5-11	2.30E+01			2.43E+01	N	2.43E+01	nc	2.43E+01	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	27	27	ug/l	HB-HB-11S	1/9	9.5-11	2.70E+01			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	91-20-3	NAPHTHALENE	1 J	35	ug/l	HB-HB-12S	4/12	1-11	3.50E+01			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	85-01-8	PHENANTHRENE	1 J	120	ug/l	HB-HB-11S	3/9	9.5-11	1.20E+02			NV	NV	NV	Y	NTX		
	108-95-2	PHENOL	34	34	ug/l	HB-HB-12S	1/9	9.5-11	3.40E+01			1.10E+03	N	1.09E+03	nc	1.09E+03	N	BSL
	129-00-0	PYRENE	1.1 J	99	ug/l	HB-HB-11S	5/9	9.5-11	9.90E+01			1.83E+01	N	1.83E+01	nc	1.83E+01	Y	ASL
	VOCs																	
	71-55-6	1,1,1-TRICHLOROETHANE	6	32 J	ug/l	HB-HB-12S	4/9	0.5-5	3.20E+01		2.00E+02	9.13E+02	N	3.17E+02	nc	3.17E+02	N	BSL
	75-34-3	1,1-DICHLOROETHANE	0.3 J	0.3 J	ug/l	HB-HB-12S	1/9	0.5-5	3.00E-01			8.96E+01	N	8.11E+01	nc	8.11E+01	N	BSL
	75-35-4	1,1-DICHLOROETHENE	0.1 J	0.1 J	ug/l	HB-HB-12S	1/9	0.5-5	1.00E-01		7.00E+00	3.53E+01	N	3.39E+01	nc	3.39E+01	N	BSL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.3 J	0.3 J	ug/l	HB-HB-12S	1/3	0.5-0.5	3.00E-01			1.46E+00	N	1.23E+00	nc	1.23E+00	N	BSL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.2 J	0.2 J	ug/l	HB-HB-12S	1/3	0.5-0.5	2.00E-01			NV	NV	nc	1.23E+00	N	BSL	
	67-64-1	ACETONE	6.9 J	6.9 J	ug/l	HB-HB-12S	1/9	10-20	6.90E+00			5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	71-43-2	BENZENE	1.7 J	32.5	ug/l	HB-HB-12S	3/9	0.5-5	3.25E+01		5.00E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	75-27-4	BROMODICHLOROMETHANE	0.6	0.6	ug/l	HB-HB-11S	1/9	0.5-5	6.00E-01			1.70E-01	C	1.81E-01	ca	1.70E-01	Y	ASL
	75-00-3	CHLOROETHANE	0.6 J	0.6 J	ug/l	HB-HB-14S	1/9	1-5	6.00E-01			3.64E+00	C	4.64E+00	ca	3.64E+00	N	BSL
	67-66-3	CHLOROFORM	5.56	27	ug/l	HB-HB-11S	2/9	0.5-5	2.70E+01			1.55E-01	C	1.66E-01	ca	1.55E-01	Y	ASL
	156-59-2	CIS-1,2-DICHLOROETHENE	0.1 J	0.1 J	ug/l	HB-HB-12S	1/9	0.5-5	1.00E-01		7.00E+01	6.08E+00	N	6.08E+00	nc	6.08E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.5 J	1.56	ug/l	HB-HB-12S	4/9	0.5-5	1.56E+00			7.00E+02	N	1.34E+02	nc	1.34E+02	N	BSL
	100-42-5	STYRENE	1.2 J	1.2 J	ug/l	HB-HB-12S	1/9	0.5-5	1.20E+00			1.62E+02	N	1.64E+02	nc	1.62E+02	N	BSL
	127-18-4	TETRACHLOROETHENE	0.14 J	1.7 J	ug/l	HB-HB-12S	4/9	0.5-5	1.70E+00			5.00E+00	C	1.04E-01	ca	1.04E-01	Y	ASL
	108-88-3	TOLUENE	2.7 J	16.9	ug/l	HB-HB-12S	4/9	0.5-5	1.69E+01			1.00E+03	N	7.23E+01	nc	7.23E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	2 J	11.7	ug/l	HB-HB-12S	4/9	0.25-5	1.17E+01			1.00E+04	N	2.06E+01	nc	2.06E+01	N	BSL

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.

(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.

(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.

(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.

(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = RBC and PRG values for chromium VI utilized.

b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

e = RBC and PRG values for 4-methylphenol (CAS # 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NUT: Compound is an essential nutrient and not screened in

NV: No Value

PRG: Preliminary Remediation Goals, USEPA, 2004

RBC: Risk Based Concentration; USEPA, October 2007

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.10b
 DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SHALLOW GROUND WATER

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-HB-11S	3.98	13.98	5/11/2001	0.3	ug/l
Total PCBs	HB-HB-11S	3.98	13.98	5/11/2001	0.3	ug/l

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.10c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SHALLOW GROUND WATER (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-11S	3/15/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-11S	5/11/2001	3.98	13.98	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-12S	3/16/2007	5.96	15.96	1330-20-7	XYLENES, TOTAL	Y		ug/l	5.06	5.06
HB-HB-12S	5/14/2001	5.96	15.96	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	2	2
HB-HB-14S	5/16/2001	6.95	11.95	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-12S	5/12/2003	5.96	15.96	XYLENES1314	XYLENES, M & P	Y		ug/l	8	
HB-HB-12S	5/12/2003	5.96	15.96	95-47-6	O-XYLENE	Y	J	ug/l	3.7	
HB-HB-12S	5/12/2003	5.96	15.96	CALCULATED	TOTAL	Y		ug/l		11.7
HB-HB-12S	8/13/2003	5.96	15.96	XYLENES1314	XYLENES, M & P	Y	J	ug/l	4.4	
HB-HB-12S	8/13/2003	5.96	15.96	95-47-6	O-XYLENE	Y	J	ug/l	1.8	
HB-HB-12S	8/13/2003	5.96	15.96	CALCULATED	TOTAL	Y	J	ug/l		6.2
HB-HB-14S	5/13/2003	6.95	11.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-14S	5/13/2003	6.95	11.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-14S	5/13/2003	6.95	11.95	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-14S	8/18/2003	6.95	11.95	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-14S	8/18/2003	6.95	11.95	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-14S	8/18/2003	6.95	11.95	CALCULATED	TOTAL	N	U	ug/l		5

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.11a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- PENN-CAN PROPERTY SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
PennCan Property - Surface Sediment	DIOXIN/FURAN (8)																	
	1746-01-6	2,3,7,8-TCDD Equivalent	0.0000006	0.0000671	mg/kg	HB-HBSED-01	7/7		6.71E-05			4.26E-06	C	3.90E-06	ca	3.90E-06	Y	ASL
	METALS																	
	7429-90-5	ALUMINUM	566 J	3250 J	mg/kg	HB-HBSED-03	7/7	-	3.25E+03			7.82E+03	N	7.61E+03	nc	7.61E+03	N	BSL
	7440-36-0	ANTIMONY	0.45 J	0.45 J	mg/kg	HB-HBSED-02	1/7	0.3-15.2	4.50E-01			3.13E+00	N	3.13E+00	nc	3.13E+00	N	BSL
	7440-38-2	ARSENIC	2.3 J	8.8 J	mg/kg	HB-HBSED-01	6/7	2.4-2.4	8.80E+00			4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX
	7440-39-3	BARIUM	16.4 J	96.2 J	mg/kg	HB-HBSED-01	7/7	-	9.62E+01			1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL
	7440-41-7	BERYLLIUM	0.085 J	0.24 J	mg/kg	HB-HBSED-03	3/7	0.89-1.3	2.40E-01			1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL
	7440-70-2	CALCIUM	102000 J	413000 J	mg/kg	HB-HBSED-02	7/7	-	4.13E+05			NV	NV	NV	NV	NV	N	NUT
	7440-47-3	CHROMIUM ^a	2.7 J	11.8 J	mg/kg	HB-HBSED-03	7/7	-	1.18E+01			2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX
	7440-50-8	COPPER	3.7 J	35.1 J	mg/kg	HB-HBSED-03	7/7	-	3.51E+01			3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL
	57-12-5	CYANIDE	2.8 J	4 J	mg/kg	HB-HBSED-03	2/7	1.1-2.69	4.00E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL
	7439-89-6	IRON	2070 J	11500 J	mg/kg	HB-HBSED-03	7/7	-	1.15E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL
	7439-92-1	LEAD	6.9 J	57 J	mg/kg	HB-HBSED-03	7/7	-	5.70E+01			NV	NV	4.00E+02	nc	4.00E+02	N	BSL
	7439-95-4	MAGNESIUM	5920	13600 J	mg/kg	HB-HBSED-03	7/7	-	1.36E+04			NV	NV	NV	NV	NV	N	NUT
	7439-96-5	MANGANESE	91.3 J	664 J	mg/kg	HB-HBSED-01	7/7	-	6.64E+02			1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL
	7439-97-6	MERCURY ^b	0.05 J	0.86	mg/kg	HB-HBSED-03	5/7	0.0407-0.062	8.60E-01			7.82E-01	N	6.11E-01	nc	6.11E-01	Y	ASL
	7440-02-0	NICKEL	1.8 J	10.8 J	mg/kg	HB-HBSED-03	4/7	7.1-10.1	1.08E+01			1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL
	7440-09-7	POTASSIUM	213 J	680 J	mg/kg	HB-HBSED-03	5/7	478-507	6.80E+02			NV	NV	NV	NV	NV	N	NUT
	7440-23-5	SODIUM	261 J	696 J	mg/kg	HB-HBSED-01	7/7	-	6.96E+02			NV	NV	NV	NV	NV	N	NUT
	7440-62-2	VANADIUM	1.7 J	11.8 J	mg/kg	HB-HBSED-03	4/7	8.9-12.7	1.18E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL
	7440-66-6	ZINC	10.7 J	109 J	mg/kg	HB-HBSED-03	7/7	-	1.09E+02			2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL
	PCBs																	
		HIGHLY CHLORINATED PCBs ^c	0.03	0.05	mg/kg	HB-HBSED-03	3/7	0.03-0.18	5.00E-02			3.19E-02	N	2.22E-02	nc	2.22E-02	Y	ASL
		TOTAL PCBs ^d	0.03	0.05	mg/kg	HB-HBSED-03	3/7	0.03-0.18	5.00E-02			3.19E-02	N	2.22E-02	nc	2.22E-02	Y	ASL
	PESTICIDES																	
	72-55-9	4,4'-DDE	0.003 J	0.003 J	mg/kg	HB-HBSED-01, HB-HBSED-02	2/7	0.01-0.044	3.00E-03			1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL
	SVOCs																	
	91-57-6	2-METHYLNAPHTHALENE	0.077 J	0.67 J	mg/kg	HB-HBSED-02	7/7	-	6.70E-01			3.13E+01	N	NV	NV	3.13E+01	N	BSL
	83-32-9	ACENAPHTHENE	0.13 J	0.8 J	mg/kg	HB-HBSED-03	6/7	0.71-0.71	8.00E-01			4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL
	208-96-8	ACENAPHTHYLENE	0.11 J	1.4	mg/kg	HB-HBSED-03	6/7	0.68-0.68	1.40E+00			NV	NV	NV	NV	NV	Y	NTX
	120-12-7	ANTHRACENE	0.11 J	1.5 J	mg/kg	HB-HBSED-03	6/7	0.68-0.68	1.50E+00			2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	0.094 J	3.2 J	mg/kg	HB-HBSED-03	7/7	-	3.20E+00			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	50-32-8	BENZO(A)PYRENE	0.27 J	3.9	mg/kg	HB-HBSED-03	6/7	0.68-0.68	3.90E+00			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.096 J	3.5	mg/kg	HB-HBSED-03	7/7	-	3.50E+00			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.18 J	2.2	mg/kg	HB-HBSED-03	6/7	0.68-0.68	2.20E+00			NV	NV	NV	NV	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.17 J	3.3	mg/kg	HB-HBSED-03	6/7	0.68-0.68	3.30E+00			2.20E+00	C	6.21E+00	ca	2.20E+00	Y	ASL
	86-74-8	CARBAZOLE	0.17 J	0.46 J	mg/kg	HB-HBSED-03	4/7	0.68-0.89	4.60E-01			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL
	218-01-9	CHRYSENE	0.097 J	3.4 J	mg/kg	HB-HBSED-03	7/7	-	3.40E+00			2.20E+01	C	6.21E+01	ca	2.20E+01	N	BSL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.075 J	0.86	mg/kg	HB-HBSED-03	5/7	0.68-1.2	8.60E-01			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.076 J	1.6 J	mg/kg	HB-HBSED-03	7/7	-	1.60E+00			7.82E+00	N	1.45E+01	nc	7.82E+00	N	BSL
	84-74-2	DI-N-BUTYL PHTHALATE	0.1 J	0.15 J	mg/kg	HB-HBSED-03	2/7	0.68-1.2	1.50E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL

TABLE 2.11a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- PENN-CAN PROPERTY SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
	206-44-0	FLUORANTHENE	0.2 J	6.7 J	mg/kg	HB-HBSED-03	7/7	-	6.70E+00			3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	86-73-7	FLUORENE	0.13 J	1 J	mg/kg	HB-HBSED-03	5/7	0.68-0.71	1.00E+00			3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.18 J	2.4	mg/kg	HB-HBSED-03	6/7	0.68-0.68	2.40E+00			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.095 J	2	mg/kg	HB-HBSED-03	8/10	0.011-0.019	2.00E+00			1.56E+02	N	5.59E+00	nc	5.59E+00	N	BSL
	85-01-8	PHENANTHRENE	0.13 J	3.2 J	mg/kg	HB-HBSED-03	7/7	-	3.20E+00			NV	NV	NV	NV	NV	Y	NTX
	129-00-0	PYRENE	0.19 J	4.1 J	mg/kg	HB-HBSED-03	7/7	-	4.10E+00			2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																	
	95-50-1	1,2-DICHLOROBENZENE	0.006 J	0.006 J	mg/kg	HB-HBSED-01	1/10	0.009-1.4	6.00E-03			7.04E+02	N	6.00E+02	sat	6.00E+02	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.002 J	0.002 J	mg/kg	HB-HBSED-01	1/10	0.009-1.4	2.00E-03			2.66E+01	C	3.45E+00	ca	3.45E+00	N	BSL
	78-93-3	2-BUTANONE	0.0063	0.014 J	mg/kg	HB-HBSED-01	4/8	0.021-0.2	1.40E-02			4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.01 J	0.074 J	mg/kg	HB-HBSED-02	7/8	0.021-0.021	7.40E-02			7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.001 J	0.001 J	mg/kg	HB-HBSED-01	1/3	0.009-0.051	1.00E-03			NV	NV	NV	NV	NV	Y	NTX
	1330-20-7	XYLENES, TOTAL	0.001 J	0.005 J	mg/kg	HB-HBSED-03	2/7	0.001-0.0255	5.00E-03			1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(8) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.11b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.11b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-01	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	856	856	ng/kg	J	0.01	8.560
HB-HBSED-01	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	54.6	54.6	ng/kg	J	0.01	0.546
HB-HBSED-01	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	33.3	33.3	ng/kg	J	0.1	3.330
HB-HBSED-01	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	22.1	22.1	ng/kg	J	0.1	2.210
HB-HBSED-01	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	226	226	ng/kg	J	0.1	22.600
HB-HBSED-01	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	16.4	16.4	ng/kg	J	0.1	1.640
HB-HBSED-01	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	66.3	66.3	ng/kg	J	0.1	6.630
HB-HBSED-01	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-01	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	12.6	12.6	ng/kg	J	1	12.600
HB-HBSED-01	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	5	2.5	ng/kg	UJ	0.03	0.075
HB-HBSED-01	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.9	1.9	ng/kg	J	1	1.900
HB-HBSED-01	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	4.45	4.45	ng/kg	J	1	0.445
HB-HBSED-01	5/11/2001	0	0.5	3268-87-9	OCDD	Y	19500	19500	ng/kg	J	0.0003	5.850
HB-HBSED-01	5/11/2001	0	0.5	39001-02-0	OCDF	Y	1480	1480	ng/kg	J	0.0003	0.444
Sample Location TEQ = 67.1												
HB-HBSED-01	6/3/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	290.842	290.842	ng/kg	J	0.01	2.908
HB-HBSED-01	6/3/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	13.802	13.802	ng/kg	J	0.01	0.138
HB-HBSED-01	6/3/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	4.875	4.875	ng/kg	J	0.1	0.488
HB-HBSED-01	6/3/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	8.15	8.15	ng/kg	J	0.1	0.815
HB-HBSED-01	6/3/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	66.446	66.446	ng/kg	J	0.1	6.645
HB-HBSED-01	6/3/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	8.02	8.02	ng/kg	J	0.1	0.802
HB-HBSED-01	6/3/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	17.33	17.33	ng/kg	J	0.1	1.733
HB-HBSED-01	6/3/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.802	1.802	ng/kg	J	1	1.802
HB-HBSED-01	6/3/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.862	1.862	ng/kg	J	0.03	0.056
HB-HBSED-01	6/3/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.129	0.0645	ng/kg	U	1	0.065
HB-HBSED-01	6/3/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.095	1.095	ng/kg	J	0.1	0.110
HB-HBSED-01	6/3/2003	0	0.5	3268-87-9	OCDD	Y	11026.597	11026.597	ng/kg	J	0.0003	3.308
HB-HBSED-01	6/3/2003	0	0.5	39001-02-0	OCDF	Y	852.189	852.189	ng/kg	J	0.0003	0.256
Sample Location TEQ = 19.1												
HB-HBSED-02	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	112	112	ng/kg	J	0.01	1.120
HB-HBSED-02	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	6.79	6.79	ng/kg	J	0.01	0.068
HB-HBSED-02	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	5.84	5.84	ng/kg	J	0.1	0.584
HB-HBSED-02	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-02	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	35.4	35.4	ng/kg	J	0.1	3.540
HB-HBSED-02	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-02	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	14.4	14.4	ng/kg	J	0.1	1.440
HB-HBSED-02	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-02	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	5	2.5	ng/kg	UJ	1	2.500
HB-HBSED-02	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	5	2.5	ng/kg	UJ	0.03	0.075
HB-HBSED-02	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-HBSED-02	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.76	0.76	ng/kg	J	0.1	0.076
HB-HBSED-02	5/11/2001	0	0.5	3268-87-9	OCDD	Y	5420	5420	ng/kg	J	0.0003	1.626
HB-HBSED-02	5/11/2001	0	0.5	39001-02-0	OCDF	Y	1240	1240	ng/kg	J	0.0003	0.372
Sample Location TEQ = 12.7												
HB-HBSED-02	6/3/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	11.849	11.849	ng/kg	J	0.01	0.118
HB-HBSED-02	6/3/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.545	0.2725	ng/kg	U	0.01	0.003
HB-HBSED-02	6/3/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.545	2.545	ng/kg	J	0.1	0.255
HB-HBSED-02	6/3/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.252	0.126	ng/kg	U	0.1	0.013
HB-HBSED-02	6/3/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.834	1.834	ng/kg	J	0.1	0.183
HB-HBSED-02	6/3/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.346	0.173	ng/kg	U	0.1	0.017
HB-HBSED-02	6/3/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.378	0.378	ng/kg	J	1	0.378
HB-HBSED-02	6/3/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.148	0.074	ng/kg	UJ	0.03	0.002
HB-HBSED-02	6/3/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.15	0.075	ng/kg	U	1	0.075
HB-HBSED-02	6/3/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	N	0.166	0.083	ng/kg	U	0.1	0.008
HB-HBSED-02	6/3/2003	0	0.5	3268-87-9	OCDD	Y	1132.151	1132.151	ng/kg	J	0.0003	0.340
HB-HBSED-02	6/3/2003	0	0.5	39001-02-0	OCDF	Y	75.146	75.146	ng/kg	J	0.0003	0.023
Sample Location TEQ = 1.4												
HB-HBSED-03	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	136	136	ng/kg	J	0.01	1.360
HB-HBSED-03	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	9.44	9.44	ng/kg	J	0.01	0.094
HB-HBSED-03	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	5.95	5.95	ng/kg	J	0.1	0.595
HB-HBSED-03	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-03	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	40.8	40.8	ng/kg	J	0.1	4.080
HB-HBSED-03	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-03	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	14.3	14.3	ng/kg	J	0.1	1.430
HB-HBSED-03	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-03	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	5	2.5	ng/kg	UJ	1	2.500
HB-HBSED-03	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	5	2.5	ng/kg	UJ	0.03	0.075
HB-HBSED-03	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-HBSED-03	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.66	1.66	ng/kg	J	0.1	0.166
HB-HBSED-03	5/11/2001	0	0.5	3268-87-9	OCDD	Y	12800	12800	ng/kg	J	0.0003	3.840
HB-HBSED-03	5/11/2001	0	0.5	39001-02-0	OCDF	Y	1220	1220	ng/kg	J	0.0003	0.366
Sample Location TEQ = 15.8												
HB-HBSED-03	6/3/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	14.57	14.57	ng/kg	J	0.01	0.146
HB-HBSED-03	6/3/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.714	0.357	ng/kg	UJ	0.01	0.004
HB-HBSED-03	6/3/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.362	0.181	ng/kg	UJ	0.1	0.018
HB-HBSED-03	6/3/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.351	0.1755	ng/kg	UJ	0.1	0.018
HB-HBSED-03	6/3/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.805	2.805	ng/kg	J	0.1	0.281
HB-HBSED-03	6/3/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.325	0.1625	ng/kg	UJ	0.1	0.016
HB-HBSED-03	6/3/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.445	0.2225	ng/kg	UJ	0.1	0.022
HB-HBSED-03	6/3/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.282	0.141	ng/kg	UJ	1	0.141
HB-HBSED-03	6/3/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.251	0.1255	ng/kg	UJ	0.03	0.004
HB-HBSED-03	6/3/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.279	0.1395	ng/kg	UJ	1	0.140
HB-HBSED-03	6/3/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.239	0.239	ng/kg	J	0.1	0.024
HB-HBSED-03	6/3/2003	0	0.5	3268-87-9	OCDD	Y	1251.471	1251.471	ng/kg	J	0.0003	0.375
HB-HBSED-03	6/3/2003	0	0.5	39001-02-0	OCDF	Y	84.03	84.03	ng/kg	J	0.0003	0.025
Sample Location TEQ = 1.2												

TABLE 2.11b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-03	6/3/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.089	8.089	ng/kg		0.01	0.081
HB-HBSED-03	6/3/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.325	0.325	ng/kg	J	0.01	0.003
HB-HBSED-03	6/3/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HxCDD	N	0.212	0.106	ng/kg	U	0.1	0.011
HB-HBSED-03	6/3/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HxCDF	Y	0.333	0.333	ng/kg	J	0.1	0.033
HB-HBSED-03	6/3/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HxCDF	N	0.25	0.125	ng/kg	U	0.1	0.013
HB-HBSED-03	6/3/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HxCDD	Y	0.667	0.667	ng/kg	J	0.1	0.067
HB-HBSED-03	6/3/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HxCDF	N	0.343	0.1715	ng/kg	U	0.1	0.017
HB-HBSED-03	6/3/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	0.169	0.0845	ng/kg	U	1	0.085
HB-HBSED-03	6/3/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	0.144	0.072	ng/kg	U	0.03	0.002
HB-HBSED-03	6/3/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.163	0.0815	ng/kg	U	1	0.082
HB-HBSED-03	6/3/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	N	0.13	0.065	ng/kg	U	0.1	0.007
HB-HBSED-03	6/3/2003	0.5	1	3268-87-9	OCDD	Y	644.304	644.304	ng/kg		0.0003	0.193
HB-HBSED-03	6/3/2003	0.5	1	39001-02-0	OCDF	Y	47.192	47.192	ng/kg		0.0003	0.014
Sample Location TEQ =												0.6

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PxCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223-241.

TABLE 2.11c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-HBSED-01	0	0.5	5/8/2001	0.04	mg/kg
Highly Chlorinated PCBs	HB-HBSED-02	0	0.5	5/8/2001	0.03	mg/kg
Highly Chlorinated PCBs	HB-HBSED-03	0	0.5	5/7/2001	0.05	mg/kg
Total PCBs	HB-HBSED-01	0	0.5	5/8/2001	0.04	mg/kg
Total PCBs	HB-HBSED-02	0	0.5	5/8/2001	0.03	mg/kg
Total PCBs	HB-HBSED-03	0	0.5	5/7/2001	0.05	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.11d
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HBSED-01	5/8/2001	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.001	0.001
HB-HBSED-01	6/3/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.013	
HB-HBSED-01	6/3/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.013	
HB-HBSED-01	6/3/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.013
HB-HBSED-02	5/8/2001	0	0.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.051	0.0255
HB-HBSED-02	6/3/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.012	
HB-HBSED-02	6/3/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.012	
HB-HBSED-02	6/3/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.012
HB-HBSED-03	5/7/2001	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.005	0.005
HB-HBSED-03	6/3/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.011	
HB-HBSED-03	6/3/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.011	
HB-HBSED-03	6/3/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.011
HB-HBSED-03	6/3/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0092	
HB-HBSED-03	6/3/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0092	
HB-HBSED-03	6/3/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0092

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.12a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
Penn-Can Property - Surface Water	METALS																	
	7429-90-5	ALUMINUM	0.108 J	1.33 J	mg/L	HB-HBSW-01	2/6	0.015-0.1	1.33E+00		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	N	BSL
	7440-38-2	ARSENIC	0.003 J	0.0118	mg/L	HB-HBSW-01	3/6	0.0016-0.01	1.18E-02		1.00E-02	4.46E-05	C	4.48E-05	ca	4.46E-05	Y	TOX
	7440-39-3	BARIUM	0.0091 J	0.0958	mg/L	HB-HBSW-01	6/6	-	9.58E-02		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	N	BSL
	7440-41-7	BERYLLIUM	0.00008 J	0.00008 J	mg/L	HB-HBSW-02	1/6	0.000076-0.005	8.00E-05		4.00E-03	7.30E-03	N	7.30E-03	nc	7.30E-03	N	BSL
	7440-70-2	CALCIUM	36.7	196	mg/L	HB-HBSW-01	6/6	-	1.96E+02			NV	NV	NV		NV	N	NUT
	7440-47-3	CHROMIUM ^a	0.0015 J	0.0026 J	mg/L	HB-HBSW-02	3/6	0.01-0.01	2.60E-03		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX
	7440-50-8	COPPER	0.0035 J	0.0035 J	mg/L	HB-HBSW-01	1/6	0.00093-0.02	3.50E-03		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	N	BSL
	57-12-5	CYANIDE	0.0102	0.027	mg/L	HB-HBSW-01	2/5	0.01-0.01	2.70E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL
	7439-89-6	IRON	0.0791 J	11.4 J	mg/L	HB-HBSW-01	6/6	-	1.14E+01		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL
	7439-92-1	LEAD	0.0097	0.0097	mg/L	HB-HBSW-01	1/6	0.00066-0.005	9.70E-03		1.50E-02	NV	NV	NV		1.50E-02	N	BSL
	7439-95-4	MAGNESIUM	4.84 J	24.8	mg/L	HB-HBSW-03	6/6	-	2.48E+01			NV	NV	NV		NV	N	NUT
	7439-96-5	MANGANESE	0.0171	1.8	mg/L	HB-HBSW-01	5/6	0.01-0.01	1.80E+00		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	Y	ASL
	7439-97-6	MERCURY ^b	0.000004	0.00003	mg/L	HB-HBSW-01	3/9	0.00018 - 0.00018	3.32E-05			3.65E-04	N	3.65E-04	nc	3.65E-04	N	BSL
	7440-02-0	NICKEL	0.0012 J	0.0029 J	mg/L	HB-HBSW-01	3/6	0.04-0.04	2.90E-03			7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL
	7440-09-7	POTASSIUM	5.9	8.93	mg/L	HB-HBSW-01	6/6	-	8.93E+00			NV	NV	NV		NV	N	NUT
	7782-49-2	SELENIUM	0.002 J	0.0023 J	mg/L	HB-HBSW-01	3/6	0.01-0.01	2.30E-03		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL
	7440-23-5	SODIUM	53	84.5	mg/L	HB-HBSW-01	6/6	-	8.45E+01			NV	NV	NV		NV	N	NUT
	7440-62-2	VANADIUM	0.00081 J	0.00081 J	mg/L	HB-HBSW-01	1/6	0.00039-0.05	8.10E-04			3.65E-03	N	3.65E-03	nc	3.65E-03	N	BSL
	7440-66-6	ZINC	0.0253	0.0253	mg/L	HB-HBSW-01	1/6	0.002-0.02	2.53E-02		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	SVOCs																	
	105-67-9	2,4-DIMETHYLPHENOL	3 J	3 J	ug/l	HB-HBSW-03	1/6	9.6-10	3.00E+00			7.30E+01	N	7.30E+01	nc	7.30E+01	N	BSL
	91-57-6	2-METHYLNAPHTHALENE	5 J	5 J	ug/l	HB-HBSW-03	1/6	9.6-10	5.00E+00			2.43E+00	N			2.43E+00	Y	ASL
	34METPH	3&4-METHYLPHENOL ^c	1 J	1 J	ug/l	HB-HBSW-03	1/6	9.6-10	1.00E+00			1.83E+01	N	1.82E+01	nc	1.82E+01	N	BSL
	83-32-9	ACENAPHTHENE	23	23	ug/l	HB-HBSW-03	1/6	9.6-10	2.30E+01			3.65E+01	N	3.65E+01	nc	3.65E+01	N	BSL
	208-96-8	ACENAPHTHYLENE	21	21	ug/l	HB-HBSW-03	1/6	9.6-10	2.10E+01			NV	NV	NV		NV	Y	NTX
	65-85-0	BENZOIC ACID	2 J	4 J	ug/l	HB-HBSW-03	2/2	-	4.00E+00			1.46E+04	N	1.46E+04	nc	1.46E+04	N	BSL
	86-74-8	CARBAZOLE	11	11	ug/l	HB-HBSW-03	1/6	9.6-10	1.10E+01			3.35E+00	C	3.36E+00	ca	3.35E+00	Y	ASL
	132-64-9	DIBENZOFURAN	24	24	ug/l	HB-HBSW-03	1/6	9.6-10	2.40E+01			3.65E+00	N	1.22E+00	nc	1.22E+00	Y	ASL
	86-73-7	FLUORENE	19	19	ug/l	HB-HBSW-03	1/6	9.6-10	1.90E+01			2.43E+01	N	2.43E+01	nc	2.43E+01	N	BSL
	91-20-3	NAPHTHALENE	12	330	ug/l	HB-HBSW-03	2/8	1-10	3.30E+02			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	85-01-8	PHENANTHRENE	14	14	ug/l	HB-HBSW-03	1/6	9.6-10	1.40E+01			NV	NV	NV		NV	Y	NTX
	108-95-2	PHENOL	4 J	4 J	ug/l	HB-HBSW-03	2/6	9.6-10	4.00E+00			1.10E+03	N	1.09E+03	nc	1.09E+03	N	BSL

TABLE 2.12a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	VOCs																	
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.5	11 J	ug/l	HB-HBSW-03	2/3	0.5-0.5	1.10E+01			1.46E+00	N	1.23E+00	nc	1.23E+00	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.2 J	0.2 J	ug/l	HB-HBSW-02	1/3	0.5-12	2.00E-01			NV		1.23E+00	nc	1.23E+00	N	BSL
	67-64-1	ACETONE	11 J	14 J	ug/l	HB-HBSW-01	2/6	20-250	1.40E+01			5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	71-43-2	BENZENE	0.8	7 J	ug/l	HB-HBSW-03	2/6	0.5-5	7.00E+00		5.00E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	100-41-4	ETHYLBENZENE	0.1 J	7 J	ug/l	HB-HBSW-03	2/6	0.5-5	7.00E+00		7.00E+02	1.34E+02	N	1.34E+02	nc	1.34E+02	N	BSL
	100-42-5	STYRENE	0.1 J	0.1 J	ug/l	HB-HBSW-02	1/6	0.5-12	1.00E-01		1.00E+02	1.62E+02	N	1.64E+02	nc	1.62E+02	N	BSL
	108-88-3	TOLUENE	2	3 J	ug/l	HB-HBSW-03	2/6	0.5-5	3.00E+00		1.00E+03	2.27E+02	N	7.23E+01	nc	7.23E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	2	12 J	ug/l	HB-HBSW-03	2/6	0.25-5	1.20E+01		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) N/A - No background screening performed.
(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.
(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
= Compound detected in 100% of samples.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for methyl mercury utilized.
c = RBC and PRG values for 4-methylphenol (CAS # 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.12b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - PENN-CAN PROPERTY SURFACE WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HBSW-01	5/8/2001	---	---	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HBSW-01	6/3/2003	---	---	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-01	6/3/2003	---	---	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-01	6/3/2003	---	---	CALCULATED	TOTAL	N	U	ug/l		5
HB-HBSW-02	5/8/2001	---	---	1330-20-7	XYLENES, TOTAL	Y		ug/l	2	2
HB-HBSW-02	6/3/2003	---	---	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-02	6/3/2003	---	---	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-02	6/3/2003	---	---	CALCULATED	TOTAL	N	U	ug/l		5
HB-HBSW-03	5/7/2001	---	---	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	12	12
HB-HBSW-03	6/2/2003	---	---	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-03	6/2/2003	---	---	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-03	6/2/2003	---	---	CALCULATED	TOTAL	N	U	ug/l		5

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.13a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE- RAILROAD AREA SURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
Railroad Area Surface Soil	METALS																	
	7429-90-5	ALUMINUM	4530	13600	mg/kg	HB-XSS-3	19/19	-	1.36E+04			7.82E+03	N	7.61E+03	nc	7.61E+03	Y	ASL
	7440-36-0	ANTIMONY	0.18 J	0.49 J	mg/kg	HB-GP-25	7/19	0.16-9.09	4.90E-01			3.13E+00	N	3.13E+00	nc	3.13E+00	N	BSL
	7440-38-2	ARSENIC	3.3	22.7	mg/kg	HB-TP-29	19/19	-	2.27E+01		1.60E+01	4.26E-01	C	3.90E-02	nc	3.90E-02	Y	TOX
	7440-39-3	BARIUM	18.6 J	879 J	mg/kg	HB-HB-07S	19/19	-	8.79E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	Y	ASL
	7440-41-7	BERYLLIUM	0.35 J	0.8	mg/kg	HB-TP-29	10/19	0.54-0.76	8.00E-01		1.40E+01	1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL
	7440-43-9	CADMIUM	0.034 J	0.15 J	mg/kg	HB-GP-25	3/19	0.027-0.76	1.50E-01		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	N	BSL
	7440-70-2	CALCIUM	6590 J	202000	mg/kg	HB-TP-29	19/19	-	2.02E+05			NV	NV		NV	N	NUT	
	7440-47-3	CHROMIUM ^a	10.1 J	33.2 J	mg/kg	HB-HB-07S	19/19	-	3.32E+01			2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX
	7440-48-4	COBALT	3.4 J	10.4	mg/kg	HB-XSS-3	19/19	-	1.04E+01			NV	N	9.03E+01	nc	9.03E+01	N	BSL
	7440-50-8	COPPER	16	64	mg/kg	HB-GP-27	19/19	-	6.40E+01		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL
	57-12-5	CYANIDE	1	1	mg/kg	HB-HB-07S	1/19	0.54-1.56	1.00E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL
	7439-89-6	IRON	10500 J	20900 J	mg/kg	HB-GP-27	19/19	-	2.09E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL
	7439-92-1	LEAD	6.8 J	849 J	mg/kg	HB-HB-07S	19/19	-	8.49E+02			NV	N	4.00E+02	nc	4.00E+02	Y	ASL
	7439-95-4	MAGNESIUM	2930	107000	mg/kg	HB-HB-08D	19/19	-	1.07E+05		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL
	7439-96-5	MANGANESE	262	522	mg/kg	HB-RISB-09	19/19	-	5.22E+02			7.82E-01	N	6.11E-01	nc	6.11E-01	Y	ASL
	7439-97-6	MERCURY ^b	0.052 J	2	mg/kg	HB-HB-07S	15/19	0.033-0.0368	2.00E+00		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL
	7440-02-0	NICKEL	9.8	24.2	mg/kg	HB-XSS-3	19/19	-	2.42E+01			NV	N	NV		NV	N	NUT
	7440-09-7	POTASSIUM	764	3100	mg/kg	HB-GP-25	19/19	-	3.10E+03		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL
	7782-49-2	SELENIUM	0.33 J	2.3	mg/kg	HB-TP-29	12/19	0.54-3.23	2.30E+00			NV	N	NV		NV	N	NUT
	7440-23-5	SODIUM	102	3000	mg/kg	HB-TP-29	19/19	-	3.00E+03			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL
	7440-62-2	VANADIUM	11.4	30.9	mg/kg	HB-HB-07S	19/19	-	3.09E+01		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL
	7440-66-6	ZINC	23.1	607	mg/kg	HB-HB-07S	19/19	-	6.07E+02									
		PCBs																
		LESS CHLORINATED PCBs ^c	0.003	0.003	mg/kg	HB-GP-25	1/19	0.02-0.2	3.00E-03			5.48E-01	N	3.93E-01	nc	3.93E-01	N	BSL
		HIGHLY CHLORINATED PCBs ^d	0.003	0.07	mg/kg	HB-RISB-09	6/19	0.02-0.2	6.90E-02			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
		TOTAL PCBs ^e	0.003	0.07	mg/kg	HB-RISB-09	6/19	0.02-0.2	6.90E-02			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
		PESTICIDES																
	72-54-8	4,4'-DDD	0.001 J	0.002 J	mg/kg	HB-GP-30	3/19	0.0037-0.05	2.00E-03		2.60E+00	2.66E+00	C	2.44E-01	nc	2.44E-01	N	BSL
	72-55-9	4,4'-DDE	0.002 J	0.02 J	mg/kg	HB-HB-08D	4/19	0.0037-0.01	2.00E-02		1.80E+00	1.88E+00	C	1.72E-01	nc	1.72E-01	N	BSL
	50-29-3	4,4'-DDT	0.0064 J	0.0064 J	mg/kg	HB-RISB-09	1/19	0.0037-0.05	6.40E-03		1.70E+00	1.88E+00	C	1.72E-01	nc	1.72E-01	N	BSL
	60-57-1	DIELDRIN	0.0045 J	0.0045 J	mg/kg	HB-RISB-09	1/19	0.0037-0.05	4.50E-03		3.90E-02	3.99E-02	C	3.04E-03	nc	3.04E-03	Y	ASL
		SVOCS																
	91-57-6	2-METHYLNAPHTHALENE	0.055 J	0.39 J	mg/kg	HB-XSS-1	11/19	0.36-0.43	3.90E-01			3.13E+01	N	NV		3.13E+01	N	BSL
	83-32-9	ACENAPHTHENE	0.053 J	0.083 J	mg/kg	HB-XSS-3	2/19	0.36-0.5	8.30E-02		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL
	208-96-8	ACENAPHTHYLENE	0.061 J	0.2 J	mg/kg	HB-GP-25	7/19	0.36-0.49	2.00E-01		1.00E+02	NV	N	NV		NV	Y	NTX
	120-12-7	ANTHRACENE	0.049 J	0.45	mg/kg	HB-XSS-3	9/19	0.36-0.49	4.50E-01		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	0.057 J	1.1	mg/kg	HB-XSS-3	16/19	0.36-0.4	1.10E+00		1.00E+00	2.20E-01	C	6.21E-02	nc	6.21E-02	Y	ASL
	50-32-8	BENZO(A)PYRENE	0.056 J	0.95	mg/kg	HB-XSS-3	15/19	0.36-0.43	9.50E-01		1.00E+00	2.20E-02	C	6.21E-03	nc	6.21E-03	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.081 J	1.7	mg/kg	HB-GP-25	16/19	0.36-0.4	1.70E+00		1.00E+00	2.20E-01	C	6.21E-02	nc	6.21E-02	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.037 J	0.48	mg/kg	HB-GP-25	15/19	0.36-0.43	4.80E-01		1.00E+02	NV	N	NV		NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.066 J	0.81	mg/kg	HB-XSS-3	13/19	0.36-0.43	8.10E-01		1.00E+00	2.20E+00	C	6.21E-01	nc	6.21E-01	Y	ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.063 J	0.36 J	mg/kg	HB-RISB-09	3/19	0.36-0.5	3.60E-01			4.56E+01	C	3.47E+00	nc	3.47E+00	N	BSL
	85-68-7	BUTYLBENZYL PHTHALATE	0.052 J	0.063 J	mg/kg	HB-GP-25	2/19	0.36-0.51	6.30E-02			1.56E+03	N	1.22E+03	nc	1.22E+03	N	BSL
	86-74-8	CARBAZOLE	0.042 J	0.1 J	mg/kg	HB-XSS-3	6/19	0.36-0.49	1.00E-01			3.19E+01	C	2.43E+00	nc	2.43E+00	N	BSL
	218-01-9	CHRYSENE	0.082 J	1.1	mg/kg	HB-GP-25	16/19	0.36-0.4	1.10E+00		1.00E+00	2.20E+01	C	6.21E+00	nc	6.21E+00	N	BSL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.055 J	0.19 J	mg/kg	HB-XSS-3	7/19	0.36-0.49	1.90E-01		3.30E-01	2.20E-02	C	6.21E-03	nc	6.21E-03	Y	ASL
	132-64-9	DIBENZOFURAN	0.045 J	0.13 J	mg/kg	HB-XSS-1	8/19	0.36-0.49	1.30E-01		1.40E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	N	BSL
	206-44-0	FLUORANTHENE	0.094 J	2	mg/kg	HB-XSS-3	16/19	0.36-0.4	2.00E+00		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	65-85-0	BENZOIC ACID	0.049 J	0.28 J	mg/kg	HB-HB-08D	2/10	1.8-2.5	2.80E-01			3.13E+04	N	1.00E+04	nc	1.00E+04	N	BSL
	100-51-6	BENZYL ALCOHOL	0.28 J	0.28 J	mg/kg	HB-HB-08D	1/19	0.36-0.51	2.80E-01			3.91E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	86-73-7	FLUORENE	0.058 J	0.12 J	mg/kg	HB-XSS-3	2/19	0.36-0.51	1.20E-01		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.055 J	0.47	mg/kg	HB-GP-25	13/19	0.36-0.43	4.70E-01		5.00E-01	2.20E-01	C	6.21E-02	nc	6.21E-02	Y	ASL
	91-20-3	NAPHTHALENE	0.012	0.27 J	mg/kg	HB-XSS-1	12/29	0.005-0.43	2.70E-01		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	N	BSL
	85-01-8	PHENANTHRENE	0.054 J	1.5	mg/kg	HB-XSS-3	16/19	0.36-0.4	1.50E+00		1.00E+02	NV	N	NV		NV	Y	NTX
	129-00-0	PYRENE	0.085 J	1.5	mg/kg	HB-XSS-3	16/19	0.36-0.4	1.50E+00		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL

TABLE 2.13a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE- RAILROAD AREA SURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	VOCs																	
	78-93-3	2-BUTANONE	0.006 J	0.022 J	mg/kg	HB-XSS-1	4/19	0.0093-0.015	2.20E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.004 J	0.37 J	mg/kg	HB-XSS-1	7/19	0.011-0.029	3.70E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.001 J	0.001 J	mg/kg	HB-GP-25	1/19	0.003-0.0098	1.00E-03		2.90E+00	1.16E+01	C	6.43E-02	nc	6.43E-02	Y	TOX
	100-41-4	ETHYLBENZENE	0.0017 J	0.0017 J	mg/kg	HB-XSS-1	1/19	0.003-0.0072	1.70E-03		3.00E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.065	0.065	mg/kg	HB-GP-25	1/19	0.0047-0.021	6.50E-02		5.10E+01	8.52E+01	C	9.11E-01	nc	9.11E-01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.002 J	0.008	mg/kg	HB-HB-09	2/10	0.003-0.004	8.00E-03			NV	NV	NV		NV	Y	NTX
	127-18-4	TETRACHLOROETHENE	0.0005 J	0.0009 J	mg/kg	HB-HB-09	2/19	0.003-0.0098	9.00E-04		5.50E+00	1.18E+00	C	4.84E-02	nc	4.84E-02	N	BSL
	79-01-6	TRICHLOROETHENE	0.0005 J	0.0005 J	mg/kg	HB-GP-30	1/19	0.003-0.0098	5.00E-04		1.00E+01	1.60E+00	C	5.30E-03	nc	5.30E-03	N	BSL
	1330-20-7	XYLENES, TOTAL	0.0008 J	0.0008 J	mg/kg	HB-GP-25	1/19	0.0008-0.0098	8.00E-04		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
(5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.13b
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SOIL (0-2 FT BGS)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-GP-25	0	0.17	2/28/2001	0.01	mg/kg
Highly Chlorinated PCBs	HB-GP-27	0	0.17	3/1/2001	0.003	mg/kg
Highly Chlorinated PCBs	HB-GP-30	0	0.17	3/2/2001	0.006	mg/kg
Highly Chlorinated PCBs	HB-HB-08D	0	0.17	2/27/2001	0.02	mg/kg
Highly Chlorinated PCBs	HB-HB-09	0	0.17	2/26/2001	0.01	mg/kg
Highly Chlorinated PCBs	HB-RISB-09	0	0.5	3/10/2003	0.069	mg/kg
Less Chlorinated PCBs	HB-GP-25	0	0.17	2/28/2001	0.003	mg/kg
Total PCBs	HB-GP-25	0	0.17	2/28/2001	0.013	mg/kg
Total PCBs	HB-GP-27	0	0.17	3/1/2001	0.003	mg/kg
Total PCBs	HB-GP-30	0	0.17	3/2/2001	0.006	mg/kg
Total PCBs	HB-HB-08D	0	0.17	2/27/2001	0.02	mg/kg
Total PCBs	HB-HB-09	0	0.17	2/26/2001	0.01	mg/kg
Total PCBs	HB-RISB-09	0	0.5	3/10/2003	0.07	mg/kg

Notes:

* Less chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.13c
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-GP-25	2/28/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0008	0.0008
HB-GP-26	2/27/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-27	3/1/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-28	2/28/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-29	2/28/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-30	3/2/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-07S	3/2/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-08D	2/27/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-09	2/26/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-TP-29	2/27/2001	2	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-RISB-08	3/4/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.006	
HB-RISB-08	3/4/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.006	
HB-RISB-08	3/4/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.006
HB-RISB-08	3/4/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0047	
HB-RISB-08	3/4/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0047	
HB-RISB-08	3/4/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0047
HB-RISB-09	3/10/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0062	
HB-RISB-09	3/10/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0062	
HB-RISB-09	3/10/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0062
HB-RISB-09	3/10/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0053	
HB-RISB-09	3/10/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0053	
HB-RISB-09	3/10/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0053
HB-XSS-1	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.0098	
HB-XSS-1	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.0098	
HB-XSS-1	12/4/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.0098
HB-XSS-1	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0072	
HB-XSS-1	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0072	
HB-XSS-1	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0072
HB-XSS-2	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0064	
HB-XSS-2	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0064	
HB-XSS-2	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0064
HB-XSS-3	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0059	
HB-XSS-3	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0059	
HB-XSS-3	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0059
HB-XSS-3	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0057	
HB-XSS-3	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0057	
HB-XSS-3	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0057

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.14a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- RAILROAD AREA SUBSURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
Railroad Area - Subsurface Soil	METALS																		
	7429-90-5	ALUMINUM	2830	24900	mg/Kg	HB-TP-30	26/26	-	2.49E+04			7.82E+03	N	7.61E+03	nc	7.61E+03	Y	ASL	
	7440-36-0	ANTIMONY	0.18 J	1.9 J	mg/Kg	HB-TP-24	12/26	0.16-9.09	1.90E+00			3.13E+00	N	3.13E+00	nc	3.13E+00	N	BSL	
	7440-38-2	ARSENIC	2	22.7	mg/Kg	HB-TP-29	26/26	-	2.27E+01		1.60E+01	4.26E-01	C	3.90E-02	nc	3.90E-02	Y	TOX	
	7440-39-3	BARIIUM	18.6 J	879 J	mg/Kg	HB-HB-07S	26/26	-	8.79E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	Y	ASL	
	7440-41-7	BERYLLIUM	0.22 J	1	mg/Kg	HB-TP-30	17/26	0.54-0.76	1.00E+00		1.40E+01	1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL	
	7440-43-9	CADMIUM	0.034 J	1.3	mg/Kg	HB-TP-24	4/26	0.027-0.76	1.30E+00		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	N	BSL	
	7440-70-2	CALCIUM	6590 J	242000	mg/Kg	HB-TP-25	26/26	-	2.42E+05			NV	NV	nc	NV	N	NUT		
	7440-47-3	CHROMIUM ^a	6.1 J	35.2 J	mg/Kg	HB-TP-30	26/26	-	3.52E+01			2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX	
	7440-48-4	COBALT	2.5 J	13.5	mg/Kg	HB-TP-30	26/26	-	1.35E+01			NV	NV	nc	9.03E+01	N	BSL		
	7440-50-8	COPPER	10.4	64.1	mg/Kg	HB-TP-24	26/26	-	6.41E+01		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL	
	57-12-5	CYANIDE	1	1	mg/Kg	HB-HB-07S	1/26	0.54-1.56	1.00E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL	
	7439-89-6	IRON	6250 J	34400 J	mg/Kg	HB-TP-30	26/26	-	3.44E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	6.8 J	849 J	mg/Kg	HB-HB-07S	26/26	-	8.49E+02			NV	NV	nc	4.00E+02	Y	ASL		
	7439-95-4	MAGNESIUM	2930	107000	mg/Kg	HB-HB-08D	26/26	-	1.07E+05			NV	NV	nc	NV	N	NUT		
	7439-96-5	MANGANESE	164	1390	mg/Kg	HB-TP-30	26/26	-	1.39E+03		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL	
	7439-97-6	MERCURY ^b	0.052 J	2	mg/kg	HB-HB-07S	20/26	0.033-0.039	2.00E+00			7.82E-01	N	6.11E-01	nc	6.11E-01	Y	ASL	
	7440-02-0	NICKEL	6	38.9	mg/Kg	HB-TP-30	26/26	-	3.89E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL	
	7440-09-7	POTASSIUM	423 J	4360	mg/Kg	HB-TP-30	26/26	-	4.36E+03			NV	NV	nc	NV	N	NUT		
	7782-49-2	SELENIUM	0.33 J	8.3	mg/Kg	HB-TP-24	19/26	0.54-3.23	8.30E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-22-4	SILVER	0.28 J	0.28 J	mg/Kg	HB-TP-24	1/26	0.079-1.51	2.80E-01		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-23-5	SODIUM	102	3000	mg/Kg	HB-TP-29	26/26	-	3.00E+03			NV	NV	nc	NV	N	NUT		
	7440-62-2	VANADIUM	5.5 J	33.4	mg/Kg	HB-TP-30	26/26	-	3.34E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	23.1	607	mg/Kg	HB-HB-07S	26/26	-	6.07E+02		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL	
	PCBs																		
		LESS CHLORINATED PCBs ^c	0.003	0.003	mg/kg	HB-GP-25	1/26	0.02-0.2	3.00E-03				5.48E-01	N	3.93E-01	nc	3.93E-01	N	BSL
		HIGHLY CHLORINATED PCBs ^d	0.003	0.069	mg/kg	HB-RISB-09	10/26	0.02-0.2	6.90E-02				3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
		TOTAL PCBs ^e	0.003	0.069	mg/kg	HB-RISB-09	10/26	0.02-0.2	6.90E-02				3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
	PESTICIDES																		
	72-54-8	4,4'-DDD	0.001 J	0.01 J	mg/kg	HB-TP-26	6/26	0.0037-0.07	1.00E-02			2.60E+00	2.66E+00	C	2.44E-01	nc	2.44E-01	N	BSL
	72-55-9	4,4'-DDE	0.002 J	0.02 J	mg/kg	HB-HB-08D	5/26	0.0037-0.07	2.00E-02			1.80E+00	1.88E+00	C	1.72E-01	nc	1.72E-01	N	BSL
	50-29-3	4,4'-DDT	0.0064 J	0.01	mg/kg	HB-TP-31	2/26	0.0037-0.07	1.00E-02			1.70E+00	1.88E+00	C	1.72E-01	nc	1.72E-01	N	BSL
	57-74-9	TOTAL CHLORDANE ^f	0.0009 J	0.0009 J	mg/kg	HB-TP-31	1/26	0.0019-0.03	9.00E-04				1.82E+00	C	1.62E+00	ca	1.62E+00	N	BSL
	60-57-1	DIELDRIN	0.0045 J	0.0045 J	mg/kg	HB-RISB-09	1/26	0.0037-0.07	4.50E-03			3.90E-02	3.99E-02	C	3.04E-03	nc	3.04E-03	Y	ASL
	SVOCs																		
	91-57-6	2-METHYLNAPHTHALENE	0.055 J	0.89	mg/kg	HB-TP-28	16/26	0.36-0.46	8.90E-01				3.13E+01	N	NV	nc	3.13E+01	N	BSL
	34METPH	3&4-METHYLPHENOL ^g	0.28 J	0.28 J	mg/kg	HB-TP-24	1/26	0.36-0.53	2.80E-01				3.91E+01	N	3.06E+01	nc	3.06E+01	N	BSL
	83-32-9	ACENAPHTHENE	0.053 J	0.39 J	mg/kg	HB-TP-24	6/26	0.36-0.5	3.90E-01		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL	
	208-96-8	ACENAPHTHYLENE	0.05 J	0.48 J	mg/kg	HB-TP-24	12/26	0.36-0.49	4.80E-01		1.00E+02	NV	NV	nc	NV	Y	NTX		
	120-12-7	ANTHRACENE	0.049 J	1	mg/kg	HB-TP-24	13/26	0.36-0.49	1.00E+00		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL	
	56-55-3	BENZ(A)ANTHRACENE	0.057 J	3.4	mg/kg	HB-TP-24	21/26	0.36-0.46	3.40E+00		1.00E+00	2.20E-01	C	6.21E-02	nc	6.21E-02	Y	ASL	
	50-32-8	BENZO(A)PYRENE	0.056 J	3.7	mg/kg	HB-TP-24	20/26	0.36-0.46	3.70E+00		1.00E+00	2.20E-02	C	6.21E-03	nc	6.21E-03	Y	ASL	
	205-99-2	BENZO(B)FLUORANTHENE	0.081 J	5.2	mg/kg	HB-TP-24	21/26	0.36-0.46	5.20E+00		1.00E+00	2.20E-01	C	6.21E-02	nc	6.21E-02	Y	ASL	
	191-24-2	BENZO(G,H,I)PERYLENE	0.037 J	1.5	mg/kg	HB-TP-24	20/26	0.36-0.46	1.50E+00		1.00E+02	NV	NV	nc	NV	Y	NTX		
	207-08-9	BENZO(K)FLUORANTHENE	0.066 J	1.7	mg/kg	HB-TP-24	18/26	0.36-0.46	1.70E+00		1.00E+00	2.20E+00	C	6.21E-01	nc	6.21E-01	Y	ASL	
	65-85-0	BENZOIC ACID	0.049 J	0.28 J	mg/kg	HB-HB-08D	2/17	1.8-3.3	2.80E-01			3.13E+04	N	1.00E+04	nc	1.00E+04	N	BSL	

TABLE 2.14a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- RAILROAD AREA SUBSURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	100-51-6	BENZYL ALCOHOL	0.28 J	0.28 J	mg/kg	HB-HB-08D	1/26	0.36-0.65	2.80E-01			3.91E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.063 J	0.36 J	mg/kg	HB-RISB-09	4/26	0.36-0.65	3.60E-01			4.56E+01	C	3.47E+00	nc	3.47E+00	N	BSL
	85-68-7	BUTYLBENZYL PHTHALATE	0.052 J	0.063 J	mg/kg	HB-GP-25	2/26	0.36-0.65	6.30E-02			1.56E+03	N	1.22E+03	nc	1.22E+03	N	BSL
	86-74-8	CARBAZOLE	0.042 J	0.98	mg/kg	HB-TP-24	10/26	0.36-0.49	9.80E-01			3.19E+01	C	2.43E+00	nc	2.43E+00	N	BSL
	218-01-9	CHRYSENE	0.082 J	4.2	mg/kg	HB-TP-24	21/26	0.36-0.46	4.20E+00		1.00E+00	2.20E+01	C	6.21E+00	nc	6.21E+00	N	BSL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.055 J	0.41 J	mg/kg	HB-TP-24	11/26	0.36-0.49	4.10E-01		3.30E-01	2.20E-02	C	6.21E-03	nc	6.21E-03	Y	ASL
	132-64-9	DIBENZOFURAN	0.045 J	0.43 J	mg/kg	HB-TP-24	12/26	0.36-0.49	4.30E-01		1.40E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	N	BSL
	84-74-2	DI-N-BUTYL PHTHALATE	0.11 J	0.11 J	mg/kg	HB-TP-24	1/26	0.36-0.53	1.10E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	206-44-0	FLUORANTHENE	0.094 J	9.2	mg/kg	HB-TP-24	21/26	0.36-0.46	9.20E+00		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	86-73-7	FLUORENE	0.058 J	0.64 J	mg/kg	HB-TP-24	5/26	0.36-0.53	6.40E-01		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.055 J	1.5	mg/kg	HB-TP-24	18/26	0.36-0.46	1.50E+00		5.00E-01	2.20E-01	C	6.21E-02	nc	6.21E-02	Y	ASL
	91-20-3	NAPHTHALENE	0.012	0.66	mg/kg	HB-TP-28	18/43	0.005-0.46	6.60E-01		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	N	BSL
	85-01-8	PHENANTHRENE	0.054 J	8.2	mg/kg	HB-TP-24	21/26	0.36-0.46	8.20E+00		1.00E+02	NV	NV	NV	nc	NV	Y	NTX
	108-95-2	PHENOL	0.096 J	0.096 J	mg/kg	HB-TP-24	1/26	0.36-0.53	9.60E-02		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.085 J	8.6	mg/kg	HB-TP-24	21/26	0.36-0.46	8.60E+00		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																	
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.005	0.005	mg/kg	HB-TP-25	1/17	0.003-0.005	5.00E-03		4.70E+01	NV		5.16E+00	nc	5.16E+00	N	BSL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.001 J	0.001 J	mg/kg	HB-TP-25	1/17	0.003-0.005	1.00E-03		4.70E+01	NV		2.13E+00	nc	2.13E+00	N	BSL
	78-93-3	2-BUTANONE	0.006 J	0.022 J	mg/kg	HB-XSS-1	6/26	0.0093-0.016	2.20E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.004 J	0.37 J	mg/kg	HB-XSS-1	8/26	0.011-0.029	3.70E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.001 J	0.026	mg/kg	HB-TP-25	3/26	0.003-0.0098	2.60E-02		2.90E+00	1.16E+01	C	6.43E-02	nc	6.43E-02	Y	TOX
	100-41-4	ETHYLBENZENE	0.0017 J	0.016	mg/kg	HB-TP-25	2/26	0.003-0.0072	1.60E-02		3.00E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.005	0.005	mg/kg	HB-TP-25	1/17	0.003-0.005	5.00E-03			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.065	0.089	mg/kg	HB-TP-26	3/26	0.0047-0.028	8.90E-02		5.10E+01	8.52E+01	C	9.11E-01	nc	9.11E-01	N	BSL
	103-65-1	N-PROPYLBENZENE	0.001 J	0.001 J	mg/kg	HB-TP-25	1/17	0.003-0.005	1.00E-03		1.00E+02	NV	NV	2.40E+01	nc	2.40E+01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.002 J	0.008	mg/kg	HB-HB-09	2/17	0.003-0.005	8.00E-03			NV	NV	NV	nc	NV	Y	NTX
	127-18-4	TETRACHLOROETHENE	0.0005 J	0.0009 J	mg/kg	HB-HB-09	2/26	0.003-0.0098	9.00E-04		5.50E+00	1.18E+00	C	4.84E-02	nc	4.84E-02	N	BSL
	108-88-3	TOLUENE	0.002 J	0.002 J	mg/kg	HB-TP-25	1/26	0.003-0.0098	2.00E-03		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL
	79-01-6	TRICHLOROETHENE	0.0005 J	0.0005 J	mg/kg	HB-GP-30	1/26	0.003-0.0098	5.00E-04		1.00E+01	1.60E+00	C	5.30E-03	nc	5.30E-03	N	BSL
	1330-20-7	XYLENES, TOTAL	0.0008 J	0.008	mg/kg	HB-TP-25	2/26	0.0008-0.0098	8.00E-03		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
f = RBC value for chlordan (CAS# 57749) and PRG value for technical chlordan (CAS# 12789-03-6) utilized.
g = RBC and PRG values for 4-methylphenol (CAS # 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.14b
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE- RAILROAD AREA SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-GP-25	0	0.17	2/28/2001	0.01	mg/kg
Highly Chlorinated PCBs	HB-GP-27	0	0.17	3/1/2001	0.003	mg/kg
Highly Chlorinated PCBs	HB-GP-30	0	0.17	3/2/2001	0.006	mg/kg
Highly Chlorinated PCBs	HB-HB-08D	0	0.17	2/27/2001	0.02	mg/kg
Highly Chlorinated PCBs	HB-HB-09	0	0.17	2/26/2001	0.01	mg/kg
Highly Chlorinated PCBs	HB-RISB-09	0	0.5	3/10/2003	0.069	mg/kg
Highly Chlorinated PCBs	HB-TP-24	2.5	3	2/28/2001	0.03	mg/kg
Highly Chlorinated PCBs	HB-TP-26	3	3	2/26/2001	0.03	mg/kg
Highly Chlorinated PCBs	HB-TP-28	8	8	2/27/2001	0.02	mg/kg
Highly Chlorinated PCBs	HB-TP-31	3	3	2/28/2001	0.007	mg/kg
Less Chlorinated PCBs	HB-GP-25	0	0.17	2/28/2001	0.003	mg/kg
Total PCBs	HB-GP-25	0	0.17	2/28/2001	0.013	mg/kg
Total PCBs	HB-GP-27	0	0.17	3/1/2001	0.003	mg/kg
Total PCBs	HB-GP-30	0	0.17	3/2/2001	0.006	mg/kg
Total PCBs	HB-HB-08D	0	0.17	2/27/2001	0.02	mg/kg
Total PCBs	HB-HB-09	0	0.17	2/26/2001	0.01	mg/kg
Total PCBs	HB-RISB-09	0	0.5	3/10/2003	0.069	mg/kg
Total PCBs	HB-TP-24	2.5	3	2/28/2001	0.03	mg/kg
Total PCBs	HB-TP-26	3	3	2/26/2001	0.03	mg/kg
Total PCBs	HB-TP-28	8	8	2/27/2001	0.02	mg/kg
Total PCBs	HB-TP-31	3	3	2/28/2001	0.007	mg/kg

Notes:

* Less Chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.14c
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-GP-25	2/28/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-25	2/28/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-GP-26	2/27/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-26	2/27/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-GP-27	3/1/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-27	3/1/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-GP-28	2/28/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-28	2/28/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-GP-29	2/28/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-29	2/28/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-GP-30	3/2/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-GP-30	3/2/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-HB-07S	3/2/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-HB-07S	3/2/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-HB-07S	3/2/2001	4	6	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-HB-07S	3/2/2001	4	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-HB-08D	2/27/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.02
HB-HB-08D	2/27/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.02
Total Chlordane =									ND
HB-HB-09	2/26/2001	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-HB-09	2/26/2001	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-RISB-08	3/4/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0019
HB-RISB-08	3/4/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0019
Total Chlordane =									ND
HB-RISB-08	3/4/2003	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-RISB-08	3/4/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-RISB-09	3/10/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0022
HB-RISB-09	3/10/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0022
Total Chlordane =									ND
HB-RISB-09	3/10/2003	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0019
HB-RISB-09	3/10/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0019
Total Chlordane =									ND
HB-TP-24	2/28/2001	2.5	3	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-TP-24	2/28/2001	2.5	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND

TABLE 2.14c
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-TP-25	2/26/2001	8	8	57-74-9	CHLORDANE	N	U	mg/kg	0.02
HB-TP-25	2/26/2001	8	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.02
Total Chlordane =									ND
HB-TP-26	2/26/2001	3	3	57-74-9	CHLORDANE	N	U	mg/kg	0.02
HB-TP-26	2/26/2001	3	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.02
Total Chlordane =									ND
HB-TP-28	2/27/2001	8	8	57-74-9	CHLORDANE	N	U	mg/kg	0.003
HB-TP-28	2/27/2001	8	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.003
Total Chlordane =									ND
HB-TP-29	2/27/2001	2	2	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-TP-29	2/27/2001	2	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-TP-30	2/28/2001	5	5	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-TP-30	2/28/2001	5	5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-TP-31	2/28/2001	3	3	57-74-9	CHLORDANE	Y	J	mg/kg	0.0009
HB-TP-31	2/28/2001	3	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									0.0009
HB-XSS-1	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0053
HB-XSS-1	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0053
Total Chlordane =									ND
HB-XSS-1	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0049
HB-XSS-1	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0049
Total Chlordane =									ND
HB-XSS-2	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0045
HB-XSS-2	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0045
Total Chlordane =									ND
HB-XSS-3	12/4/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0041
HB-XSS-3	12/4/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0041
Total Chlordane =									ND
HB-XSS-3	12/4/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0042
HB-XSS-3	12/4/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0042
Total Chlordane =									ND

TABLE 2.14d
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-GP-25	2/28/2001	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0008	0.0008
HB-GP-26	2/27/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-27	3/1/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-28	2/28/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-29	2/28/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-GP-30	3/2/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-07S	3/2/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-07S	3/2/2001	4	6	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-HB-08D	2/27/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-HB-09	2/26/2001	0	0.17	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-TP-24	2/28/2001	2.5	3	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.005	0.0025
HB-TP-25	2/26/2001	8	8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.008	0.008
HB-TP-26	2/26/2001	3	3	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-TP-28	2/27/2001	8	8	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-TP-29	2/27/2001	2	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.004	0.002
HB-TP-30	2/28/2001	5	5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-TP-31	2/28/2001	3	3	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.003	0.0015
HB-RISB-08	3/4/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.006	
HB-RISB-08	3/4/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.006	
HB-RISB-08	3/4/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.006
HB-RISB-08	3/4/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0047	
HB-RISB-08	3/4/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0047	
HB-RISB-08	3/4/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0047
HB-RISB-09	3/10/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0062	
HB-RISB-09	3/10/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0062	
HB-RISB-09	3/10/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0062
HB-RISB-09	3/10/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0053	
HB-RISB-09	3/10/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0053	
HB-RISB-09	3/10/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0053
HB-XSS-1	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.0098	
HB-XSS-1	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.0098	
HB-XSS-1	12/4/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.0098
HB-XSS-1	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0072	
HB-XSS-1	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0072	
HB-XSS-1	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0072
HB-XSS-2	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0064	
HB-XSS-2	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0064	
HB-XSS-2	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0064
HB-XSS-3	12/4/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0059	
HB-XSS-3	12/4/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0059	
HB-XSS-3	12/4/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0059
HB-XSS-3	12/4/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0057	
HB-XSS-3	12/4/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0057	
HB-XSS-3	12/4/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0057

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.15a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)	
Railroad Area - Shallow Ground Water	METALS																
	7429-90-5	ALUMINUM	0.046 J	15.1 J	mg/L	HB-HB-09	9/9	-	1.51E+01		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00 Y	ASL
	7440-38-2	ARSENIC	0.0079 J	0.0079 J	mg/L	HB-HB-09	1/12	0.0016-0.01	7.90E-03		1.00E-02	4.46E-05	C	4.46E-05	ca	4.46E-05 Y	TOX
	7440-39-3	BARIUM	0.018 J	0.276	mg/L	HB-HB-09	10/12	0.003-0.02	2.76E-01		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01 Y	ASL
	7440-70-2	CALCIUM	59.8	285	mg/L	HB-HB-09	12/12	-	2.85E+02			NV	NV	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	0.0046 J	0.0529	mg/L	HB-HB-09	7/12	0.004-0.01	5.29E-02		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02 Y	TOX
	7440-50-8	COPPER	0.0027 J	0.04	mg/L	HB-HB-09	4/12	0.01-0.02	4.00E-02		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01 N	BSL
	57-12-5	CYANIDE	0.0108	0.0146	mg/L	HB-HB-09	2/12	0.01-0.01	1.46E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02 N	BSL
	7439-89-6	IRON	0.029 J	15 J	mg/L	HB-HB-09	11/12	0.1-0.1	1.50E+01		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00 Y	ASL
	7439-92-1	LEAD	0.0018 J	0.0156	mg/L	HB-HB-09	5/12	0.00066-0.01	1.56E-02		1.50E-02	NV	NV	NV	nc	1.50E-02 Y	ASL
	7439-95-4	MAGNESIUM	14.2	167 J	mg/L	HB-HB-09	12/12	-	1.67E+02			NV	NV	NV	N	NUT	
	7439-96-5	MANGANESE	0.121 J	2.7	mg/L	HB-HB-07S	8/12	0.0087-0.05	2.70E+00		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02 Y	ASL
	7440-02-0	NICKEL	0.0035 J	0.0378 J	mg/L	HB-HB-09	4/12	0.04-0.05	3.78E-02		7.30E-02	7.30E-02	N	7.30E-02	nc	7.30E-02 N	BSL
	7440-09-7	POTASSIUM	1.2 J	21.7 J	mg/L	HB-HB-09	10/12	0.81-2	2.17E+01			NV	NV	NV	N	NUT	
	7782-49-2	SELENIUM	0.0053	0.0059	mg/L	HB-HB-08S	2/12	0.0018-0.05	5.90E-03		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02 N	BSL
	7440-22-4	SILVER	0.0136	0.0136	mg/L	HB-HB-08S	1/12	0.00073-0.01	1.36E-02		1.00E-01	1.83E-02	N	1.82E-02	nc	1.82E-02 N	BSL
	7440-23-5	SODIUM	13.2	2280	mg/L	HB-HB-09	12/12	-	2.28E+03			NV	NV	NV	N	NUT	
	7440-62-2	VANADIUM	0.00073 J	0.0101 J	mg/L	HB-HB-09	4/12	0.05-0.05	1.01E-02			3.65E-03	N	3.65E-03	nc	3.65E-03 Y	ASL
	7440-66-6	ZINC	0.0127 J	0.0477	mg/L	HB-HB-09	6/12	0.02-0.02	4.77E-02		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00 N	BSL
	SVOCs																
	105-67-9	2,4-DIMETHYLPHENOL	1.5 J	1.5 J	ug/l	HB-HB-08S	1/12	9.4-11	1.50E+00			7.30E+01	N	7.30E+01	nc	7.30E+01 N	BSL
	91-57-6	2-METHYLNAPHTHALENE	1.2 J	1.2 J	ug/l	HB-HB-09	1/12	9.4-11	1.20E+00			2.43E+00	N	NV	2.43E+00	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1.2 J	110	ug/l	HB-HB-08S	9/12	10-10	1.10E+02		6.00E+00	4.78E+00	C	4.80E+00	ca	4.78E+00 Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	1 J	1.1 J	ug/l	HB-HB-09	2/12	9.4-11	1.10E+00			3.65E+02	N	3.65E+02	nc	3.65E+02 N	BSL
	87-68-3	HEXACHLOROBUTADIENE	1 J	1 J	ug/l	HB-HB-07S	1/15	1-11	1.00E+00			8.59E-01	C	8.62E-01	ca	8.59E-01 Y	ASL
	91-20-3	NAPHTHALENE	1	15	ug/l	HB-HB-08S	6/15	1-11	1.50E+01			6.51E-01	N	6.20E-01	nc	6.20E-01 Y	ASL
	85-01-8	PHENANTHRENE	1.4 J	1.4 J	ug/l	HB-HB-09	1/12	9.4-11	1.40E+00			NV	NV	NV	Y	NTX	
	108-95-2	PHENOL	1 J	1 J	ug/l	HB-HB-09	1/12	9.4-11	1.00E+00			1.10E+03	N	1.09E+03	nc	1.09E+03 N	BSL
	VOCs																
	87-61-6	1,2,3-TRICHLOROBENZENE	1 J	1 J	ug/l	HB-HB-07S	1/3	1-1	1.00E+00			NV	NV	NV	Y	NTX	
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.1 J	0.1 J	ug/l	HB-HB-08S	1/3	0.5-0.5	1.00E-01			1.46E+00	N	1.23E+00	nc	1.23E+00 N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.12 J	0.12 J	ug/l	HB-HB-07S	1/15	0.5-10	1.20E-01		7.50E+01	2.81E-01	C	5.02E-01	ca	2.81E-01 N	BSL
	78-93-3	2-BUTANONE	4.9 J	4.9 J	ug/l	HB-HB-07S	1/12	10-10	4.90E+00			6.97E+02	N	6.97E+02	nc	6.97E+02 N	BSL
	67-64-1	ACETONE	3 J	79	ug/l	HB-HB-07S	3/12	10-20	7.90E+01			5.48E+02	N	5.48E+02	nc	5.48E+02 N	BSL
	71-43-2	BENZENE	2.15	2.15	ug/l	HB-HB-08S	1/12	0.5-5	2.15E+00		5.00E+00	3.36E-01	C	3.54E-01	ca	3.36E-01 Y	TOX
	100-41-4	ETHYLBENZENE	0.34 J	0.34 J	ug/l	HB-HB-08S	1/12	0.5-5	3.40E-01			1.34E+02	N	1.34E+02	nc	1.34E+02 N	BSL
	100-42-5	STYRENE	0.62	0.62	ug/l	HB-HB-08S	1/12	0.5-5	6.20E-01		1.00E+02	1.62E+02	N	1.64E+02	nc	1.62E+02 N	BSL
	108-88-3	TOLUENE	0.2 J	0.72	ug/l	HB-HB-08S	2/12	0.5-5	7.20E-01		1.00E+03	2.27E+02	N	7.23E+01	nc	7.23E+01 N	BSL
	1330-20-7	XYLENES, TOTAL	0.2 J	1.23	ug/l	HB-HB-08S	2/12	0.25-5	1.23E+00		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01 N	BSL

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.

(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.

(6) USEPA Region 3 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.

(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.

(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = RBC and PRG values for chromium VI utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NUT: Compound is an essential nutrient and not screened in

NV: No Value

PRG: Preliminary Remediation Goals, USEPA, 2004

RBC: Risk Based Concentration; USEPA, October, 2007

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.15b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SHALLOW GROUND WATER (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-07S	5/10/2001	3	8	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-07S	5/19/2003	3	8	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-07S	5/19/2003	3	8	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-07S	5/19/2003	3	8	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-07S	8/22/2003	3	8	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-07S	8/22/2003	3	8	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-07S	8/22/2003	3	8	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-07S	3/19/2007	3	8	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-HB-08S	5/11/2001	5	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.2	0.2
HB-HB-08S	5/19/2003	5	10	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-08S	5/19/2003	5	10	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-08S	5/19/2003	5	10	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-08S	8/26/2003	5	10	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-08S	8/26/2003	5	10	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-08S	8/26/2003	5	10	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-08S	3/19/2007	5	10	1330-20-7	XYLENES, TOTAL	Y		ug/l	1.23	1.23
HB-HB-09	5/10/2001	5	15	1330-20-7	XYLENES, TOTAL	N	U	ug/l	0.5	0.25
HB-HB-09	5/19/2003	5	15	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-09	5/19/2003	5	15	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-09	5/19/2003	5	15	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-09	8/22/2003	5	15	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HB-09	8/22/2003	5	15	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HB-09	8/22/2003	5	15	CALCULATED	TOTAL	N	U	ug/l		5
HB-HB-09S	3/19/2007	4.96	14.96	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.16a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
Railroad Area - Surface Sediment	DIOXIN/FURAN (8)																	
	1746-01-6	2,3,7,8-TCDD Equivalent	0.00000019	0.000034	mg/kg	HB-HBSED-05	6/6		3.40E-05			4.26E-06	C	3.90E-06	ca	3.90E-06	Y	ASL
	METALS																	
	7429-90-5	ALUMINUM	1650	7330 J	mg/kg	HB-HBSED-04	6/6	-	7.33E+03			7.82E+03	N	7.61E+03	nc	7.61E+03	N	BSL
	7440-36-0	ANTIMONY	0.87 J	0.87 J	mg/kg	HB-HBSED-05	1/6	0.24-12.7	8.70E-01			3.13E+00	N	3.13E+00	nc	3.13E+00	N	BSL
	7440-38-2	ARSENIC	3.8	9.6 J	mg/kg	HB-HBSED-04	6/6	-	9.60E+00			4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX
	7440-39-3	BARIUM	36.4 J	120 J	mg/kg	HB-HBSED-05	6/6	-	1.20E+02			1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL
	7440-41-7	BERYLLIUM	0.22 J	0.41 J	mg/kg	HB-HBSED-05	2/6	0.81-1.1	4.10E-01			1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL
	7440-43-9	CADMIUM	1.1 J	1.3 J	mg/kg	HB-HBSED-05	3/6	0.16-1	1.30E+00			3.91E+00	N	3.70E+00	nc	3.70E+00	N	BSL
	7440-70-2	CALCIUM	77900 J	312000	mg/kg	HB-HBSED-04	6/6	-	3.12E+05			NV	NV	NV	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	9	41.3 J	mg/kg	HB-HBSED-05	6/6	-	4.13E+01			2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX
	7440-48-4	COBALT	1.6 J	4.8 J	mg/kg	HB-HBSED-05	2/6	8.1-10.6	4.80E+00			NV	9.03E-01	nc	9.03E-01	N	BSL	
	7440-50-8	COPPER	6	147 J	mg/kg	HB-HBSED-05	6/6	-	1.47E+02			3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL
	57-12-5	CYANIDE	5.39 J	5.39 J	mg/kg	HB-HBSED-04	1/6	0.86-2.16	5.39E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL
	7439-89-6	IRON	2900	17400 J	mg/kg	HB-HBSED-05	6/6	-	1.74E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL
	7439-92-1	LEAD	16.6	201 J	mg/kg	HB-HBSED-05	6/6	-	2.01E+02			NV	4.00E+02	nc	4.00E+02	N	BSL	
	7439-95-4	MAGNESIUM	16100 J	35300 J	mg/kg	HB-HBSED-04	6/6	-	3.53E+04			NV	NV	NV	NV	N	NUT	
	7439-96-5	MANGANESE	33.1	305 J	mg/kg	HB-HBSED-05	6/6	-	3.05E+02			1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL
	7439-97-6	MERCURY ^b	0.054 J	6.5	mg/kg	HB-HBSED-05	6/6	-	6.50E+00			7.82E-01	N	6.11E-01	nc	6.11E-01	Y	ASL
	7440-02-0	NICKEL	4.5 J	21.1 J	mg/kg	HB-HBSED-05	6/6	-	2.11E+01			1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL
	7440-09-7	POTASSIUM	167 J	1490 J	mg/kg	HB-HBSED-05	4/6	323-401	1.49E+03			NV	NV	NV	NV	N	NUT	
	7782-49-2	SELENIUM	1.9 J	3 J	mg/kg	HB-HBSED-05	3/6	0.31-2	3.00E+00			3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL
	7440-22-4	SILVER	0.52 J	0.52 J	mg/kg	HB-HBSED-05	1/6	0.12-2.1	5.20E-01			3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL
	7440-23-5	SODIUM	677 J	5730 J	mg/kg	HB-HBSED-05	6/6	-	5.73E+03			NV	NV	NV	NV	N	NUT	
	7440-62-2	VANADIUM	6.2 J	23.1 J	mg/kg	HB-HBSED-05	6/6	-	2.31E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL
	7440-66-6	ZINC	21.5 J	269 J	mg/kg	HB-HBSED-05	6/6	-	2.69E+02			2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL
PCBs																		
		HIGHLY CHLORINATED PCBs ^c	0.01	0.86	mg/kg	HB-HBSED-05	2/6	0.03-0.14	8.60E-01			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
		TOTAL PCBs ^d	0.01	0.86	mg/kg	HB-HBSED-05	2/6	0.03-0.14	8.60E-01			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
PESTICIDES																		
	72-54-8	4,4'-DDD	0.045 J	0.045 J	mg/kg	HB-HBSED-05	1/6	0.006-0.09	4.50E-02			2.66E+00	C	2.44E+00	ca	2.44E+00	N	BSL
	72-55-9	4,4'-DDE	0.004 J	0.004 J	mg/kg	HB-HBSED-04	1/6	0.028-0.09	4.00E-03			1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL
	57-74-9	TOTAL CHLORDANE ^e	0.010	0.010	mg/kg	HB-HBSED-05	1/6	0.003-0.018	1.00E-02			1.82E+00	C	1.62E+00	ca	1.62E+00	N	BSL
SVOCs																		
	91-57-6	2-METHYLNAPHTHALENE	0.11 J	0.13	mg/kg	HB-HBSED-04	3/6	0.57-7.1	1.30E-01			3.13E+01	N	NV		3.13E+01	N	BSL
	83-32-9	ACENAPHTHENE	0.058	1.2 J	mg/kg	HB-HBSED-05	4/6	0.57-0.7	1.20E+00			4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL
	208-96-8	ACENAPHTHYLENE	0.064	2.1 J	mg/kg	HB-HBSED-05	4/6	0.57-0.7	2.10E+00			NV	NV	NV	NV	Y	NTX	
	120-12-7	ANTHRACENE	0.1	4 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	4.00E+00			2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	0.39 J	13 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	1.30E+01			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	50-32-8	BENZO(A)PYRENE	0.4 J	14 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	1.40E+01			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.063 J	12 J	mg/kg	HB-HBSED-05	6/6	-	1.20E+01			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.29 J	10 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	1.00E+01			NV	NV	NV	NV	Y	NTX	
	207-08-9	BENZO(K)FLUORANTHENE	0.37 J	11 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	1.10E+01			2.20E+00	C	6.21E+00	ca	2.20E+00	Y	ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.061	6.7 J	mg/kg	HB-HBSED-05	3/6	0.57-1.9	6.70E+00			4.56E+01	C	3.47E+01	ca	3.47E+01	N	BSL
	85-68-7	BUTYLBENZYL PHTHALATE	0.21 J	0.21 J	mg/kg	HB-HBSED-05	1/6	0.55-7.1	2.10E-01			1.56E+03	N	1.22E+03	nc	1.22E+03	N	BSL
	86-74-8	CARBAZOLE	0.063	1.6 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	1.60E+00			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL
	218-01-9	CHRYSENE	0.059 J	15 J	mg/kg	HB-HBSED-05	6/6	-	1.50E+01			2.20E+01	C	6.21E+01	ca	2.20E+01	N	BSL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.087 J	3.2 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	3.20E+00			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL

TABLE 2.16a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
	132-64-9	DIBENZOFURAN	0.081	0.78 J	mg/kg	HB-HBSED-05	4/6	0.57-5.9	7.80E-01			7.82E+00	N	1.45E+01	nc	7.82E+00	N	BSL
	84-74-2	DI-N-BUTYL PHTHALATE	0.059	0.059	mg/kg	HB-HBSED-04	1/6	0.57-7.1	5.90E-02			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	206-44-0	FLUORANTHENE	0.089 J	31 J	mg/kg	HB-HBSED-05	6/6	-	3.10E+01			3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	86-73-7	FLUORENE	0.46 J	1.5 J	mg/kg	HB-HBSED-05	3/6	0.55-0.7	1.50E+00			3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.25 J	9 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	9.00E+00			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.14	0.18 J	mg/kg	HB-HBSED-04	3/8	0.009-7.1	1.80E-01			1.56E+02	N	5.59E+00	nc	5.59E+00	N	BSL
	85-01-8	PHENANTHRENE	0.54	15 J	mg/kg	HB-HBSED-05	5/6	0.57-0.57	1.50E+01			NV	NV	NV	NV	Y	NTX	
	129-00-0	PYRENE	0.079 J	24 J	mg/kg	HB-HBSED-05	6/6	-	2.40E+01			2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																	
	106-46-7	1,4-DICHLOROBENZENE	0.007 J	0.11	mg/kg	HB-HBSED-04	3/8	0.004-7.1	1.10E-01			2.66E+01	C	3.45E+00	ca	3.45E+00	N	BSL
	78-93-3	2-BUTANONE	0.012 J	0.02	mg/kg	HB-HBSED-05	3/8	0.017-0.022	2.00E-02			4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.006 J	0.086	mg/kg	HB-HBSED-05	7/8	0.042-0.042	8.60E-02			7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	75-15-0	CARBON DISULFIDE	0.0018 J	0.0022	mg/kg	HB-HBSED-05	2/5	0.017-0.022	2.20E-03			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL
	108-88-3	TOLUENE	0.002 J	0.002 J	mg/kg	HB-HBSED-05	1/6	0.004-0.011	2.00E-03			6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC
(5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG
(6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(8) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.16b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.16b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-04	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	5.63	5.63	ng/kg	J	0.01	0.056
HB-HBSED-04	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	5	2.5	ng/kg	UJ	0.01	0.025
HB-HBSED-04	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-04	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-04	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-04	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-04	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-04	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-04	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	5	2.5	ng/kg	UJ	1	2.500
HB-HBSED-04	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	5	2.5	ng/kg	UJ	0.03	0.075
HB-HBSED-04	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-HBSED-04	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.3	1.3	ng/kg	J	0.1	0.130
HB-HBSED-04	5/11/2001	0	0.5	3268-87-9	OCDD	Y	177	177	ng/kg	J	0.0003	0.053
HB-HBSED-04	5/11/2001	0	0.5	39001-02-0	OCDF	Y	13.1	13.1	ng/kg	J	0.0003	0.004
Sample Location TEQ = 4.8												
HB-HBSED-04	6/4/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	1.118	1.118	ng/kg	J	0.01	0.011
HB-HBSED-04	6/4/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.203	0.1015	ng/kg	UJ	0.01	0.001
HB-HBSED-04	6/4/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.098	0.049	ng/kg	U	0.1	0.005
HB-HBSED-04	6/4/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.181	0.181	ng/kg	J	0.1	0.018
HB-HBSED-04	6/4/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.078	0.039	ng/kg	U	0.1	0.004
HB-HBSED-04	6/4/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.094	0.047	ng/kg	U	0.1	0.005
HB-HBSED-04	6/4/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.09	0.045	ng/kg	U	0.1	0.005
HB-HBSED-04	6/4/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.082	0.041	ng/kg	UJ	1	0.041
HB-HBSED-04	6/4/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.155	0.155	ng/kg	J	0.03	0.005
HB-HBSED-04	6/4/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.049	0.0245	ng/kg	U	1	0.025
HB-HBSED-04	6/4/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.815	0.815	ng/kg	J	0.1	0.082
HB-HBSED-04	6/4/2003	0	0.5	3268-87-9	OCDD	Y	51.526	51.526	ng/kg	J	0.0003	0.015
Sample Location TEQ = 0.2												
HB-HBSED-04	6/4/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	0.391	0.391	ng/kg	J	0.01	0.004
HB-HBSED-04	6/4/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.243	0.1215	ng/kg	UJ	0.01	0.001
HB-HBSED-04	6/4/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.16	0.08	ng/kg	UJ	0.1	0.008
HB-HBSED-04	6/4/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.15	0.075	ng/kg	UJ	0.1	0.008
HB-HBSED-04	6/4/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.119	0.0595	ng/kg	UJ	0.1	0.006
HB-HBSED-04	6/4/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.153	0.0765	ng/kg	UJ	0.1	0.008
HB-HBSED-04	6/4/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.148	0.074	ng/kg	UJ	0.1	0.007
HB-HBSED-04	6/4/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	0.163	0.0815	ng/kg	UJ	1	0.082
HB-HBSED-04	6/4/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	0.09	0.045	ng/kg	UJ	0.03	0.001
HB-HBSED-04	6/4/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.122	0.061	ng/kg	UJ	1	0.061
HB-HBSED-04	6/4/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	N	0.093	0.0465	ng/kg	UJ	0.1	0.005
HB-HBSED-04	6/4/2003	0.5	1	3268-87-9	OCDD	Y	11.531	11.531	ng/kg	J	0.0003	0.003
Sample Location TEQ = 0.2												
HB-HBSED-05	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	199	199	ng/kg	J	0.01	1.990
HB-HBSED-05	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	11.8	11.8	ng/kg	J	0.01	0.118
HB-HBSED-05	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	19.5	19.5	ng/kg	J	0.1	1.950
HB-HBSED-05	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	25.4	25.4	ng/kg	J	0.1	2.540
HB-HBSED-05	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	47.9	47.9	ng/kg	J	0.1	4.790
HB-HBSED-05	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	16.3	16.3	ng/kg	J	0.1	1.630
HB-HBSED-05	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	47.9	47.9	ng/kg	J	0.1	4.790
HB-HBSED-05	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-05	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	11.9	11.9	ng/kg	J	1	11.900
HB-HBSED-05	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	7.01	7.01	ng/kg	J	0.03	0.210
HB-HBSED-05	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.17	2.17	ng/kg	J	1	2.170
HB-HBSED-05	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	7.22	7.22	ng/kg	J	0.1	0.722
HB-HBSED-05	5/11/2001	0	0.5	3268-87-9	OCDD	Y	2760	2760	ng/kg	J	0.0003	0.828
HB-HBSED-05	5/11/2001	0	0.5	39001-02-0	OCDF	Y	446	446	ng/kg	J	0.0003	0.134
Sample Location TEQ = 34.0												

TABLE 2.16b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-05	6/4/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	75.204	75.204	ng/kg	J	0.01	0.752
HB-HBSED-05	6/4/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.251	4.251	ng/kg	J	0.01	0.043
HB-HBSED-05	6/4/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.378	6.378	ng/kg	J	0.1	0.638
HB-HBSED-05	6/4/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	10.68	10.68	ng/kg	EMPC	0.1	1.068
HB-HBSED-05	6/4/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	15.321	15.321	ng/kg	J	0.1	1.532
HB-HBSED-05	6/4/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	4.837	4.837	ng/kg	J	0.1	0.484
HB-HBSED-05	6/4/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	16.502	16.502	ng/kg	J	0.1	1.650
HB-HBSED-05	6/4/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.7	0.35	ng/kg	UJ	0.1	0.035
HB-HBSED-05	6/4/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	3.803	3.803	ng/kg	J	1	3.803
HB-HBSED-05	6/4/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	3.234	3.234	ng/kg	EMPC	0.03	0.097
HB-HBSED-05	6/4/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	0.963	0.963	ng/kg	J	1	0.963
HB-HBSED-05	6/4/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.628	2.628	ng/kg	J	0.1	0.263
HB-HBSED-05	6/4/2003	0	0.5	3268-87-9	OCDD	Y	3179.123	3179.123	ng/kg	J	0.0003	0.954
HB-HBSED-05	6/4/2003	0	0.5	39001-02-0	OCDF	Y	129.141	129.141	ng/kg	J	0.0003	0.039
Sample Location TEQ = 12.3												
HB-HBSED-05	6/4/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	85.328	85.328	ng/kg		0.01	0.853
HB-HBSED-05	6/4/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.541	4.541	ng/kg	J	0.01	0.045
HB-HBSED-05	6/4/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	5.695	5.695	ng/kg		0.1	0.570
HB-HBSED-05	6/4/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	14.386	14.386	ng/kg	EMPC	0.1	1.439
HB-HBSED-05	6/4/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	17.703	17.703	ng/kg		0.1	1.770
HB-HBSED-05	6/4/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.523	7.523	ng/kg		0.1	0.752
HB-HBSED-05	6/4/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	14.866	14.866	ng/kg		0.1	1.487
HB-HBSED-05	6/4/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.569	0.2845	ng/kg	U	0.1	0.028
HB-HBSED-05	6/4/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	4.497	4.497	ng/kg		1	4.497
HB-HBSED-05	6/4/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	3.894	3.894	ng/kg	EMPC	0.03	0.117
HB-HBSED-05	6/4/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	0.819	0.819	ng/kg	J	1	0.819
HB-HBSED-05	6/4/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	3.39	3.39	ng/kg		0.1	0.339
HB-HBSED-05	6/4/2003	0.5	1	3268-87-9	OCDD	Y	2209.454	2209.454	ng/kg		0.0003	0.663
HB-HBSED-05	6/4/2003	0.5	1	39001-02-0	OCDF	Y	146.351	146.351	ng/kg		0.0003	0.044
Sample Location TEQ = 13.4												

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 EMPC = Estimated Maximum Possible Concentration
 N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223-241.

TABLE 2.16c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-HBSED-04	0	0.5	5/8/2001	0.01	mg/kg
Highly Chlorinated PCBs	HB-HBSED-05	0	0.5	5/8/2001	0.86	mg/kg
Total PCBs	HB-HBSED-04	0	0.5	5/8/2001	0.01	mg/kg
Total PCBs	HB-HBSED-05	0	0.5	5/8/2001	0.86	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.16d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HBSED-04	5/8/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.003
HB-HBSED-04	5/8/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.003
Total Chlordane =									ND
HB-HBSED-04	6/4/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.014
HB-HBSED-04	6/4/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.014
Total Chlordane =									ND
HB-HBSED-04	6/4/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.018
HB-HBSED-04	6/4/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.018
Total Chlordane =									ND
HB-HBSED-05	5/8/2001	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.04
HB-HBSED-05	5/8/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.01
Total Chlordane =									0.01
HB-HBSED-05	6/4/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.018
HB-HBSED-05	6/4/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.018
Total Chlordane =									ND
HB-HBSED-05	6/4/2003	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.015
HB-HBSED-05	6/4/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.015
Total Chlordane =									ND

TABLE 2.17a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
Railroad Area - Surface Water	METALS																	
	7429-90-5	ALUMINUM	0.11 J	2.13	mg/L	HB-HBSW-05	4/4	-	2.13E+00		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	N	BSL
	7440-38-2	ARSENIC	0.003 J	0.003 J	mg/L	HB-HBSW-05	1/4	0.01-0.01	3.00E-03		1.00E-02	4.46E-05	C	4.46E-05	ca	4.46E-05	Y	TOX
	7440-39-3	BARIUM	0.0698	0.0911	mg/L	HB-HBSW-05	3/4	0.02-0.02	9.11E-02		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	N	BSL
	7440-70-2	CALCIUM	56.8	186	mg/L	HB-HBSW-05	4/4	-	1.86E+02			NV	NV	NV	N	NV	N	NUT
	7440-47-3	CHROMIUM ^a	0.0043 J	0.0043 J	mg/L	HB-HBSW-05	1/4	0.01-0.01	4.30E-03		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX
	7440-50-8	COPPER	0.0116 J	0.0225	mg/L	HB-HBSW-05	2/4	0.02-0.02	2.25E-02		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	N	BSL
	57-12-5	CYANIDE	0.012	0.012	mg/L	HB-HBSW-05	1/4	0.01-0.01	1.20E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL
	7439-89-6	IRON	0.155 J	3.7	mg/L	HB-HBSW-05	4/4	-	3.70E+00		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL
	7439-92-1	LEAD	0.0085	0.0363	mg/L	HB-HBSW-05	3/4	0.005-0.005	3.63E-02		1.50E-02	NV	NV	NV	Y	1.50E-02	Y	ASL
	7439-95-4	MAGNESIUM	27.2	47.6	mg/L	HB-HBSW-04	4/4	-	4.76E+01			NV	NV	NV	N	NV	N	NUT
	7439-96-5	MANGANESE	0.138	0.379	mg/L	HB-HBSW-05	3/4	0.01-0.01	3.79E-01		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	Y	ASL
	7439-97-6	MERCURY ^b	0.00003	0.00005	mg/L	HB-HBSW-05	2/6	0.00018 - 0.0002	4.76E-05			3.65E-04	N	3.65E-04	nc	3.65E-04	N	BSL
	7440-02-0	NICKEL	0.0018 J	0.0018 J	mg/L	HB-HBSW-05	1/4	0.04-0.04	1.80E-03			7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL
	7440-09-7	POTASSIUM	3.73	12	mg/L	HB-HBSW-05	4/4	-	1.20E+01			NV	NV	NV	N	NV	N	NUT
	7782-49-2	SELENIUM	0.0026 J	0.0026 J	mg/L	HB-HBSW-05	1/4	0.005-0.01	2.60E-03		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL
	7440-23-5	SODIUM	63.4	902	mg/L	HB-HBSW-05	4/4	-	9.02E+02			NV	NV	NV	N	NV	N	NUT
	7440-62-2	VANADIUM	0.0013 J	0.0013 J	mg/L	HB-HBSW-05	1/4	0.05-0.05	1.30E-03			3.65E-03	N	3.65E-03	nc	3.65E-03	N	BSL
	7440-66-6	ZINC	0.0223	0.0685	mg/L	HB-HBSW-05	3/4	0.02-0.02	6.85E-02		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	SVOCs																	
	56-55-3	BENZ(A)ANTHRACENE	1.6 J	1.6 J	ug/l	HB-HBSW-05	1/4	9.4-11	1.60E+00		2.00E-01	3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	50-32-8	BENZO(A)PYRENE	2 J	2 J	ug/l	HB-HBSW-05	1/4	9.4-11	2.00E+00			3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	1.6 J	1.6 J	ug/l	HB-HBSW-05	1/4	9.4-11	1.60E+00			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	1.9 J	1.9 J	ug/l	HB-HBSW-05	1/4	9.4-11	1.90E+00			NV	NV	NV	Y	NV	Y	NTX
	207-88-9	BENZO(K)FLUORANTHENE	1.6 J	1.6 J	ug/l	HB-HBSW-05	1/4	9.4-11	1.60E+00			3.00E-01	C	9.21E-01	ca	3.00E-01	Y	ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	5.2 J	5.2 J	ug/l	HB-HBSW-05	1/4	9.4-11	5.20E+00		6.00E+00	4.78E+00	C	4.80E+00	ca	4.78E+00	Y	ASL
	218-01-9	CHRYSENE	1.1 J	2 J	ug/l	HB-HBSW-05	2/4	9.4-11	2.00E+00			3.00E+00	C	9.21E+00	ca	3.00E+00	N	BSL
	206-44-0	FLUORANTHENE	1.7 J	3.7 J	ug/l	HB-HBSW-05	2/4	9.4-11	3.70E+00			1.46E+02	N	1.46E+02	nc	1.46E+02	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	1.4 J	1.4 J	ug/l	HB-HBSW-05	1/4	9.4-11	1.40E+00			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	91-20-3	NAPHTHALENE	4	4	ug/l	HB-HBSW-05	1/5	9.4-11	4.00E+00			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	85-01-8	PHENANTHRENE	1.3 J	1.3 J	ug/l	HB-HBSW-05	1/4	9.4-11	1.30E+00			NV	NV	NV	Y	NV	Y	NTX
	129-00-0	PYRENE	1.6 J	3 J	ug/l	HB-HBSW-05	2/4	9.4-11	3.00E+00			1.83E+01	N	1.83E+01	nc	1.83E+01	N	BSL
	VOCs																	
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.2 J	0.2 J	ug/l	HB-HBSW-05	1/1	-	2.00E-01			1.46E+00	N	1.23E+00	nc	1.23E+00	N	BSL
	71-43-2	BENZENE	0.3 J	0.3 J	ug/l	HB-HBSW-05	1/4	5-5	3.00E-01		5.00E+00	3.36E-01	C	3.36E-01	ca	3.36E-01	Y	TOX
	108-88-3	TOLUENE	0.8	0.8	ug/l	HB-HBSW-05	1/4	5-5	8.00E-01		1.00E+03	2.27E+02	N	7.23E+01	nc	7.23E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	0.8	0.8	ug/l	HB-HBSW-05	1/4	5-5	8.00E-01		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
- (2) Concentration used for screening is the maximum detected concentration.
- (3) N/A - No background screening performed.
- (4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.
- (5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
- (6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
- (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
- (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = RBC and PRG values for chromium VI utilized.

b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.17b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - RAILROAD AREA SURFACE WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HBSW-04	6/4/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-04	6/4/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-04	6/4/2003			1330-20-7	TOTAL	N	U	ug/l		5
HB-HBSW-05	5/8/2001			1330-20-7	XYLENES, TOTAL	Y		ug/l	0.8	0.8
HB-HBSW-05	6/4/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-05	6/4/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-05	6/4/2003			1330-20-7	TOTAL	N	U	ug/l		5
HB-HBSW-05	9/9/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-05	9/9/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-05	9/9/2003			1330-20-7	TOTAL	N	U	ug/l		5

Notes:

a - Total Xylene value utilized in the risk assessment.

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)	
Harbor Brook Surface Sediment	DIOXIN/FURAN (8)																
	1746-01-6	2,3,7,8-TCDD Equivalent	0.0000007	0.0000406	mg/Kg	HB-T-2-3	14/14		4.06E-05			4.26E-06	C	3.90E-06	ca	3.90E-06 Y ASL	
	METALS																
	7429-90-5	ALUMINUM	2210 J	5890	mg/kg	HB-CSXSED-2	26/26	-	5.89E+03			7.82E+03	N	7.61E+03	nc	7.61E+03 N BSL	
	7440-36-0	ANTIMONY	0.55 J	0.99 J	mg/kg	HB-H3	3/32	0.36-11.4	9.90E-01			3.13E+00	N	3.13E+00	nc	3.13E+00 N BSL	
	7440-38-2	ARSENIC	1.6	6.5	mg/kg	HB-T-1-3, HB-T-4-1	27/29	1.05-1.5	6.50E+00			4.26E-01	C	3.90E-01	ca	3.90E-01 Y TOX	
	7440-39-3	BARIUM	21.4 J	327 J	mg/kg	HB-H2	32/32	-	3.27E+02			1.56E+03	N	5.37E+02	nc	5.37E+02 N BSL	
	7440-41-7	BERYLLIUM	0.1 J	1.9	mg/kg	HB-T-2-1	15/32	0.08-0.95	1.90E+00			1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL	
	7440-43-9	CADMIUM	0.27 J	19.2	mg/kg	HB-T-3-3	26/32	0.72-0.95	1.92E+01			3.91E+00	N	3.70E+00	nc	3.70E+00 Y ASL	
	7440-70-2	CALCIUM	72900	340000	mg/kg	HB-HBSED-15	26/26	-	3.40E+05			NV	NV	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	7	211	mg/kg	HB-T-5-1	26/26	-	2.11E+02			2.35E+01	N	3.01E+01	ca	2.35E+01 Y TOX	
	7440-48-4	COBALT	0.97 J	13.8 J	mg/kg	HB-T-4-2	24/32	6.3-9.5	1.38E+01			NV	9.03E+02	ca	9.03E+02 N BSL		
	7440-50-8	COPPER	20.1	308	mg/kg	HB-CSXSED-2	24/24	-	3.08E+02			3.13E+02	N	3.13E+02	nc	3.13E+02 N BSL	
	57-12-5	CYANIDE	3.43	4.76	mg/kg	HB-HBSED-15	2/31	0.67-2.51	4.76E+00			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL	
	7439-89-6	IRON	4160	21200	mg/kg	HB-CSXSED-2	26/26	-	2.12E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL	
	7439-92-1	LEAD	43.7	479	mg/kg	HB-T-4-1	22/22	-	4.79E+02			NV	4.00E+02	nc	4.00E+02 Y ASL		
	7439-95-4	MAGNESIUM	6620	59100	mg/kg	HB-T-5-2	26/26	-	5.91E+04			NV	NV	NV	N	NUT	
	7439-96-5	MANGANESE	153	366	mg/kg	HB-HBSED-14	26/26	-	3.66E+02			1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL	
	7439-97-6	MERCURY ^b	0.08	52	mg/kg	HB-T-5-3	25/26	0.04-0.04	5.20E+01			7.82E-01	N	6.11E-01	nc	6.11E-01 Y ASL	
	7440-02-0	NICKEL	6	64.4	mg/kg	HB-T-5-3	26/26	-	6.44E+01			1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL	
	7440-09-7	POTASSIUM	147 J	1210	mg/kg	HB-CSXSED-2	31/31	-	1.21E+03			NV	NV	NV	N	NUT	
	7782-49-2	SELENIUM	0.8 J	4.9 J	mg/kg	HB-S-2	19/32	0.64-4.6	4.90E+00			3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL	
	7440-22-4	SILVER	0.14 J	9.8	mg/kg	HB-T-5-3	11/32	0.19-1.9	9.80E+00			3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL	
	7440-23-5	SODIUM	693	4140	mg/kg	HB-T-3-3	24/30	782-1880	4.14E+03			NV	NV	NV	N	NUT	
	7440-28-0	THALLIUM	0.37 J	4.9 J	mg/kg	HB-S-2	4/32	0.33-2	4.90E+00			5.48E-01	N	5.16E-01	nc	5.16E-01 Y ASL	
	7440-62-2	VANADIUM	5.1 J	18.1	mg/kg	HB-T-4-2	31/32	7.2-7.2	1.81E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL	
	7440-66-6	ZINC	49.8	497	mg/kg	HB-T-4-2	26/26	-	4.97E+02			2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL	
	PCBs																
		LESS CHLORINATED PCBs ^c	0.12	0.12	mg/kg	HB-T-4-2	1/34	0.041-10	1.20E-01			5.48E-01	N	3.93E-01	nc	3.93E-01 N	BSL
		HIGHLY CHLORINATED PCBs ^d	0.067	4.7	mg/kg	HB-H6	23/34	0.041-10	4.70E+00			3.19E-01	C	2.22E-01	ca	2.22E-01 Y	ASL
		TOTAL PCBs ^e	0.067	10.1	mg/kg	HB-H6	23/34	0.041-10	1.01E+01			3.19E-01	C	2.22E-01	ca	2.22E-01 Y	ASL
	PESTICIDES																
	72-54-8	4,4'-DDD	0.028 J	0.059 J	mg/kg	HB-H7	4/32	0.021-0.079	5.90E-02			2.66E+00	C	2.44E+00	ca	2.44E+00 N	BSL
	72-55-9	4,4'-DDE	0.0063 J	0.021 J	mg/kg	HB-H5	6/32	0.021-0.063	2.10E-02			1.88E+00	C	1.72E+00	ca	1.72E+00 N	BSL
	50-29-3	4,4'-DDT	0.014 J	0.075 J	mg/kg	HB-H6	4/32	0.025-0.089	7.50E-02			1.88E+00	C	1.72E+00	ca	1.72E+00 N	BSL
	309-00-2	ALDRIN	0.0045 J	0.0091 J	mg/kg	HB-H3	2/32	0.013-0.053	9.10E-03			3.76E-02	C	2.86E-02	ca	2.86E-02 N	BSL
	57-74-9	TOTAL CHLORDANE ^f	5.30E-03	7.40E-02	mg/kg	HB-H5	6/31	0.013-0.5	2.20E-02			1.82E+00	C	1.62E+00	ca	1.62E+00 N	BSL
	60-57-1	DIELDRIN	0.011 J	0.069 J	mg/kg	HB-H5	6/32	0.021-0.11	6.90E-02			3.99E-02	C	3.04E-02	ca	3.04E-02 Y	ASL
	33213-65-9	ENDOSULFAN II ^g	0.0087 J	0.023 J	mg/kg	HB-H4	2/32	0.025-0.12	2.30E-02			4.69E+01	N	3.67E+01	nc	3.67E+01 N	BSL
	1031-07-8	ENDOSULFAN SULFATE ^g	0.1	0.1	mg/kg	HB-T-5-1	1/32	0.025-0.12	1.00E-01			4.69E+01	N	3.67E+01	nc	3.67E+01 N	BSL
	72-20-8	ENDRIN	0.027 J	0.027 J	mg/kg	HB-H5	1/32	0.021-0.11	2.70E-02			2.35E+00	N	1.83E+00	nc	1.83E+00 N	BSL
	7421-93-4	ENDRIN ALDEHYDE ^h	0.0022 J	0.1 J	mg/kg	HB-H6	5/32	0.025-0.089	1.00E-01			2.35E+00	N	1.83E+00	nc	1.83E+00 N	BSL
	53494-70-5	ENDRIN KETONE ^h	0.083 J	0.083 J	mg/kg	HB-H7	1/32	0.025-0.12	8.30E-02			2.35E+00	N	1.83E+00	nc	1.83E+00 N	BSL
	58-89-9	GAMMA-BHC (LINDANE)	0.005 J	0.0096 J	mg/kg	HB-H5	3/32	0.013-0.053	9.60E-03			4.91E-01	C	4.37E-01	ca	4.37E-01 N	BSL
	76-44-8	HEPTACHLOR	0.0013 J	0.015 J	mg/kg	HB-H7	3/32	0.013-0.053	1.50E-02			1.42E-01	C	1.08E-01	ca	1.08E-01 N	BSL
	1024-57-3	HEPTACHLOR EPOXIDE	0.004 J	0.03 J	mg/kg	HB-H3	6/32	0.013-0.043	3.00E-02			7.02E-02	C	5.34E-02	ca	5.34E-02 N	BSL
	SVOCs																
	105-67-9	2,4-DIMETHYLPHENOL	3	23	mg/kg	HB-T-3-3	4/32	0.43-30	2.30E+01			1.56E+02	N	1.22E+02	nc	1.22E+02 Y	BSL
	91-57-6	2-METHYLNAPHTHALENE	0.092	210	mg/kg	HB-S-1	23/27	0.49-11	2.10E+02			3.13E+01	N	NV	nc	3.13E+01 Y	ASL
	95-48-7	2-METHYLPHENOL	0.27	5.5	mg/kg	HB-T-3-3	5/31	0.083-15	5.50E+00			3.91E+02	N	3.06E+02	nc	3.06E+02 N	BSL
34METPH	3&4-METHYLPHENOL ⁱ	0.5	9.6	mg/kg	HB-T-3-3	7/30	0.083-15	9.60E+00			3.91E+01	N	3.06E+01	nc	3.06E+01 N	BSL	
83-32-9	ACENAPHTHENE	0.11 J	91	mg/kg	HB-HBSED-19	27/28	4-4	9.10E+01			4.69E+02	N	3.68E+02	nc	3.68E+02 N	BSL	
208-96-8	ACENAPHTHYLENE	0.073 J	51	mg/kg	HB-S-1	24/29	0.55-5.8	5.10E+01			NV	NV	NV	Y	NTX		
120-12-7	ANTHRACENE	0.13 J	72	mg/kg	HB-S-1	25/27	2.1-4	7.20E+01			2.35E+03	N	2.19E+03	nc	2.19E+03 N	BSL	
56-55-3	BENZ(A)ANTHRACENE	0.47 J	25	mg/kg	HB-T-5-1	26/27	4-4	2.50E+01			2.20E-01	C	6.21E-01	ca	2.20E-01 Y	ASL	
50-32-8	BENZO(A)PYRENE	0.77	25	mg/kg	HB-T-5-1	26/28	2.3-4	2.50E+01			2.20E-02	C	6.21E-02	ca	2.20E-02 Y	ASL	
205-99-2	BENZO(B)FLUORANTHENE	0.58	18	mg/kg	HB-T-5-1	25/27	2.3-4	1.80E+01			2.20E-01	C	6.21E-01	ca	2.20E-01 Y	ASL	
191-24-2	BENZO(G,H,I)PERYLENE	0.68	14	mg/kg	HB-T-5-1	27/29	2.3-4	1.40E+01			NV	NV	NV	Y	NTX		
207-08-9	BENZO(K)FLUORANTHENE	0.53 J	21	mg/kg	HB-T-5-1	27/29	2.3-4	2.10E+01			2.20E+00	C	6.21E+00	ca	2.20E+00 Y	ASL	
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.11 J	4	mg/kg	HB-T-3-3	20/31	0.28-15	4.00E+00			4.56E+01	C	3.47E+01	ca	3.47E+01 N	BSL

TABLE 2.18a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- HARBOR BROOK SURFACE SEDIMENT
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
	86-74-8	CARBAZOLE	0.09 J	9.9 J	mg/kg	HB-HBSED-19	24/29	0.55-5.8	9.90E+00			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL
	218-01-9	CHRYSENE	0.55	24	mg/kg	HB-T-5-1	26/27	4-4	2.40E+01			2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.16 J	5.2	mg/kg	HB-T-5-1	19/31	0.55-18	5.20E+00			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.13 J	100	mg/kg	HB-S-1	25/28	0.8-4	1.00E+02			7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.11	0.7	mg/kg	HB-T-1-2	3/32	0.13-18	7.00E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	206-44-0	FLUORANTHENE	0.87	90	mg/kg	HB-S-1	26/26	-	9.00E+01			3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	86-73-7	FLUORENE	0.085 J	110	mg/kg	HB-S-1	25/27	0.083-4	1.10E+02			3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	118-74-1	HEXACHLORO BENZENE	0.53 J	0.53 J	mg/kg	HB-H2	1/31	0.083-18	5.30E-01			3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.66	14	mg/kg	HB-T-5-1	26/29	2.3-4.8	1.40E+01			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.11	240	mg/kg	HB-T-4-1	26/26	-	2.40E+02			1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	85-01-8	PHENANTHRENE	0.51 J	260	mg/kg	HB-S-1	26/26	-	2.60E+02			NV	NV	NV	NV	Y	NTX	
	108-95-2	PHENOL	0.23 J	2.3	mg/kg	HB-T-3-3	7/32	0.083-18	2.30E+00			2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.73	61	mg/kg	HB-S-1	25/26	4-4	6.10E+01			2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCS																	
	120-82-1	1,2,4-TRICHLOROBENZENE	3.4	8.1	mg/kg	HB-T-5-3	2/26	0.083-11	8.10E+00			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	0.27	5.9	mg/kg	HB-T-1-3	3/30	0.083-18	5.90E+00			7.04E+02	N	6.00E+01	nc	6.00E+01	N	BSL
	541-73-1	1,3-DICHLOROBENZENE	0.74 J	0.95	mg/kg	HB-T-1-3	2/31	0.083-18	9.50E-01			2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.23 J	29	mg/kg	HB-T-5-3	5/30	0.083-18	2.90E+01			2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL
	78-93-3	2-BUTANONE	0.007 J	0.055 J	mg/kg	HB-T-5-1	7/31	0.015-17	5.50E-02			4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.027 J	1.2 J	mg/kg	HB-T-3-1	10/29	0.056-33	1.20E+00			7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.003 J	29	mg/kg	HB-T-5-3	22/30	0.016-4.8	2.90E+01			1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.0017	0.012 J	mg/kg	HB-HBSED-19	7/32	0.016-17	1.20E-02			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL
	108-90-7	CHLOROBENZENE	0.001 J	240	mg/kg	HB-T-5-3	12/30	0.0074-8.3	2.40E+02			1.56E+02	N	1.51E+01	nc	1.51E+01	Y	ASL
	100-41-4	ETHYLBENZENE	0.0031 J	24	mg/kg	HB-T-5-3	24/27	0.016-4.8	2.40E+01			7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.012 J	9.5	mg/kg	HB-T-3-2	3/30	0.0074-140	9.50E+00			8.52E+01	C	9.11E+00	ca	9.11E+00	Y	ASL
	100-42-5	STYRENE	0.011 J	0.073	mg/kg	HB-HBSED-19	2/32	0.0074-8.3	7.30E-02			1.56E+03	N	1.70E+02	nc	1.70E+02	N	BSL
	108-88-3	TOLUENE	0.005 J	88	mg/kg	HB-T-5-3	20/29	0.0082-4.8	8.80E+01			6.26E+02	N	5.20E+01	nc	5.20E+01	Y	ASL
	1330-20-7	XYLENES, TOTAL	0.0047	314	mg/kg	HB-T-5-3	28/30	1.1-4.8	3.14E+02			1.56E+03	N	2.71E+01	nc	2.71E+01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(8) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.18b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
f = Where criteria are not available, RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
g = RBC and PRG values for Endosulfan (CAS# 115297) utilized.
h = RBC and PRG values for Endrin (CAS # 72208) utilized.
i = RBC and PRG values for 4-methylphenol utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.18b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-14	11/14/2002	0	0.33	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	4.301	4.301	ng/kg		0.01	0.043
HB-HBSED-14	11/14/2002	0	0.33	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.548	0.274	ng/kg	U	0.01	0.003
HB-HBSED-14	11/14/2002	0	0.33	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.291	0.1455	ng/kg	U	0.1	0.015
HB-HBSED-14	11/14/2002	0	0.33	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.227	0.1135	ng/kg	U	0.1	0.011
HB-HBSED-14	11/14/2002	0	0.33	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.073	1.073	ng/kg	J	0.1	0.107
HB-HBSED-14	11/14/2002	0	0.33	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.217	0.1085	ng/kg	U	0.1	0.011
HB-HBSED-14	11/14/2002	0	0.33	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.27	0.135	ng/kg	U	0.1	0.014
HB-HBSED-14	11/14/2002	0	0.33	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.275	0.1375	ng/kg	U	0.1	0.014
HB-HBSED-14	11/14/2002	0	0.33	40321-76-4	1,2,3,7,8-PECDD	N	0.38	0.19	ng/kg	U	1	0.190
HB-HBSED-14	11/14/2002	0	0.33	57117-41-6	1,2,3,7,8-PCDF	N	0.273	0.1365	ng/kg	U	0.03	0.004
HB-HBSED-14	11/14/2002	0	0.33	1746-01-6	2,3,7,8-TCDD	N	0.377	0.1885	ng/kg	U	1	0.189
HB-HBSED-14	11/14/2002	0	0.33	51207-31-9	2,3,7,8-TCDF	N	0.353	0.1765	ng/kg	U	0.1	0.018
HB-HBSED-14	11/14/2002	0	0.33	3268-87-9	OCDD	Y	161.945	161.945	ng/kg		0.0003	0.049
HB-HBSED-14	11/14/2002	0	0.33	39001-02-0	OCDF	Y	10.146	10.146	ng/kg	EMPC	0.0003	0.003
Sample Location TEQ = 0.7												
HB-HBSED-15	11/14/2002	0	0.25	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	22.716	22.716	ng/kg		0.01	0.227
HB-HBSED-15	11/14/2002	0	0.25	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.817	0.4085	ng/kg	U	0.01	0.004
HB-HBSED-15	11/14/2002	0	0.25	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.417	0.2085	ng/kg	U	0.1	0.021
HB-HBSED-15	11/14/2002	0	0.25	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.497	1.497	ng/kg	J	0.1	0.150
HB-HBSED-15	11/14/2002	0	0.25	57653-85-7	1,2,3,6,7,8-HXCDD	Y	4.201	4.201	ng/kg		0.1	0.420
HB-HBSED-15	11/14/2002	0	0.25	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.919	0.919	ng/kg	J	0.1	0.092
HB-HBSED-15	11/14/2002	0	0.25	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.269	3.269	ng/kg		0.1	0.327
HB-HBSED-15	11/14/2002	0	0.25	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.366	0.183	ng/kg	U	0.1	0.018
HB-HBSED-15	11/14/2002	0	0.25	40321-76-4	1,2,3,7,8-PECDD	N	0.489	0.2445	ng/kg	U	1	0.245
HB-HBSED-15	11/14/2002	0	0.25	57117-41-6	1,2,3,7,8-PCDF	N	0.349	0.1745	ng/kg	U	0.03	0.005
HB-HBSED-15	11/14/2002	0	0.25	1746-01-6	2,3,7,8-TCDD	N	0.496	0.248	ng/kg	U	1	0.248
HB-HBSED-15	11/14/2002	0	0.25	51207-31-9	2,3,7,8-TCDF	N	0.399	0.1995	ng/kg	U	0.1	0.020
HB-HBSED-15	11/14/2002	0	0.25	3268-87-9	OCDD	Y	881.1	881.1	ng/kg		0.0003	0.264
HB-HBSED-15	11/14/2002	0	0.25	39001-02-0	OCDF	Y	75.394	75.394	ng/kg		0.0003	0.023
Sample Location TEQ = 2.1												
HB-HBSED-16	6/2/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	62.956	62.956	ng/kg	J	0.01	0.630
HB-HBSED-16	6/2/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	1.703	0.8515	ng/kg	UJ	0.01	0.009
HB-HBSED-16	6/2/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.212	1.212	ng/kg	J	0.1	0.121
HB-HBSED-16	6/2/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	4.006	4.006	ng/kg		0.1	0.401
HB-HBSED-16	6/2/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	6.542	6.542	ng/kg		0.1	0.654
HB-HBSED-16	6/2/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.01	2.01	ng/kg	J	0.1	0.201
HB-HBSED-16	6/2/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.741	3.741	ng/kg	J	0.1	0.374
HB-HBSED-16	6/2/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.61	0.305	ng/kg	U	0.1	0.031
HB-HBSED-16	6/2/2003	0	0.5	57117-41-6	1,2,3,7,8-PCDF	Y	0.998	0.998	ng/kg	J	0.03	0.030
HB-HBSED-16	6/2/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.389	0.1945	ng/kg	U	1	0.195
HB-HBSED-16	6/2/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.96	2.96	ng/kg		0.1	0.296
HB-HBSED-16	6/2/2003	0	0.5	3268-87-9	OCDD	Y	1700.214	1700.214	ng/kg	J	0.0003	0.510
HB-HBSED-16	6/2/2003	0	0.5	39001-02-0	OCDF	Y	239.938	239.938	ng/kg	J	0.0003	0.072
Sample Location TEQ = 3.5												

TABLE 2.18b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-19	6/4/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	37.284	37.284	ng/kg	UJ EMPC	0.01	0.373
HB-HBSED-19	6/4/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	1.44	0.72	ng/kg		0.01	0.007
HB-HBSED-19	6/4/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.221	2.221	ng/kg		0.1	0.222
HB-HBSED-19	6/4/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.592	2.592	ng/kg		0.1	0.259
HB-HBSED-19	6/4/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.593	5.593	ng/kg		0.1	0.559
HB-HBSED-19	6/4/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.358	5.358	ng/kg	U J U J J	0.1	0.536
HB-HBSED-19	6/4/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.738	0.369	ng/kg		0.1	0.037
HB-HBSED-19	6/4/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.462	1.462	ng/kg		1	1.462
HB-HBSED-19	6/4/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.366	0.183	ng/kg		1	0.183
HB-HBSED-19	6/4/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.03	2.03	ng/kg		0.1	0.203
HB-HBSED-19	6/4/2003	0	0.5	3268-87-9	OCDD	Y	1049.111	1049.111	ng/kg	J	0.0003	0.315
HB-HBSED-19	6/4/2003	0	0.5	39001-02-0	OCDF	Y	188.842	188.842	ng/kg	J	0.0003	0.057
Sample Location TEQ =												4.2
HB-HBSED-19	6/4/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	43.776	43.776	ng/kg	J	0.01	0.438
HB-HBSED-19	6/4/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.813	1.813	ng/kg		0.1	0.181
HB-HBSED-19	6/4/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDD	Y	3.814	3.814	ng/kg		0.1	0.381
HB-HBSED-19	6/4/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.999	7.999	ng/kg		0.1	0.800
HB-HBSED-19	6/4/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.719	2.719	ng/kg		0.1	0.272
HB-HBSED-19	6/4/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.163	6.163	ng/kg	U J J J J	0.1	0.616
HB-HBSED-19	6/4/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.708	0.354	ng/kg		0.1	0.035
HB-HBSED-19	6/4/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	0.819	0.819	ng/kg		0.03	0.025
HB-HBSED-19	6/4/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	0.419	0.419	ng/kg		1	0.419
HB-HBSED-19	6/4/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	1.58	1.58	ng/kg		0.1	0.158
HB-HBSED-19	6/4/2003	0.5	1	3268-87-9	OCDD	Y	1109.094	1109.094	ng/kg	J	0.0003	0.333
HB-HBSED-19	6/4/2003	0.5	1	39001-02-0	OCDF	Y	128.018	128.018	ng/kg	J	0.0003	0.038
Sample Location TEQ =												3.7
HB-HBSED-20	6/4/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	16.022	16.022	ng/kg	J J	0.01	0.160
HB-HBSED-20	6/4/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.005	2.005	ng/kg		0.1	0.201
HB-HBSED-20	6/4/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDD	Y	1.636	1.636	ng/kg		0.1	0.164
HB-HBSED-20	6/4/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.672	5.672	ng/kg		0.1	0.567
HB-HBSED-20	6/4/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.141	1.141	ng/kg		0.1	0.114
HB-HBSED-20	6/4/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.559	5.559	ng/kg	J U U J J	0.1	0.556
HB-HBSED-20	6/4/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.205	0.1025	ng/kg		0.1	0.010
HB-HBSED-20	6/4/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.119	0.0595	ng/kg		1	0.060
HB-HBSED-20	6/4/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.804	0.804	ng/kg		0.1	0.080
HB-HBSED-20	6/4/2003	0	0.5	3268-87-9	OCDD	Y	646.345	646.345	ng/kg		0.0003	0.194
HB-HBSED-20	6/4/2003	0	0.5	39001-02-0	OCDF	Y	29.292	29.292	ng/kg	J	0.0003	0.009
Sample Location TEQ =												2.1

TABLE 2.18b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-S-1	1/31/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	37.747	37.747	ng/kg	J	0.01	0.377
HB-S-1	1/31/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	8.997	8.997	ng/kg	J	0.1	0.900
HB-S-1	1/31/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.553	5.553	ng/kg	J	0.1	0.555
HB-S-1	1/31/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	4.234	4.234	ng/kg	J	0.1	0.423
HB-S-1	1/31/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.759	1.759	ng/kg	J	0.1	0.176
HB-S-1	1/31/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.53	0.265	ng/kg	UJ	0.1	0.027
HB-S-1	1/31/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	2.058	2.058	ng/kg	J	0.03	0.062
HB-S-1	1/31/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.632	0.316	ng/kg	UJ	1	0.316
HB-S-1	1/31/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.17	1.17	ng/kg	J	0.1	0.117
HB-S-1	1/31/2001	0	0.5	3268-87-9	OCDD	Y	1420.293	1420.293	ng/kg	J	0.0003	0.426
HB-S-1	1/31/2001	0	0.5	39001-02-0	OCDF	Y	121.707	121.707	ng/kg	J	0.0003	0.037
Sample Location TEQ = 3.4												
HB-S-2	1/31/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	27.814	27.814	ng/kg		0.01	0.278
HB-S-2	1/31/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.367	1.367	ng/kg	J	0.1	0.137
HB-S-2	1/31/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.068	2.068	ng/kg	J	0.1	0.207
HB-S-2	1/31/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.113	5.113	ng/kg		0.1	0.511
HB-S-2	1/31/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.825	1.825	ng/kg	J	0.1	0.183
HB-S-2	1/31/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.401	0.2005	ng/kg	U	0.1	0.020
HB-S-2	1/31/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.334	0.167	ng/kg	UJ	0.03	0.005
HB-S-2	1/31/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.568	0.284	ng/kg	UJ	1	0.284
HB-S-2	1/31/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.33	1.33	ng/kg		0.1	0.133
HB-S-2	1/31/2001	0	0.5	3268-87-9	OCDD	Y	912.452	912.452	ng/kg		0.0003	0.274
HB-S-2	1/31/2001	0	0.5	39001-02-0	OCDF	Y	74.485	74.485	ng/kg		0.0003	0.022
Sample Location TEQ = 2.1												
HB-T-1-2	1/24/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	84.417	84.417	ng/kg		0.01	0.844
HB-T-1-2	1/24/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	27.021	27.021	ng/kg		0.01	0.270
HB-T-1-2	1/24/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	4.534	4.534	ng/kg		0.1	0.453
HB-T-1-2	1/24/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	90.244	90.244	ng/kg		0.1	9.024
HB-T-1-2	1/24/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	17.131	17.131	ng/kg		0.1	1.713
HB-T-1-2	1/24/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	29.579	29.579	ng/kg		0.1	2.958
HB-T-1-2	1/24/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	7.816	7.816	ng/kg	J	0.1	0.782
HB-T-1-2	1/24/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	20.203	20.203	ng/kg		0.1	2.020
HB-T-1-2	1/24/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	54.334	54.334	ng/kg		0.03	1.630
HB-T-1-2	1/24/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.314	1.314	ng/kg		1	1.314
HB-T-1-2	1/24/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	63.5	63.5	ng/kg		0.1	6.350
HB-T-1-2	1/24/2001	0	0.5	3268-87-9	OCDD	Y	673.904	673.904	ng/kg		0.0003	0.202
HB-T-1-2	1/24/2001	0	0.5	39001-02-0	OCDF	Y	154.272	154.272	ng/kg		0.0003	0.046
Sample Location TEQ = 27.6												

TABLE 2.18b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-2-3	1/25/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	328.425	328.425	ng/kg	J	0.01	3.284
HB-T-2-3	1/25/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	25.007	25.007	ng/kg	J	0.01	0.250
HB-T-2-3	1/25/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.235	6.235	ng/kg	J	0.1	0.624
HB-T-2-3	1/25/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	61.935	61.935	ng/kg	J	0.1	6.194
HB-T-2-3	1/25/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	51.72	51.72	ng/kg	J	0.1	5.172
HB-T-2-3	1/25/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	24.718	24.718	ng/kg	J	0.1	2.472
HB-T-2-3	1/25/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	18.23	18.23	ng/kg	J	0.1	1.823
HB-T-2-3	1/25/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	15.997	15.997	ng/kg	J	0.1	1.600
HB-T-2-3	1/25/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	9.719	9.719	ng/kg		1	9.719
HB-T-2-3	1/25/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	28.773	28.773	ng/kg		0.03	0.863
HB-T-2-3	1/25/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.583	2.583	ng/kg		1	2.583
HB-T-2-3	1/25/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	54.69	54.69	ng/kg	J	0.1	5.469
HB-T-2-3	1/25/2001	0	0.5	3268-87-9	OCDD	Y	1477.489	1477.489	ng/kg	J	0.0003	0.443
HB-T-2-3	1/25/2001	0	0.5	39001-02-0	OCDF	Y	298.975	298.975	ng/kg	J	0.0003	0.090
Sample Location TEQ = 40.6												
HB-T-3-2	1/26/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	41.508	41.508	ng/kg	J	0.01	0.415
HB-T-3-2	1/26/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	3.149	3.149	ng/kg		0.01	0.031
HB-T-3-2	1/26/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.975	1.975	ng/kg		0.1	0.198
HB-T-3-2	1/26/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.704	7.704	ng/kg		0.1	0.770
HB-T-3-2	1/26/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.522	8.522	ng/kg		0.1	0.852
HB-T-3-2	1/26/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.486	3.486	ng/kg		0.1	0.349
HB-T-3-2	1/26/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.648	5.648	ng/kg	J	0.1	0.565
HB-T-3-2	1/26/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	3.04	3.04	ng/kg		0.03	0.091
HB-T-3-2	1/26/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	0.874	0.874	ng/kg	J	1	0.874
HB-T-3-2	1/26/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	6.7	6.7	ng/kg	J	0.1	0.670
HB-T-3-2	1/26/2001	0	0.5	3268-87-9	OCDD	Y	1197.39	1197.39	ng/kg	J	0.0003	0.359
HB-T-3-2	1/26/2001	0	0.5	39001-02-0	OCDF	Y	109.18	109.18	ng/kg		0.0003	0.033
Sample Location TEQ = 5.2												
HB-T-4-2	1/29/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	16.947	16.947	ng/kg	J	0.01	0.169
HB-T-4-2	1/29/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.21	0.105	ng/kg	UJ	0.01	0.001
HB-T-4-2	1/29/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.713	0.713	ng/kg	J	0.1	0.071
HB-T-4-2	1/29/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.012	2.012	ng/kg	J	0.1	0.201
HB-T-4-2	1/29/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	4.659	4.659	ng/kg	J	0.1	0.466
HB-T-4-2	1/29/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.139	1.139	ng/kg	J	0.1	0.114
HB-T-4-2	1/29/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.319	0.1595	ng/kg	UJ	0.1	0.016
HB-T-4-2	1/29/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.893	0.893	ng/kg	J	1	0.893
HB-T-4-2	1/29/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.123	1.123	ng/kg	J	0.03	0.034
HB-T-4-2	1/29/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.232	0.116	ng/kg	U	1	0.116
HB-T-4-2	1/29/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.23	2.23	ng/kg		0.1	0.223
HB-T-4-2	1/29/2001	0	0.5	3268-87-9	OCDD	Y	514.462	514.462	ng/kg		0.0003	0.154
HB-T-4-2	1/29/2001	0	0.5	39001-02-0	OCDF	Y	52.439	52.439	ng/kg		0.0003	0.016
Sample Location TEQ = 2.5												

TABLE 2.18b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-4-2	1/29/2001	0	1.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	40.656	40.656	ng/kg	J	0.01	0.407
HB-T-4-2	1/29/2001	0	1.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.943	1.943	ng/kg	J	0.01	0.019
HB-T-4-2	1/29/2001	0	1.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.762	1.762	ng/kg	J	0.1	0.176
HB-T-4-2	1/29/2001	0	1.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	8.026	8.026	ng/kg	J	0.1	0.803
HB-T-4-2	1/29/2001	0	1.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.051	8.051	ng/kg	J	0.1	0.805
HB-T-4-2	1/29/2001	0	1.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.778	3.778	ng/kg	J	0.1	0.378
HB-T-4-2	1/29/2001	0	1.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.045	2.045	ng/kg	J	0.1	0.205
HB-T-4-2	1/29/2001	0	1.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.596	1.596	ng/kg	J	0.1	0.160
HB-T-4-2	1/29/2001	0	1.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.967	1.967	ng/kg	J	1	1.967
HB-T-4-2	1/29/2001	0	1.5	1746-01-6	2,3,7,8-TCDD	Y	0.495	0.495	ng/kg	J	1	0.495
HB-T-4-2	1/29/2001	0	1.5	51207-31-9	2,3,7,8-TCDF	Y	5.8	5.8	ng/kg		0.1	0.580
HB-T-4-2	1/29/2001	0	1.5	3268-87-9	OCDD	Y	693.792	693.792	ng/kg	J	0.0003	0.208
HB-T-4-2	1/29/2001	0	1.5	39001-02-0	OCDF	Y	77.33	77.33	ng/kg	J	0.0003	0.023
Sample Location TEQ = 6.2												
HB-T-5-1	1/30/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	59.982	59.982	ng/kg	J	0.01	0.600
HB-T-5-1	1/30/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.433	0.2165	ng/kg	UJ	0.01	0.002
HB-T-5-1	1/30/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.282	1.282	ng/kg	J	0.1	0.128
HB-T-5-1	1/30/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	56.608	56.608	ng/kg	J	0.1	5.661
HB-T-5-1	1/30/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.597	8.597	ng/kg	J	0.1	0.860
HB-T-5-1	1/30/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	17.493	17.493	ng/kg	J	0.1	1.749
HB-T-5-1	1/30/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.418	2.418	ng/kg	J	0.1	0.242
HB-T-5-1	1/30/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.519	0.2595	ng/kg	UJ	0.1	0.026
HB-T-5-1	1/30/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	2.904	2.904	ng/kg		1	2.904
HB-T-5-1	1/30/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	16.127	16.127	ng/kg		0.03	0.484
HB-T-5-1	1/30/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.331	0.1655	ng/kg	U	1	0.166
HB-T-5-1	1/30/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	24.1	24.1	ng/kg		0.1	2.410
HB-T-5-1	1/30/2001	0	0.5	3268-87-9	OCDD	Y	705.376	705.376	ng/kg	J	0.0003	0.212
HB-T-5-1	1/30/2001	0	0.5	39001-02-0	OCDF	Y	158.594	158.594	ng/kg	J	0.0003	0.048
Sample Location TEQ = 15.5												

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans
 EMPC = Estimated Maximum Possible Concentration
 N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.18c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-H2	0	1.3	11/7/1996	0.23	mg/kg
Highly Chlorinated PCBs	HB-H3	0	1	11/7/1996	0.55	mg/kg
Highly Chlorinated PCBs	HB-H4	0	1	11/7/1996	0.79	mg/kg
Highly Chlorinated PCBs	HB-H5	0	1	11/7/1996	1	mg/kg
Highly Chlorinated PCBs	HB-H6	0	0.8	11/7/1996	4.7	mg/kg
Highly Chlorinated PCBs	HB-H7	0	1	11/7/1996	2.67	mg/kg
Highly Chlorinated PCBs	HB-HBSED-16	0	0.5	6/2/2003	0.37	mg/kg
Highly Chlorinated PCBs	HB-HBSED-19	0	0.5	6/3/2003	0.21	mg/kg
Highly Chlorinated PCBs	HB-HBSED-19	0.5	1	6/3/2003	0.13	mg/kg
Highly Chlorinated PCBs	HB-T-1-1	0	0.5	1/24/2001	0.32	mg/kg
Highly Chlorinated PCBs	HB-T-1-2	0	0.5	1/24/2001	0.89	mg/kg
Highly Chlorinated PCBs	HB-T-2-1	0	0.5	1/25/2001	0.27	mg/kg
Highly Chlorinated PCBs	HB-T-2-2	0	0.5	1/25/2001	0.11	mg/kg
Highly Chlorinated PCBs	HB-T-2-3	0	0.5	1/25/2001	3.7	mg/kg
Highly Chlorinated PCBs	HB-T-3-1	0	0.5	1/26/2001	0.16	mg/kg
Highly Chlorinated PCBs	HB-T-3-2	0	0.5	1/26/2001	1.29	mg/kg
Highly Chlorinated PCBs	HB-T-3-3	0	0.5	1/26/2001	0.16	mg/kg
Highly Chlorinated PCBs	HB-T-4-1	0	0.5	1/29/2001	0.19	mg/kg
Highly Chlorinated PCBs	HB-T-4-2	0	0.5	1/29/2001	0.067	mg/kg
Highly Chlorinated PCBs	HB-T-4-2	0	1.5	1/29/2001	0.3	mg/kg
Highly Chlorinated PCBs	HB-T-4-3	0	0.5	1/29/2001	0.091	mg/kg
Highly Chlorinated PCBs	HB-T-5-1	0	0.5	1/30/2001	0.37	mg/kg
Highly Chlorinated PCBs	HB-T-5-2	0	0.5	1/31/2001	0.13	mg/kg
Less Chlorinated PCBs	HB-T-4-2	0	0.5	1/29/2001	0.12	mg/kg
Total PCBs	HB-H2	0	1.3	11/7/1996	0.23	mg/kg
Total PCBs	HB-H3	0	1	11/7/1996	0.55	mg/kg
Total PCBs	HB-H4	0	1	11/7/1996	0.79	mg/kg
Total PCBs	HB-H5	0	1	11/7/1996	1	mg/kg
Total PCBs	HB-H6	0	0.8	11/7/1996	4.7	mg/kg
Total PCBs	HB-H7	0	1	11/7/1996	2.67	mg/kg
Total PCBs	HB-HBSED-16	0	0.5	6/2/2003	0.37	mg/kg
Total PCBs	HB-HBSED-19	0	0.5	6/3/2003	0.21	mg/kg
Total PCBs	HB-HBSED-19	0.5	1	6/3/2003	0.13	mg/kg
Total PCBs	HB-T-1-1	0	0.5	1/24/2001	0.32	mg/kg
Total PCBs	HB-T-1-2	0	0.5	1/24/2001	0.89	mg/kg
Total PCBs	HB-T-2-1	0	0.5	1/25/2001	0.27	mg/kg
Total PCBs	HB-T-2-2	0	0.5	1/25/2001	0.11	mg/kg
Total PCBs	HB-T-2-3	0	0.5	1/25/2001	3.7	mg/kg
Total PCBs	HB-T-3-1	0	0.5	1/26/2001	0.16	mg/kg
Total PCBs	HB-T-3-2	0	0.5	1/26/2001	1.29	mg/kg

TABLE 2.18c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-T-3-3	0	0.5	1/26/2001	0.16	mg/kg
Total PCBs	HB-T-4-1	0	0.5	1/29/2001	0.19	mg/kg
Total PCBs	HB-T-4-2	0	0.5	1/29/2001	0.187	mg/kg
Total PCBs	HB-T-4-2	0	1.5	1/29/2001	0.3	mg/kg
Total PCBs	HB-T-4-3	0	0.5	1/29/2001	0.091	mg/kg
Total PCBs	HB-T-5-1	0	0.5	1/30/2001	0.37	mg/kg
Total PCBs	HB-T-5-2	0	0.5	1/31/2001	0.13	mg/kg

Notes:

* Less chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher, if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.18d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-CSXSED-1	11/14/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.022
HB-CSXSED-1	11/14/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.022
Total Chlordane =									ND
HB-CSXSED-2	11/14/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-CSXSED-2	11/14/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-H3	11/7/1996	0	1	57-74-9	CHLORDANE	N	U	mg/kg	0.04
HB-H3	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.021
Total Chlordane =									0.021
HB-H4	11/7/1996	0	1	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-H4	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0091
Total Chlordane =									0.0091
HB-H5	11/7/1996	0	1	57-74-9	CHLORDANE	Y	J	mg/kg	0.048
HB-H5	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.026
Total Chlordane =									0.074
HB-H6	11/7/1996	0	0.8	57-74-9	CHLORDANE	N	U	mg/kg	0.053
HB-H6	11/7/1996	0	0.8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.028
Total Chlordane =									0.028
HB-H7	11/7/1996	0	1	57-74-9	CHLORDANE	N	U	mg/kg	0.045
HB-H7	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0053
Total Chlordane =									0.0053
HB-HBSED-14	11/14/2002	0	0.33	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-HBSED-14	11/14/2002	0	0.33	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-HBSED-15	11/14/2002	0	0.25	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-HBSED-15	11/14/2002	0	0.25	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-HBSED-16	6/2/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.015
HB-HBSED-16	6/2/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.015
Total Chlordane =									ND
HB-HBSED-19	6/3/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.014
HB-HBSED-19	6/3/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.014
Total Chlordane =									ND
HB-HBSED-19	6/3/2003	0.5	1	57-74-9	CHLORDANE	Y		mg/kg	0.022
HB-HBSED-19	6/3/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.022
Total Chlordane =									0.022
HB-HBSED-20	6/3/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.013
HB-HBSED-20	6/3/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.013
Total Chlordane =									ND
HB-S-1	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.025
HB-S-1	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.025
Total Chlordane =									ND

TABLE 2.18d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-S-2	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.043
HB-S-2	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.043
Total Chlordane =									ND
HB-T-1-1	1/24/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.041
HB-T-1-1	1/24/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.041
Total Chlordane =									ND
HB-T-1-2	1/24/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.041
HB-T-1-2	1/24/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.041
Total Chlordane =									ND
HB-T-1-3	1/24/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-1-3	1/24/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-2-1	1/25/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-T-2-1	1/25/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-T-2-2	1/25/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-2-2	1/25/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-2-3	1/25/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-T-2-3	1/25/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-T-3-1	1/26/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.025
HB-T-3-1	1/26/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.025
Total Chlordane =									ND
HB-T-3-2	1/26/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-T-3-2	1/26/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-T-3-3	1/26/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.035
HB-T-3-3	1/26/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.035
Total Chlordane =									ND
HB-T-4-1	1/29/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-4-1	1/29/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-4-2	1/29/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-4-2	1/29/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-4-3	1/29/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-T-4-3	1/29/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-T-5-1	1/30/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-5-1	1/30/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND

TABLE 2.18d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-T-5-2	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.021
HB-T-5-2	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.021
Total Chlordane =									ND
HB-T-5-3	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.041
HB-T-5-3	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.041
Total Chlordane =									ND
HB-T-5-OIL	2/14/2001	0	0	57-74-9	CHLORDANE	N	UJ	mg/kg	5
HB-T-5-OIL	2/14/2001	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	5
Total Chlordane =									ND

TABLE 2.18e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-H3	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	1.2	1.2
HB-H4	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.044	0.044
HB-H5	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.071	0.071
HB-H6	11/7/1996	0	0.8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	81	81
HB-H7	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.89	0.89
HB-CSXSED-1	11/14/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.41	
HB-CSXSED-1	11/14/2002	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.28	
HB-CSXSED-1	11/14/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.69
HB-CSXSED-2	11/14/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	1.5	
HB-CSXSED-2	11/14/2002	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	1	
HB-CSXSED-2	11/14/2002	0	0.5	CALCULATED	TOTAL	Y		mg/kg		2.5
HB-HBSED-14	11/14/2002	0	0.33	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0035	
HB-HBSED-14	11/14/2002	0	0.33	95-47-6	O-XYLENE	Y	J	mg/kg	0.0023	
HB-HBSED-14	11/14/2002	0	0.33	CALCULATED	TOTAL	Y	J	mg/kg		0.0058
HB-HBSED-15	11/14/2002	0	0.25	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.75	
HB-HBSED-15	11/14/2002	0	0.25	95-47-6	O-XYLENE	Y	J	mg/kg	0.38	
HB-HBSED-15	11/14/2002	0	0.25	CALCULATED	TOTAL	Y	J	mg/kg		1.13
HB-HBSED-16	6/2/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.19	
HB-HBSED-16	6/2/2003	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	0.085	
HB-HBSED-16	6/2/2003	0	0.5	CALCULATED	TOTAL	Y		mg/kg		0.275
HB-HBSED-19	6/3/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.35	
HB-HBSED-19	6/3/2003	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	1.6	
HB-HBSED-19	6/3/2003	0	0.5	CALCULATED	TOTAL	Y		mg/kg		1.95
HB-HBSED-19	6/3/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.2	
HB-HBSED-19	6/3/2003	0.5	1	95-47-6	O-XYLENE	Y		mg/kg	1.4	
HB-HBSED-19	6/3/2003	0.5	1	CALCULATED	TOTAL	Y		mg/kg		1.6
HB-HBSED-20	6/3/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.013	
HB-HBSED-20	6/3/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.0048	
HB-HBSED-20	6/3/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.0178
HB-S-1	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	12	
HB-S-1	1/31/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	4.1	
HB-S-1	1/31/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		16.1
HB-S-2	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0047	
HB-S-2	1/31/2001	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.016	
HB-S-2	1/31/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.0047
HB-T-1-1	1/24/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.033	
HB-T-1-1	1/24/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	0.06	
HB-T-1-1	1/24/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		0.093
HB-T-1-2	1/24/2001	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	4.8	
HB-T-1-2	1/24/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	4.8	
HB-T-1-2	1/24/2001	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		4.8
HB-T-1-3	1/24/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.029	
HB-T-1-3	1/24/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.045	
HB-T-1-3	1/24/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.029
HB-T-2-1	1/25/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.062	
HB-T-2-1	1/25/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	0.033	
HB-T-2-1	1/25/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		0.095
HB-T-2-2	1/25/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.3	
HB-T-2-2	1/25/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	1	
HB-T-2-2	1/25/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.3
HB-T-2-3	1/25/2001	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	1.1	
HB-T-2-3	1/25/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	1.1	
HB-T-2-3	1/25/2001	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		1.1
HB-T-3-1	1/26/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.74	
HB-T-3-1	1/26/2001	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.88	
HB-T-3-1	1/26/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		1.62

TABLE 2.18e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-T-3-2	1/26/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.53	
HB-T-3-2	1/26/2001	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.67	
HB-T-3-2	1/26/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		1.2
HB-T-3-3	1/26/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	38	
HB-T-3-3	1/26/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	12	
HB-T-3-3	1/26/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		50
HB-T-4-1	1/29/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	7.5	
HB-T-4-1	1/29/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	6.3	
HB-T-4-1	1/29/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		13.8
HB-T-4-2	1/29/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	38	
HB-T-4-2	1/29/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	15	
HB-T-4-2	1/29/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		53
HB-T-4-3	1/29/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	13	
HB-T-4-3	1/29/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	5.7	
HB-T-4-3	1/29/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		18.7
HB-T-5-1	1/30/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.023	
HB-T-5-1	1/30/2001	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.013	
HB-T-5-1	1/30/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.036
HB-T-5-2	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	4.3	
HB-T-5-2	1/31/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	1.6	
HB-T-5-2	1/31/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		5.9
HB-T-5-3	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	240	
HB-T-5-3	1/31/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	74	
HB-T-5-3	1/31/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		314

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.19a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- HARBOR BROOK SUBSURFACE SEDIMENT
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Upper Sediment (0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)
Harbor Brook Upper Sediment	DIOXIN/FURAN (8)															
	1746-01-6	2,3,7,8-TCDD Equivalent	0.0000002	0.000131	mg/kg	HB-T-1-2	33/33		1.31E-04			4.26E-06	C	3.90E-06	ca	3.90E-06 Y ASL
	METALS															
	7429-90-5	ALUMINUM	55.6	7240	mg/kg	HB-T-3-2	63/63	-	7.24E+03			7.82E+03	N	7.61E+03	nc	7.61E+03 N BSL
	7440-36-0	ANTIMONY	0.55 J	1.5 J	mg/kg	HB-H4	4/72	0.33-11.4	1.50E+00			3.13E+00	N	3.13E+00	nc	3.13E+00 N BSL
	7440-38-2	ARSENIC	1.6	14.5	mg/kg	HB-T-1-2	49/67	0.86-1.64	1.45E+01			4.26E-01	C	3.90E-01	ca	3.90E-01 Y TOX
	7440-39-3	BARIIUM	21.4 J	752	mg/kg	HB-T-1-2	71/72	1.83-1.83	7.52E+02			1.56E+03	N	5.37E+02	nc	5.37E+02 Y ASL
	7440-41-7	BERYLLIUM	0.1 J	2 J	mg/kg	HB-T-2-3	44/72	0.08-0.95	2.00E+00			1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL
	7440-43-9	CADMIUM	0.15 J	19.2	mg/kg	HB-T-3-3	40/72	0.14-0.95	1.92E+01			3.91E+00	N	3.70E+00	nc	3.70E+00 Y ASL
	7440-70-2	CALCIUM	2970	366000	mg/kg	HB-T-2-3	63/63	-	3.66E+05			NV	NV	NV	N	NUT
	7440-47-3	CHROMIUM ⁶	5.5	253	mg/kg	HB-T-1-2	46/63	0.22-13.8	2.53E+02			2.35E+01	N	3.01E+01	ca	2.35E+01 Y TOX
	7440-48-4	COBALT	0.97 J	21.2	mg/kg	HB-T-2-3	62/72	0.82-9.5	2.12E+01			NV	NV	9.03E+02	ca	9.03E+02 N BSL
	7440-50-8	COPPER	10.3	308	mg/kg	HB-CSXSED-2	47/61	0.43-11.4	3.08E+02			3.13E+02	N	3.13E+02	nc	3.13E+02 N BSL
	57-12-5	CYANIDE	1.92	4.76	mg/kg	HB-HBSED-15	9/70	0.61-2.51	4.76E+00			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	7439-89-6	IRON	115	21200	mg/kg	HB-CSXSED-2	63/63	-	2.12E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL
	7439-92-1	LEAD	1.7 J	753	mg/kg	HB-T-1-2	48/59	0.34-1.2	7.53E+02			NV	NV	4.00E+02	nc	4.00E+02 Y ASL
	7439-95-4	MAGNESIUM	125 J	59100	mg/kg	HB-T-5-2	63/63	-	5.91E+04			NV	NV	NV	N	NUT
	7439-96-5	MANGANESE	2.6 J	723	mg/kg	HB-T-2-3	63/63	-	7.23E+02			1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL
	7439-97-6	MERCURY ⁰	0 J	52	mg/kg	HB-T-5-3	56/63	0.0063-0.04	5.20E+01			7.82E-01	N	NV	NV	7.82E-01 Y ASL
	7440-02-0	NICKEL	2.5 J	64.4	mg/kg	HB-T-5-3	64/65	1.27-1.27	6.44E+01			1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL
	7440-09-7	POTASSIUM	147 J	1210	mg/kg	HB-CSXSED-2	66/71	95.39-181.19	1.21E+03			NV	NV	NV	N	NUT
	7782-49-2	SELENIUM	0.8 J	4.9 J	mg/kg	HB-S-2	54/72	0.64-4.6	4.90E+00			3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-22-4	SILVER	0.14 J	9.8	mg/kg	HB-T-5-3	17/72	0.08-1.9	9.80E+00			3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-23-5	SODIUM	693	19900	mg/kg	HB-T-3-2	62/68	782-1880	1.99E+04			NV	NV	NV	N	NUT
	7440-28-0	THALLIUM	0.25 J	4.9 J	mg/kg	HB-S-2	15/72	0.29-2	4.90E+00			5.48E-01	N	5.16E-01	nc	5.16E-01 Y ASL
	7440-62-2	VANADIUM	0.91 J	25.9	mg/kg	HB-T-2-3	70/72	3.5-7.2	2.59E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL
	7440-66-6	ZINC	16.5	497	mg/kg	HB-T-4-2	51/63	2.5-16.4	4.97E+02			2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL
	PCBs															
		LESS CHLORINATED PCBs ^c	0.12	0.12	mg/kg	HB-T-4-2	1/72	0.041-10	1.20E-01			5.48E-01	N	3.93E-01	nc	3.93E-01 N BSL
		HIGHLY CHLORINATED PCBs ^d	0.067	3.7	mg/kg	HB-T-2-3	27/72	0.041-10	3.70E+00			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
		TOTAL PCBs ^e	0.091	3.7	mg/kg	HB-T-2-3	27/72	0.041-10	3.70E+00			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
	PEST															
	72-54-8	4,4'-DDD	0.027 J	0.059 J	mg/kg	HB-H7	5/72	0.0025-0.5	5.90E-02			2.66E+00	C	2.44E+00	ca	2.44E+00 N BSL
	72-55-9	4,4'-DDE	0.0063 J	0.021 J	mg/kg	HB-H5	7/72	0.0025-0.5	2.10E-02			1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	50-29-3	4,4'-DDT	0.014 J	1.7 J	mg/kg	HB-T-3-OIL	6/72	0.0048-0.089	1.70E+00			1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	309-00-2	ALDRIN	0.0045 J	0.0091 J	mg/kg	HB-H3	3/72	0.0025-0.5	9.10E-03			3.76E-02	C	2.86E-02	ca	2.86E-02 N BSL
	57-74-9	TOTAL CHLORDANE ^g	0.0053	0.074	mg/kg	HB-H5	6/68	0.0025-5	7.40E-02			1.82E+00	C	1.62E+00	ca	1.62E+00 N BSL
	319-86-8	DELTA-BHC	0.0045 J	0.0045 J	mg/kg	HB-H3	1/72	0.0025-0.5	4.50E-03			NV	NV	NV	Y	NTX
	60-57-1	DIELDRIN	0.01 J	0.069 J	mg/kg	HB-H5	7/72	0.0025-0.5	6.90E-02			3.99E-02	C	3.04E-02	ca	3.04E-02 Y ASL
	33213-65-9	ENDOSULFAN II ⁹	0.0087 J	0.023 J	mg/kg	HB-H4	2/72	0.0048-1	2.30E-02			4.69E+01	N	3.67E+01	nc	3.67E+01 N BSL
	1031-07-8	ENDOSULFAN SULFATE ⁹	0.018 J	6.5 J	mg/kg	HB-T-3-OIL	3/72	0.0048-0.12	6.50E+00			4.69E+01	N	3.67E+01	nc	3.67E+01 N BSL
	72-20-8	ENDRIN	0.0081 J	0.027 J	mg/kg	HB-H5	2/72	0.0025-0.5	2.70E-02			2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL
	7421-93-4	ENDRIN ALDEHYDE ^h	0.0022 J	0.1 J	mg/kg	HB-H6	7/72	0.0048-1	1.00E-01			2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL
	53494-70-5	ENDRIN KETONE ^h	0.083 J	0.083 J	mg/kg	HB-H7	1/72	0.0048-1	8.30E-02			2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL
	58-89-9	GAMMA-BHC (LINDANE)	0.00071 J	0.0096 J	mg/kg	HB-H5	4/72	0.0025-0.5	9.60E-03			4.91E-01	C	4.37E-01	ca	4.37E-01 N BSL
	76-44-8	HEPTACHLOR	0.0013 J	0.015 J	mg/kg	HB-H7	5/72	0.0025-0.5	1.50E-02			1.42E-01	C	1.08E-01	ca	1.08E-01 N BSL
	1024-57-3	HEPTACHLOR EPOXIDE	0.004 J	0.03 J	mg/kg	HB-H3	7/72	0.0025-0.5	3.00E-02			7.02E-02	C	5.34E-02	ca	5.34E-02 N BSL
	SVOC															
	105-67-9	2,4-DIMETHYLPHENOL	0.11 J	23	mg/kg	HB-T-3-3	11/72	0.23-10000	2.30E+01			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	91-57-6	2-METHYLNAPHTHALENE	0.067	32000	mg/kg	HB-T-3-OIL	53/64	0.044-11	3.20E+04			3.13E+01	N	NV	NV	3.13E+01 Y ASL
	95-48-7	2-METHYLPHENOL	0.092	6.8	mg/kg	HB-T-3-2	14/71	0.044-10000	6.80E+00			3.91E+02	N	3.06E+02	nc	3.06E+02 N BSL
	34METPH	3,8,4-METHYLPHENOL ⁱ	0.053	12	mg/kg	HB-T-3-2	26/68	0.048-10000	1.20E+01			3.91E+01	N	3.06E+01	nc	3.06E+01 N BSL
	83-32-9	ACENAPHTHENE	0.086 J	6500 J	mg/kg	HB-T-3-OIL	56/65	0.044-4	6.50E+03			4.69E+02	N	3.68E+02	nc	3.68E+02 Y ASL
	208-96-8	ACENAPHTHYLENE	0.063	8300 J	mg/kg	HB-T-3-OIL	50/68	0.044-11	8.30E+03			NV	NV	NV	Y	NTX
	120-12-7	ANTHRACENE	0.11 J	5100 J	mg/kg	HB-T-3-OIL	54/65	0.048-4	5.10E+03			2.35E+03	N	2.19E+03	nc	2.19E+03 Y ASL
	56-55-3	BENZ(A)ANTHRACENE	0.069	1900 J	mg/kg	HB-T-3-OIL	57/65	0.056-5.7	1.90E+03			2.20E-01	C	6.21E-01	ca	6.21E-01 Y ASL
	50-32-8	BENZO(A)PYRENE	0.061	53	mg/kg	HB-T-5-1	51/66	0.056-10000	5.30E+01			2.20E-02	C	6.21E-02	ca	6.21E-02 Y ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.12	35	mg/kg	HB-T-5-1	45/65	0.056-10000	3.50E+01			2.20E-01	C	6.21E-01	ca	6.21E-01 Y ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.065	29	mg/kg	HB-T-5-1	49/69	0.056-10000	2.90E+01			NV	NV	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.06	42	mg/kg	HB-T-5-1	52/68	0.056-10000	4.20E+01			2.20E+00	C	6.21E+00	ca	6.21E+00 Y ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.055	4	mg/kg	HB-T-3-3	24/71	0.044-10000	4.00E+00			4.56E+01	C	3.47E+01	ca	3.47E+01 N BSL
	86-74-8	CARBAZOLE	0.069	1700 J	mg/kg	HB-T-3-OIL	52/69	0.044-26	1.70E+03			3.19E+01	C	2.43E+01	ca	2.43E+01 Y ASL

TABLE 2.19a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- HARBOR BROOK SUBSURFACE SEDIMENT
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Upper Sediment (0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
	218-01-9	CHRYSENE	0.069	1700 J	mg/kg	HB-T-3-OIL	58/66	0.056-5.7	1.70E+03			2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.055	11	mg/kg	HB-T-5-1	34/71	0.048-10000	1.10E+01			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.061 J	11000	mg/kg	HB-T-3-OIL	56/67	0.044-4	1.10E+04			7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.048	0.7	mg/kg	HB-T-1-2	8/72	0.048-10000	7.00E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	206-44-0	FLUORANTHENE	0.076	8300 J	mg/kg	HB-T-3-OIL	58/61	0.056-1.2	8.30E+03			3.13E+02	N	2.29E+02	nc	2.29E+02	Y	ASL
	86-73-7	FLUORENE	0.07	12000	mg/kg	HB-T-3-OIL	57/65	0.048-4	1.20E+04			3.13E+02	N	2.75E+02	nc	2.75E+02	Y	ASL
	118-74-1	HEXACHLOROBENZENE	0.53 J	0.53 J	mg/kg	HB-H2	1/71	0.044-10000	5.30E-01			3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.11	28	mg/kg	HB-T-5-1	47/68	0.056-10000	2.80E+01			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.11	97000	mg/kg	HB-T-3-OIL	55/61	0.048-0.061	9.70E+04			1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	85-01-8	PHENANTHRENE	0.08	23000	mg/kg	HB-T-3-OIL	56/59	0.06-1.2	2.30E+04			NV	NV	NV			Y	NTX
	108-95-2	PHENOL	0.065 J	12	mg/kg	HB-T-3-2	16/71	0.044-10000	1.20E+01			2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.084	5700 J	mg/kg	HB-T-3-OIL	58/62	0.056-4	5.70E+03			2.35E+02	N	2.32E+02	nc	2.32E+02	Y	ASL
	VOC																	
	71-55-6	1,1,1-TRICHLOROETHANE	0.084 J	0.084 J	mg/kg	HB-H3	1/73	0.0073-25	8.40E-02			1.56E+04	N	1.20E+02	nc	1.20E+02	N	BSL
	120-82-1	1,2,4-TRICHLOROBENZENE	3.4	8.1	mg/kg	HB-T-5-3	2/63	0.044-10000	8.10E+00			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	0.27	5.9	mg/kg	HB-T-1-3	4/70	0.044-10000	5.90E+00			7.04E+02	N	6.00E+01	nc	6.00E+01	N	BSL
	541-73-1	1,3-DICHLOROBENZENE	0.74 J	1.6	mg/kg	HB-T-1-2	3/71	0.044-10000	1.60E+00			2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.23 J	29	mg/kg	HB-T-5-3	7/70	0.044-10000	2.90E+01			2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL
	78-93-3	2-BUTANONE	0.007 J	13 J	mg/kg	HB-T-4-2	18/71	0.015-50	1.30E+01			4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	591-78-6	2-HEXANONE	12 J	12 J	mg/kg	HB-T-4-2	1/73	0.015-50	1.20E+01			NV	NV	NV			Y	NTX
	108-10-1	4-METHYL-2-PENTANONE	9.5 J	9.5 J	mg/kg	HB-T-4-2	1/73	0.015-50	9.50E+00			NV	NV	5.28E+02	nc	5.28E+02	N	BSL
	67-64-1	ACETONE	0.016 J	1.2 J	mg/kg	HB-T-3-1, HB-T-3-2	22/68	0.056-100	1.20E+00			7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.002 J	1100	mg/kg	HB-T-3-OIL	57/71	0.0083-4.8	1.10E+03			1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.0017	0.022 J	mg/kg	HB-T-2-3, HB-T-3-2	17/73	0.015-50	2.20E-02			7.82E+02	N	3.55E+01	nc	3.55E+01	Y	BSL
	108-90-7	CHLOROBENZENE	0.001 J	240	mg/kg	HB-T-5-3	15/70	0.0073-25	2.40E+02			1.56E+02	N	1.51E+01	nc	1.51E+01	Y	ASL
	100-41-4	ETHYLBENZENE	0.0023 J	1800	mg/kg	HB-T-3-OIL	56/68	0.0083-4.8	1.80E+03			7.82E+02	N	3.95E+01	nc	3.95E+01	Y	ASL
	75-09-2	METHYLENE CHLORIDE	0.003 J	30	mg/kg	HB-T-3-2	15/70	0.0074-140	3.00E+01			8.52E+01	C	9.11E+00	ca	9.11E+00	Y	ASL
	100-42-5	STYRENE	0.011 J	1700	mg/kg	HB-T-3-OIL	3/73	0.0073-21	1.70E+03			1.56E+03	N	1.70E+02	nc	1.70E+02	Y	ASL
	108-88-3	TOLUENE	0.0022 J	4400	mg/kg	HB-T-3-OIL	53/70	0.0073-4.8	4.40E+03			6.26E+02	N	5.20E+01	nc	5.20E+01	Y	ASL
	1330-20-7	XYLENES, TOTAL	0.0047	10500	mg/kg	HB-T-3-OIL	63/72	0.008-34.8	1.05E+04			1.56E+03	N	2.71E+01	nc	2.71E+01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.19b.
- = Compound detected in 100% of samples.
- NA = Not applicable, minimum and maximum values are calculated.
- a = RBC and PRG values for chromium VI utilized.
- b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
- c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
- d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on 1254.
- e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on 1254.
- f = Where criteria are not available, RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
- g = RBC and PRG values for Endosulfan (CAS# 115297) utilized.
- h = RBC and PRG values for Endrin (CAS# 72208) utilized.
- i = RBC and PRG values for 4-methylphenol utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-14	11/14/2002	0	0.33	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	4.301	4.301	ng/kg		0.01	0.043
HB-HBSED-14	11/14/2002	0	0.33	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.548	0.274	ng/kg	U	0.01	0.003
HB-HBSED-14	11/14/2002	0	0.33	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.291	0.1455	ng/kg	U	0.1	0.015
HB-HBSED-14	11/14/2002	0	0.33	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.227	0.1135	ng/kg	U	0.1	0.011
HB-HBSED-14	11/14/2002	0	0.33	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.073	1.073	ng/kg	J	0.1	0.107
HB-HBSED-14	11/14/2002	0	0.33	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.217	0.1085	ng/kg	U	0.1	0.011
HB-HBSED-14	11/14/2002	0	0.33	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.27	0.135	ng/kg	U	0.1	0.014
HB-HBSED-14	11/14/2002	0	0.33	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.275	0.1375	ng/kg	U	0.1	0.014
HB-HBSED-14	11/14/2002	0	0.33	40321-76-4	1,2,3,7,8-PECDD	N	0.38	0.19	ng/kg	U	1	0.190
HB-HBSED-14	11/14/2002	0	0.33	57117-41-6	1,2,3,7,8-PECDF	N	0.273	0.1365	ng/kg	U	0.03	0.004
HB-HBSED-14	11/14/2002	0	0.33	1746-01-6	2,3,7,8-TCDD	N	0.377	0.1885	ng/kg	U	1	0.189
HB-HBSED-14	11/14/2002	0	0.33	51207-31-9	2,3,7,8-TCDF	N	0.353	0.1765	ng/kg	U	0.1	0.018
HB-HBSED-14	11/14/2002	0	0.33	3268-87-9	OCDD	Y	161.945	161.945	ng/kg		0.0003	0.049
HB-HBSED-14	11/14/2002	0	0.33	39001-02-0	OCDF	Y	10.146	10.146	ng/kg	EMPC	0.0003	0.003
Sample Location TEQ =												0.7
HB-HBSED-15	11/14/2002	0	0.25	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	22.716	22.716	ng/kg		0.01	0.227
HB-HBSED-15	11/14/2002	0	0.25	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.817	0.4085	ng/kg	U	0.01	0.004
HB-HBSED-15	11/14/2002	0	0.25	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.417	0.2085	ng/kg	U	0.1	0.021
HB-HBSED-15	11/14/2002	0	0.25	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.497	1.497	ng/kg	J	0.1	0.150
HB-HBSED-15	11/14/2002	0	0.25	57653-85-7	1,2,3,6,7,8-HXCDD	Y	4.201	4.201	ng/kg		0.1	0.420
HB-HBSED-15	11/14/2002	0	0.25	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.919	0.919	ng/kg	J	0.1	0.092
HB-HBSED-15	11/14/2002	0	0.25	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.269	3.269	ng/kg		0.1	0.327
HB-HBSED-15	11/14/2002	0	0.25	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.366	0.183	ng/kg	U	0.1	0.018
HB-HBSED-15	11/14/2002	0	0.25	40321-76-4	1,2,3,7,8-PECDD	N	0.489	0.2445	ng/kg	U	1	0.245
HB-HBSED-15	11/14/2002	0	0.25	57117-41-6	1,2,3,7,8-PECDF	N	0.349	0.1745	ng/kg	U	0.03	0.005
HB-HBSED-15	11/14/2002	0	0.25	1746-01-6	2,3,7,8-TCDD	N	0.496	0.248	ng/kg	U	1	0.248
HB-HBSED-15	11/14/2002	0	0.25	51207-31-9	2,3,7,8-TCDF	N	0.399	0.1995	ng/kg	U	0.1	0.020
HB-HBSED-15	11/14/2002	0	0.25	3268-87-9	OCDD	Y	881.1	881.1	ng/kg		0.0003	0.264
HB-HBSED-15	11/14/2002	0	0.25	39001-02-0	OCDF	Y	75.394	75.394	ng/kg		0.0003	0.023
Sample Location TEQ =												2.1
HB-HBSED-16	6/2/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	62.956	62.956	ng/kg	J	0.01	0.630
HB-HBSED-16	6/2/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	1.703	0.8515	ng/kg	UJ	0.01	0.009
HB-HBSED-16	6/2/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.212	1.212	ng/kg	J	0.1	0.121
HB-HBSED-16	6/2/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	4.006	4.006	ng/kg		0.1	0.401
HB-HBSED-16	6/2/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	6.542	6.542	ng/kg		0.1	0.654
HB-HBSED-16	6/2/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.01	2.01	ng/kg	J	0.1	0.201
HB-HBSED-16	6/2/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.741	3.741	ng/kg	J	0.1	0.374
HB-HBSED-16	6/2/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.61	0.305	ng/kg	U	0.1	0.031
HB-HBSED-16	6/2/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.998	0.998	ng/kg	J	0.03	0.030
HB-HBSED-16	6/2/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.389	0.1945	ng/kg	U	1	0.195
HB-HBSED-16	6/2/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.96	2.96	ng/kg		0.1	0.296
HB-HBSED-16	6/2/2003	0	0.5	3268-87-9	OCDD	Y	1700.214	1700.214	ng/kg	J	0.0003	0.510
HB-HBSED-16	6/2/2003	0	0.5	39001-02-0	OCDF	Y	239.938	239.938	ng/kg	J	0.0003	0.072
Sample Location TEQ =												3.5

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-19	6/4/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	37.284	37.284	ng/kg		0.01	0.373
HB-HBSED-19	6/4/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	1.44	0.72	ng/kg	UJ	0.01	0.007
HB-HBSED-19	6/4/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.221	2.221	ng/kg	J	0.1	0.222
HB-HBSED-19	6/4/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.592	2.592	ng/kg	EMPC	0.1	0.259
HB-HBSED-19	6/4/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.593	5.593	ng/kg		0.1	0.559
HB-HBSED-19	6/4/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.358	5.358	ng/kg		0.1	0.536
HB-HBSED-19	6/4/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.738	0.369	ng/kg	U	0.1	0.037
HB-HBSED-19	6/4/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.462	1.462	ng/kg	J	1	1.462
HB-HBSED-19	6/4/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.366	0.183	ng/kg	U	1	0.183
HB-HBSED-19	6/4/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.03	2.03	ng/kg		0.1	0.203
HB-HBSED-19	6/4/2003	0	0.5	3268-87-9	OCDD	Y	1049.111	1049.111	ng/kg	J	0.0003	0.315
HB-HBSED-19	6/4/2003	0	0.5	39001-02-0	OCDF	Y	188.842	188.842	ng/kg	J	0.0003	0.057
Sample Location TEQ =												4.2
HB-HBSED-19	6/4/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	43.776	43.776	ng/kg		0.01	0.438
HB-HBSED-19	6/4/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.813	1.813	ng/kg	J	0.1	0.181
HB-HBSED-19	6/4/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.814	3.814	ng/kg		0.1	0.381
HB-HBSED-19	6/4/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.999	7.999	ng/kg		0.1	0.800
HB-HBSED-19	6/4/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.719	2.719	ng/kg		0.1	0.272
HB-HBSED-19	6/4/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.163	6.163	ng/kg		0.1	0.616
HB-HBSED-19	6/4/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.708	0.354	ng/kg	U	0.1	0.035
HB-HBSED-19	6/4/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	0.819	0.819	ng/kg	J	0.03	0.025
HB-HBSED-19	6/4/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	0.419	0.419	ng/kg	J	1	0.419
HB-HBSED-19	6/4/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	1.58	1.58	ng/kg		0.1	0.158
HB-HBSED-19	6/4/2003	0.5	1	3268-87-9	OCDD	Y	1109.094	1109.094	ng/kg	J	0.0003	0.333
HB-HBSED-19	6/4/2003	0.5	1	39001-02-0	OCDF	Y	128.018	128.018	ng/kg	J	0.0003	0.038
Sample Location TEQ =												3.7
HB-HBSED-20	6/4/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	16.022	16.022	ng/kg		0.01	0.160
HB-HBSED-20	6/4/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.005	2.005	ng/kg	J	0.1	0.201
HB-HBSED-20	6/4/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.636	1.636	ng/kg	J	0.1	0.164
HB-HBSED-20	6/4/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.672	5.672	ng/kg		0.1	0.567
HB-HBSED-20	6/4/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.141	1.141	ng/kg	J	0.1	0.114
HB-HBSED-20	6/4/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.559	5.559	ng/kg		0.1	0.556
HB-HBSED-20	6/4/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.205	0.1025	ng/kg	U	0.1	0.010
HB-HBSED-20	6/4/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.119	0.0595	ng/kg	U	1	0.060
HB-HBSED-20	6/4/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.804	0.804	ng/kg	J	0.1	0.080
HB-HBSED-20	6/4/2003	0	0.5	3268-87-9	OCDD	Y	646.345	646.345	ng/kg	J	0.0003	0.194
HB-HBSED-20	6/4/2003	0	0.5	39001-02-0	OCDF	Y	29.292	29.292	ng/kg	J	0.0003	0.009
Sample Location TEQ =												2.1

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-S-1	1/31/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	37.747	37.747	ng/kg	J	0.01	0.377
HB-S-1	1/31/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	8.997	8.997	ng/kg	J	0.1	0.900
HB-S-1	1/31/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.553	5.553	ng/kg	J	0.1	0.555
HB-S-1	1/31/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	4.234	4.234	ng/kg	J	0.1	0.423
HB-S-1	1/31/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.759	1.759	ng/kg	J	0.1	0.176
HB-S-1	1/31/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.53	0.265	ng/kg	UJ	0.1	0.027
HB-S-1	1/31/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	2.058	2.058	ng/kg	J	0.03	0.062
HB-S-1	1/31/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.632	0.316	ng/kg	UJ	1	0.316
HB-S-1	1/31/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.17	1.17	ng/kg	J	0.1	0.117
HB-S-1	1/31/2001	0	0.5	3268-87-9	OCDD	Y	1420.293	1420.293	ng/kg	J	0.0003	0.426
HB-S-1	1/31/2001	0	0.5	39001-02-0	OCDF	Y	121.707	121.707	ng/kg	J	0.0003	0.037
Sample Location TEQ =												3.4
HB-S-2	1/31/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	27.814	27.814	ng/kg		0.01	0.278
HB-S-2	1/31/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.367	1.367	ng/kg	J	0.1	0.137
HB-S-2	1/31/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.068	2.068	ng/kg	J	0.1	0.207
HB-S-2	1/31/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.113	5.113	ng/kg		0.1	0.511
HB-S-2	1/31/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.825	1.825	ng/kg	J	0.1	0.183
HB-S-2	1/31/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.401	0.2005	ng/kg	U	0.1	0.020
HB-S-2	1/31/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.334	0.167	ng/kg	UJ	0.03	0.005
HB-S-2	1/31/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.568	0.284	ng/kg	UJ	1	0.284
HB-S-2	1/31/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.33	1.33	ng/kg		0.1	0.133
HB-S-2	1/31/2001	0	0.5	3268-87-9	OCDD	Y	912.452	912.452	ng/kg		0.0003	0.274
HB-S-2	1/31/2001	0	0.5	39001-02-0	OCDF	Y	74.485	74.485	ng/kg		0.0003	0.022
Sample Location TEQ =												2.1
HB-T-1-2	1/24/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	84.417	84.417	ng/kg		0.01	0.844
HB-T-1-2	1/24/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	27.021	27.021	ng/kg		0.01	0.270
HB-T-1-2	1/24/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	4.534	4.534	ng/kg		0.1	0.453
HB-T-1-2	1/24/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	90.244	90.244	ng/kg		0.1	9.024
HB-T-1-2	1/24/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	17.131	17.131	ng/kg		0.1	1.713
HB-T-1-2	1/24/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	29.579	29.579	ng/kg		0.1	2.958
HB-T-1-2	1/24/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	7.816	7.816	ng/kg	J	0.1	0.782
HB-T-1-2	1/24/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	20.203	20.203	ng/kg		0.1	2.020
HB-T-1-2	1/24/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	54.334	54.334	ng/kg		0.03	1.630
HB-T-1-2	1/24/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.314	1.314	ng/kg		1	1.314
HB-T-1-2	1/24/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	63.5	63.5	ng/kg		0.1	6.350
HB-T-1-2	1/24/2001	0	0.5	3268-87-9	OCDD	Y	673.904	673.904	ng/kg		0.0003	0.202
HB-T-1-2	1/24/2001	0	0.5	39001-02-0	OCDF	Y	154.272	154.272	ng/kg		0.0003	0.046
Sample Location TEQ =												27.6

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-1-2	1/24/2001	0.5	1.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	325.41	325.41	ng/kg	J	0.01	3.254
HB-T-1-2	1/24/2001	0.5	1.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	76.486	76.486	ng/kg	J	0.01	0.765
HB-T-1-2	1/24/2001	0.5	1.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	437.923	437.923	ng/kg	J	0.1	43.792
HB-T-1-2	1/24/2001	0.5	1.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	33.969	33.969	ng/kg	J	0.1	3.397
HB-T-1-2	1/24/2001	0.5	1.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	441.64	441.64	ng/kg	J	0.1	44.164
HB-T-1-2	1/24/2001	0.5	1.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	11.567	11.567	ng/kg	J	0.1	1.157
HB-T-1-2	1/24/2001	0.5	1.5	57117-41-6	1,2,3,7,8-PECDF	Y	226.03	226.03	ng/kg		0.03	6.781
HB-T-1-2	1/24/2001	0.5	1.5	51207-31-9	2,3,7,8-TCDF	Y	277.9	277.9	ng/kg	J	0.1	27.790
HB-T-1-2	1/24/2001	0.5	1.5	3268-87-9	OCDD	Y	904.29	904.29	ng/kg	J	0.0003	0.271
HB-T-1-2	1/24/2001	0.5	1.5	39001-02-0	OCDF	Y	318.455	318.455	ng/kg	J	0.0003	0.096
Sample Location TEQ = 131.5												
HB-T-1-2	1/24/2001	1.5	4.4	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	147.994	147.994	ng/kg		0.01	1.480
HB-T-1-2	1/24/2001	1.5	4.4	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	18.017	18.017	ng/kg		0.01	0.180
HB-T-1-2	1/24/2001	1.5	4.4	70648-26-9	1,2,3,4,7,8-HXCDF	Y	57.625	57.625	ng/kg		0.1	5.763
HB-T-1-2	1/24/2001	1.5	4.4	57653-85-7	1,2,3,6,7,8-HXCDD	Y	19.152	19.152	ng/kg		0.1	1.915
HB-T-1-2	1/24/2001	1.5	4.4	57117-44-9	1,2,3,6,7,8-HXCDF	Y	15.949	15.949	ng/kg		0.1	1.595
HB-T-1-2	1/24/2001	1.5	4.4	19408-74-3	1,2,3,7,8,9-HXCDD	Y	8.373	8.373	ng/kg	J	0.1	0.837
HB-T-1-2	1/24/2001	1.5	4.4	72918-21-9	1,2,3,7,8,9-HXCDF	Y	13.712	13.712	ng/kg		0.1	1.371
HB-T-1-2	1/24/2001	1.5	4.4	57117-41-6	1,2,3,7,8-PECDF	Y	28.216	28.216	ng/kg		0.03	0.846
HB-T-1-2	1/24/2001	1.5	4.4	1746-01-6	2,3,7,8-TCDD	Y	2.372	2.372	ng/kg		1	2.372
HB-T-1-2	1/24/2001	1.5	4.4	51207-31-9	2,3,7,8-TCDF	Y	73.82	73.82	ng/kg		0.1	7.382
HB-T-1-2	1/24/2001	1.5	4.4	3268-87-9	OCDD	Y	343.845	343.845	ng/kg	J	0.0003	0.103
HB-T-1-2	1/24/2001	1.5	4.4	39001-02-0	OCDF	Y	155.744	155.744	ng/kg	J	0.0003	0.047
Sample Location TEQ = 23.9												
HB-T-1-2	1/24/2001	4.4	7.4	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.449	8.449	ng/kg		0.01	0.084
HB-T-1-2	1/24/2001	4.4	7.4	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.433	0.2165	ng/kg	U	0.01	0.002
HB-T-1-2	1/24/2001	4.4	7.4	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.843	3.843	ng/kg		0.1	0.384
HB-T-1-2	1/24/2001	4.4	7.4	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.049	3.049	ng/kg		0.1	0.305
HB-T-1-2	1/24/2001	4.4	7.4	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.841	1.841	ng/kg	J	0.1	0.184
HB-T-1-2	1/24/2001	4.4	7.4	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.514	1.514	ng/kg	J	0.1	0.151
HB-T-1-2	1/24/2001	4.4	7.4	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.344	0.344	ng/kg	J	0.1	0.034
HB-T-1-2	1/24/2001	4.4	7.4	57117-41-6	1,2,3,7,8-PECDF	Y	2.504	2.504	ng/kg		0.03	0.075
HB-T-1-2	1/24/2001	4.4	7.4	1746-01-6	2,3,7,8-TCDD	N	0.228	0.114	ng/kg	U	1	0.114
HB-T-1-2	1/24/2001	4.4	7.4	51207-31-9	2,3,7,8-TCDF	Y	6.2	6.2	ng/kg		0.1	0.620
HB-T-1-2	1/24/2001	4.4	7.4	3268-87-9	OCDD	Y	24.337	24.337	ng/kg	J	0.0003	0.007
HB-T-1-2	1/24/2001	4.4	7.4	39001-02-0	OCDF	Y	4.24	4.24	ng/kg	J	0.0003	0.001
Sample Location TEQ = 2.0												

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-1-2	1/24/2001	7.4	8.1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	20.747	20.747	ng/kg		0.01	0.207
HB-T-1-2	1/24/2001	7.4	8.1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.847	0.847	ng/kg		0.01	0.008
HB-T-1-2	1/24/2001	7.4	8.1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	14.214	14.214	ng/kg		0.1	1.421
HB-T-1-2	1/24/2001	7.4	8.1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.555	5.555	ng/kg		0.1	0.556
HB-T-1-2	1/24/2001	7.4	8.1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.913	7.913	ng/kg		0.1	0.791
HB-T-1-2	1/24/2001	7.4	8.1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.656	5.656	ng/kg	J	0.1	0.566
HB-T-1-2	1/24/2001	7.4	8.1	72918-21-9	1,2,3,7,8,9-HXCDF	N	8.628	4.314	ng/kg	U	0.1	0.431
HB-T-1-2	1/24/2001	7.4	8.1	57117-41-6	1,2,3,7,8-PECDF	Y	8.633	8.633	ng/kg		0.03	0.259
HB-T-1-2	1/24/2001	7.4	8.1	1746-01-6	2,3,7,8-TCDD	Y	1.199	1.199	ng/kg		1	1.199
HB-T-1-2	1/24/2001	7.4	8.1	51207-31-9	2,3,7,8-TCDF	Y	15.46	15.46	ng/kg		0.1	1.546
HB-T-1-2	1/24/2001	7.4	8.1	3268-87-9	OCDD	Y	65.484	65.484	ng/kg		0.0003	0.020
HB-T-1-2	1/24/2001	7.4	8.1	39001-02-0	OCDF	N	18.399	9.1995	ng/kg	UJ	0.0003	0.003
Sample Location TEQ =												7.0
HB-T-2-3	1/25/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	328.425	328.425	ng/kg	J	0.01	3.284
HB-T-2-3	1/25/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	25.007	25.007	ng/kg	J	0.01	0.250
HB-T-2-3	1/25/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.235	6.235	ng/kg	J	0.1	0.624
HB-T-2-3	1/25/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	61.935	61.935	ng/kg	J	0.1	6.194
HB-T-2-3	1/25/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	51.72	51.72	ng/kg	J	0.1	5.172
HB-T-2-3	1/25/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	24.718	24.718	ng/kg	J	0.1	2.472
HB-T-2-3	1/25/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	18.23	18.23	ng/kg	J	0.1	1.823
HB-T-2-3	1/25/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	15.997	15.997	ng/kg	J	0.1	1.600
HB-T-2-3	1/25/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	9.719	9.719	ng/kg		1	9.719
HB-T-2-3	1/25/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	28.773	28.773	ng/kg		0.03	0.863
HB-T-2-3	1/25/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.583	2.583	ng/kg		1	2.583
HB-T-2-3	1/25/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	54.69	54.69	ng/kg	J	0.1	5.469
HB-T-2-3	1/25/2001	0	0.5	3268-87-9	OCDD	Y	1477.489	1477.489	ng/kg	J	0.0003	0.443
HB-T-2-3	1/25/2001	0	0.5	39001-02-0	OCDF	Y	298.975	298.975	ng/kg	J	0.0003	0.090
Sample Location TEQ =												40.6
HB-T-2-3	1/25/2001	0.5	1.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	228.292	228.292	ng/kg	J	0.01	2.2829
HB-T-2-3	1/25/2001	0.5	1.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	23.728	23.728	ng/kg	J	0.01	0.2373
HB-T-2-3	1/25/2001	0.5	1.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	1.324	0.662	ng/kg	UJ	0.1	0.0662
HB-T-2-3	1/25/2001	0.5	1.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	58.593	58.593	ng/kg	J	0.1	5.8593
HB-T-2-3	1/25/2001	0.5	1.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	15.164	15.164	ng/kg	J	0.1	1.5164
HB-T-2-3	1/25/2001	0.5	1.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.68	6.68	ng/kg	J	0.1	0.6680
HB-T-2-3	1/25/2001	0.5	1.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	11.423	11.423	ng/kg	J	0.1	1.1423
HB-T-2-3	1/25/2001	0.5	1.5	40321-76-4	1,2,3,7,8-PECDD	Y	4.151	4.151	ng/kg		1	4.151
HB-T-2-3	1/25/2001	0.5	1.5	57117-41-6	1,2,3,7,8-PECDF	Y	14.57	14.57	ng/kg		0.03	0.4371
HB-T-2-3	1/25/2001	0.5	1.5	1746-01-6	2,3,7,8-TCDD	N	1.090	0.545	ng/kg	U	1	0.545
HB-T-2-3	1/25/2001	0.5	1.5	51207-31-9	2,3,7,8-TCDF	Y	53.5	53.5	ng/kg		0.1	5.350
HB-T-2-3	1/25/2001	0.5	1.5	3268-87-9	OCDD	Y	420.017	420.017	ng/kg	J	0.0003	0.126
HB-T-2-3	1/25/2001	0.5	1.5	39001-02-0	OCDF	Y	302.27	302.27	ng/kg	J	0.0003	0.091
Sample Location TEQ =												22.5

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-2-3	1/25/2001	1.5	4.4	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	3.292	3.292	ng/kg		0.01	0.033
HB-T-2-3	1/25/2001	1.5	4.4	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.211	0.1055	ng/kg	U	0.01	0.001
HB-T-2-3	1/25/2001	1.5	4.4	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.096	1.096	ng/kg	J	0.1	0.110
HB-T-2-3	1/25/2001	1.5	4.4	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.951	0.951	ng/kg	J	0.1	0.095
HB-T-2-3	1/25/2001	1.5	4.4	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.105	1.105	ng/kg	J	0.1	0.111
HB-T-2-3	1/25/2001	1.5	4.4	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.142	0.071	ng/kg	U	0.1	0.007
HB-T-2-3	1/25/2001	1.5	4.4	57117-41-6	1,2,3,7,8-PECDF	Y	1.431	1.431	ng/kg	J	0.03	0.043
HB-T-2-3	1/25/2001	1.5	4.4	1746-01-6	2,3,7,8-TCDD	Y	0.274	0.274	ng/kg	J	1	0.274
HB-T-2-3	1/25/2001	1.5	4.4	51207-31-9	2,3,7,8-TCDF	Y	3.56	3.56	ng/kg		0.1	0.356
Sample Location TEQ = 1.0												
HB-T-2-3	1/25/2001	4.4	7.4	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	4.315	4.315	ng/kg		0.01	0.043
HB-T-2-3	1/25/2001	4.4	7.4	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.125	0.0625	ng/kg	U	0.1	0.006
HB-T-2-3	1/25/2001	4.4	7.4	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.127	2.127	ng/kg	J	0.1	0.213
HB-T-2-3	1/25/2001	4.4	7.4	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.662	0.662	ng/kg	J	0.1	0.066
HB-T-2-3	1/25/2001	4.4	7.4	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.904	0.904	ng/kg	J	0.1	0.090
HB-T-2-3	1/25/2001	4.4	7.4	19408-74-3	1,2,3,7,8,9-HXCDD	Y	0.681	0.681	ng/kg	J	0.1	0.068
HB-T-2-3	1/25/2001	4.4	7.4	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.171	0.0855	ng/kg	U	0.1	0.009
HB-T-2-3	1/25/2001	4.4	7.4	40321-76-4	1,2,3,7,8-PECDD	N	0.1	0.05	ng/kg	U	1	0.050
HB-T-2-3	1/25/2001	4.4	7.4	57117-41-6	1,2,3,7,8-PECDF	Y	1.195	1.195	ng/kg	J	0.03	0.036
HB-T-2-3	1/25/2001	4.4	7.4	1746-01-6	2,3,7,8-TCDD	N	0.07	0.035	ng/kg	U	1	0.035
HB-T-2-3	1/25/2001	4.4	7.4	51207-31-9	2,3,7,8-TCDF	Y	2.14	2.14	ng/kg	J	0.1	0.214
HB-T-2-3	1/25/2001	4.4	7.4	3268-87-9	OCDD	Y	8.939	8.939	ng/kg		0.0003	0.003
HB-T-2-3	1/25/2001	4.4	7.4	39001-02-0	OCDF	Y	2.137	2.137	ng/kg	J	0.0003	0.001
Sample Location TEQ = 0.8												
HB-T-2-3	1/25/2001	7.4	9.8	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.254	0.127	ng/kg	U	0.01	0.001
HB-T-2-3	1/25/2001	7.4	9.8	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.11	0.055	ng/kg	U	0.1	0.006
HB-T-2-3	1/25/2001	7.4	9.8	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.674	0.674	ng/kg	J	0.1	0.067
HB-T-2-3	1/25/2001	7.4	9.8	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.126	0.063	ng/kg	U	0.1	0.006
HB-T-2-3	1/25/2001	7.4	9.8	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.109	0.0545	ng/kg	U	0.1	0.005
HB-T-2-3	1/25/2001	7.4	9.8	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.116	0.058	ng/kg	U	0.1	0.006
HB-T-2-3	1/25/2001	7.4	9.8	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.123	0.0615	ng/kg	U	0.1	0.006
HB-T-2-3	1/25/2001	7.4	9.8	40321-76-4	1,2,3,7,8-PECDD	N	0.096	0.048	ng/kg	U	1	0.048
HB-T-2-3	1/25/2001	7.4	9.8	57117-41-6	1,2,3,7,8-PECDF	Y	0.307	0.307	ng/kg	J	0.03	0.009
HB-T-2-3	1/25/2001	7.4	9.8	1746-01-6	2,3,7,8-TCDD	N	0.086	0.043	ng/kg	U	1	0.043
HB-T-2-3	1/25/2001	7.4	9.8	39001-02-0	OCDF	N	0.185	0.0925	ng/kg	U	0.0003	0.0000
Sample Location TEQ = 0.2												

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-3-2	1/26/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	41.508	41.508	ng/kg		0.01	0.415
HB-T-3-2	1/26/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	3.149	3.149	ng/kg		0.01	0.031
HB-T-3-2	1/26/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.975	1.975	ng/kg	J	0.1	0.198
HB-T-3-2	1/26/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.704	7.704	ng/kg		0.1	0.770
HB-T-3-2	1/26/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.522	8.522	ng/kg		0.1	0.852
HB-T-3-2	1/26/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.486	3.486	ng/kg		0.1	0.349
HB-T-3-2	1/26/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.648	5.648	ng/kg	J	0.1	0.565
HB-T-3-2	1/26/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	3.04	3.04	ng/kg		0.03	0.091
HB-T-3-2	1/26/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	0.874	0.874	ng/kg	J	1	0.874
HB-T-3-2	1/26/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	6.7	6.7	ng/kg	J	0.1	0.670
HB-T-3-2	1/26/2001	0	0.5	3268-87-9	OCDD	Y	1197.39	1197.39	ng/kg	J	0.0003	0.359
HB-T-3-2	1/26/2001	0	0.5	39001-02-0	OCDF	Y	109.18	109.18	ng/kg		0.0003	0.033
Sample Location TEQ =											5.2	
HB-T-3-2	1/26/2001	0.5	1.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	16.926	16.926	ng/kg	J	0.01	0.169
HB-T-3-2	1/26/2001	0.5	1.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.728	1.728	ng/kg	J	0.01	0.017
HB-T-3-2	1/26/2001	0.5	1.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.256	0.128	ng/kg	UJ	0.1	0.013
HB-T-3-2	1/26/2001	0.5	1.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	4.121	4.121	ng/kg	J	0.1	0.412
HB-T-3-2	1/26/2001	0.5	1.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.767	3.767	ng/kg	J	0.1	0.377
HB-T-3-2	1/26/2001	0.5	1.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.809	1.809	ng/kg	J	0.1	0.181
HB-T-3-2	1/26/2001	0.5	1.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.715	1.715	ng/kg	J	0.1	0.172
HB-T-3-2	1/26/2001	0.5	1.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.349	0.1745	ng/kg	UJ	0.1	0.017
HB-T-3-2	1/26/2001	0.5	1.5	57117-41-6	1,2,3,7,8-PECDF	Y	2.076	2.076	ng/kg	J	0.03	0.062
HB-T-3-2	1/26/2001	0.5	1.5	1746-01-6	2,3,7,8-TCDD	N	0.403	0.2015	ng/kg	U	1	0.202
HB-T-3-2	1/26/2001	0.5	1.5	51207-31-9	2,3,7,8-TCDF	Y	4.4	4.4	ng/kg		0.1	0.440
HB-T-3-2	1/26/2001	0.5	1.5	3268-87-9	OCDD	Y	336.176	336.176	ng/kg	J	0.0003	0.101
HB-T-3-2	1/26/2001	0.5	1.5	39001-02-0	OCDF	Y	32.192	32.192	ng/kg	J	0.0003	0.010
Sample Location TEQ =											2.2	
HB-T-3-2	1/26/2001	1.5	4.4	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.567	8.567	ng/kg		0.01	0.086
HB-T-3-2	1/26/2001	1.5	4.4	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.876	0.876	ng/kg	J	0.01	0.009
HB-T-3-2	1/26/2001	1.5	4.4	70648-26-9	1,2,3,4,7,8-HXCDF	Y	5.803	5.803	ng/kg		0.1	0.580
HB-T-3-2	1/26/2001	1.5	4.4	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.431	1.431	ng/kg	J	0.1	0.143
HB-T-3-2	1/26/2001	1.5	4.4	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.384	2.384	ng/kg	J	0.1	0.238
HB-T-3-2	1/26/2001	1.5	4.4	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.161	1.161	ng/kg	J	0.1	0.116
HB-T-3-2	1/26/2001	1.5	4.4	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.386	0.193	ng/kg	U	0.1	0.019
HB-T-3-2	1/26/2001	1.5	4.4	57117-41-6	1,2,3,7,8-PECDF	Y	3.225	3.225	ng/kg		0.03	0.097
HB-T-3-2	1/26/2001	1.5	4.4	1746-01-6	2,3,7,8-TCDD	N	0.556	0.278	ng/kg	U	1	0.278
HB-T-3-2	1/26/2001	1.5	4.4	51207-31-9	2,3,7,8-TCDF	Y	6.1	6.1	ng/kg		0.1	0.610
HB-T-3-2	1/26/2001	1.5	4.4	3268-87-9	OCDD	Y	18.368	18.368	ng/kg		0.0003	0.006
HB-T-3-2	1/26/2001	1.5	4.4	39001-02-0	OCDF	Y	4.173	4.173	ng/kg	J	0.0003	0.001
Sample Location TEQ =											2.2	

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-3-2	1/26/2001	4.4	7.4	67562-39-4	1,2,3,4,6,7,8-HPCDF	N	0.256	0.128	ng/kg	U	0.01	0.001
HB-T-3-2	1/26/2001	4.4	7.4	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.322	0.161	ng/kg	U	0.01	0.002
HB-T-3-2	1/26/2001	4.4	7.4	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.242	0.121	ng/kg	U	0.1	0.012
HB-T-3-2	1/26/2001	4.4	7.4	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.178	0.089	ng/kg	U	0.1	0.009
HB-T-3-2	1/26/2001	4.4	7.4	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.277	0.1385	ng/kg	U	0.1	0.014
HB-T-3-2	1/26/2001	4.4	7.4	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.208	0.104	ng/kg	U	0.1	0.010
HB-T-3-2	1/26/2001	4.4	7.4	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.255	0.1275	ng/kg	U	0.1	0.013
HB-T-3-2	1/26/2001	4.4	7.4	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.235	0.1175	ng/kg	U	0.1	0.012
HB-T-3-2	1/26/2001	4.4	7.4	40321-76-4	1,2,3,7,8-PECDD	N	0.312	0.156	ng/kg	U	1	0.156
HB-T-3-2	1/26/2001	4.4	7.4	57117-41-6	1,2,3,7,8-PECDF	N	0.239	0.1195	ng/kg	U	0.03	0.004
HB-T-3-2	1/26/2001	4.4	7.4	1746-01-6	2,3,7,8-TCDD	N	0.373	0.1865	ng/kg	U	1	0.187
HB-T-3-2	1/26/2001	4.4	7.4	51207-31-9	2,3,7,8-TCDF	N	0.84	0.42	ng/kg	U	0.1	0.042
HB-T-3-2	1/26/2001	4.4	7.4	39001-02-0	OCDF	N	0.557	0.2785	ng/kg	U	0.0003	0.0001
Sample Location TEQ = 0.5												
HB-T-3-2	1/26/2001	7.4	9.2	67562-39-4	1,2,3,4,6,7,8-HPCDF	N	0.158	0.079	ng/kg	U	0.01	0.001
HB-T-3-2	1/26/2001	7.4	9.2	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.199	0.0995	ng/kg	U	0.01	0.001
HB-T-3-2	1/26/2001	7.4	9.2	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.134	0.067	ng/kg	U	0.1	0.007
HB-T-3-2	1/26/2001	7.4	9.2	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.078	0.039	ng/kg	U	0.1	0.004
HB-T-3-2	1/26/2001	7.4	9.2	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.153	0.0765	ng/kg	U	0.1	0.008
HB-T-3-2	1/26/2001	7.4	9.2	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.092	0.046	ng/kg	U	0.1	0.005
HB-T-3-2	1/26/2001	7.4	9.2	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.141	0.0705	ng/kg	U	0.1	0.007
HB-T-3-2	1/26/2001	7.4	9.2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.104	0.052	ng/kg	U	0.1	0.005
HB-T-3-2	1/26/2001	7.4	9.2	40321-76-4	1,2,3,7,8-PECDD	N	0.142	0.071	ng/kg	U	1	0.071
HB-T-3-2	1/26/2001	7.4	9.2	57117-41-6	1,2,3,7,8-PECDF	N	0.083	0.0415	ng/kg	U	0.03	0.001
HB-T-3-2	1/26/2001	7.4	9.2	1746-01-6	2,3,7,8-TCDD	N	0.128	0.064	ng/kg	U	1	0.064
HB-T-3-2	1/26/2001	7.4	9.2	51207-31-9	2,3,7,8-TCDF	N	0.098	0.049	ng/kg	U	0.1	0.005
HB-T-3-2	1/26/2001	7.4	9.2	39001-02-0	OCDF	N	0.246	0.123	ng/kg	U	0.0003	0.0000
Sample Location TEQ = 0.2												
HB-T-3-OIL	2/13/2001	0	0	40321-76-4	1,2,3,7,8-PECDD	N	9.751	4.8755	ng/kg	UJ	1	4.876
HB-T-3-OIL	2/13/2001	0	0	57117-41-6	1,2,3,7,8-PECDF	N	16.464	8.232	ng/kg	UJ	0.03	0.247
HB-T-3-OIL	2/13/2001	0	0	1746-01-6	2,3,7,8-TCDD	N	40.201	20.1005	ng/kg	UJ	1	20.101
HB-T-3-OIL	2/13/2001	0	0	51207-31-9	2,3,7,8-TCDF	N	41.48	20.74	ng/kg	UJ	0.1	2.074
Sample Location TEQ = 27.3												
HB-T-4-2	1/29/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	16.947	16.947	ng/kg	J	0.01	0.169
HB-T-4-2	1/29/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.21	0.105	ng/kg	UJ	0.01	0.001
HB-T-4-2	1/29/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.713	0.713	ng/kg	J	0.1	0.071
HB-T-4-2	1/29/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.012	2.012	ng/kg	J	0.1	0.201
HB-T-4-2	1/29/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	4.659	4.659	ng/kg	J	0.1	0.466
HB-T-4-2	1/29/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.139	1.139	ng/kg	J	0.1	0.114
HB-T-4-2	1/29/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.319	0.1595	ng/kg	UJ	0.1	0.016
HB-T-4-2	1/29/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.893	0.893	ng/kg	J	1	0.893
HB-T-4-2	1/29/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.123	1.123	ng/kg	J	0.03	0.034
HB-T-4-2	1/29/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.232	0.116	ng/kg	U	1	0.116
HB-T-4-2	1/29/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.23	2.23	ng/kg		0.1	0.223
HB-T-4-2	1/29/2001	0	0.5	3268-87-9	OCDD	Y	514.462	514.462	ng/kg		0.0003	0.154
HB-T-4-2	1/29/2001	0	0.5	39001-02-0	OCDF	Y	52.439	52.439	ng/kg		0.0003	0.016
Sample Location TEQ = 2.5												

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-4-2	1/29/2001	0	1.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	40.656	40.656	ng/kg	J	0.01	0.407
HB-T-4-2	1/29/2001	0	1.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.943	1.943	ng/kg	J	0.01	0.019
HB-T-4-2	1/29/2001	0	1.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.762	1.762	ng/kg	J	0.1	0.176
HB-T-4-2	1/29/2001	0	1.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	8.026	8.026	ng/kg	J	0.1	0.803
HB-T-4-2	1/29/2001	0	1.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.051	8.051	ng/kg	J	0.1	0.805
HB-T-4-2	1/29/2001	0	1.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.778	3.778	ng/kg	J	0.1	0.378
HB-T-4-2	1/29/2001	0	1.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.045	2.045	ng/kg	J	0.1	0.205
HB-T-4-2	1/29/2001	0	1.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.596	1.596	ng/kg	J	0.1	0.160
HB-T-4-2	1/29/2001	0	1.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.967	1.967	ng/kg	J	1	1.967
HB-T-4-2	1/29/2001	0	1.5	1746-01-6	2,3,7,8-TCDD	Y	0.495	0.495	ng/kg	J	1	0.495
HB-T-4-2	1/29/2001	0	1.5	51207-31-9	2,3,7,8-TCDF	Y	5.8	5.8	ng/kg		0.1	0.580
HB-T-4-2	1/29/2001	0	1.5	3268-87-9	OCDD	Y	693.792	693.792	ng/kg	J	0.0003	0.208
HB-T-4-2	1/29/2001	0	1.5	39001-02-0	OCDF	Y	77.33	77.33	ng/kg	J	0.0003	0.023
Sample Location TEQ = 6.2												
HB-T-4-2	1/29/2001	1.5	3	40321-76-4	1,2,3,7,8-PECDD	N	1.14	0.57	ng/kg	U	1	0.570
HB-T-4-2	1/29/2001	1.5	3	57117-41-6	1,2,3,7,8-PECDF	Y	3.391	3.391	ng/kg		0.03	0.102
HB-T-4-2	1/29/2001	1.5	3	1746-01-6	2,3,7,8-TCDD	N	2.032	1.016	ng/kg	U	1	1.016
HB-T-4-2	1/29/2001	1.5	3	51207-31-9	2,3,7,8-TCDF	Y	6.3	6.3	ng/kg	J	0.1	0.630
HB-T-4-2	1/29/2001	1.5	3	3268-87-9	OCDD	Y	95.605	95.605	ng/kg	J	0.0003	0.029
Sample Location TEQ = 2.3												
HB-T-4-2	1/29/2001	3	5.2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	1.992	1.992	ng/kg	J	0.01	0.020
HB-T-4-2	1/29/2001	3	5.2	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.286	0.143	ng/kg	U	0.01	0.001
HB-T-4-2	1/29/2001	3	5.2	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.189	0.0945	ng/kg	U	0.1	0.009
HB-T-4-2	1/29/2001	3	5.2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.029	1.029	ng/kg	J	0.1	0.103
HB-T-4-2	1/29/2001	3	5.2	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.216	0.108	ng/kg	U	0.1	0.011
HB-T-4-2	1/29/2001	3	5.2	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.144	0.072	ng/kg	U	0.1	0.007
HB-T-4-2	1/29/2001	3	5.2	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.199	0.0995	ng/kg	UJ	0.1	0.010
HB-T-4-2	1/29/2001	3	5.2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.163	0.0815	ng/kg	U	0.1	0.008
HB-T-4-2	1/29/2001	3	5.2	40321-76-4	1,2,3,7,8-PECDD	N	0.262	0.131	ng/kg	U	1	0.131
HB-T-4-2	1/29/2001	3	5.2	1746-01-6	2,3,7,8-TCDD	N	0.293	0.1465	ng/kg	U	1	0.147
HB-T-4-2	1/29/2001	3	5.2	51207-31-9	2,3,7,8-TCDF	Y	0.427	0.427	ng/kg	J	0.1	0.043
Sample Location TEQ = 0.5												
HB-T-4-2	1/29/2001	5.2	7.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.336	0.168	ng/kg	U	0.01	0.002
HB-T-4-2	1/29/2001	5.2	7.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.207	0.1035	ng/kg	UJ	0.1	0.010
HB-T-4-2	1/29/2001	5.2	7.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.241	0.1205	ng/kg	UJ	0.1	0.012
HB-T-4-2	1/29/2001	5.2	7.5	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.237	0.1185	ng/kg	UJ	0.1	0.012
HB-T-4-2	1/29/2001	5.2	7.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.282	0.141	ng/kg	UJ	0.1	0.014
HB-T-4-2	1/29/2001	5.2	7.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.218	0.109	ng/kg	UJ	0.1	0.011
HB-T-4-2	1/29/2001	5.2	7.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.318	0.159	ng/kg	UJ	0.1	0.016
HB-T-4-2	1/29/2001	5.2	7.5	40321-76-4	1,2,3,7,8-PECDD	N	0.264	0.132	ng/kg	U	1	0.132
HB-T-4-2	1/29/2001	5.2	7.5	57117-41-6	1,2,3,7,8-PECDF	N	0.131	0.0655	ng/kg	U	0.03	0.002
HB-T-4-2	1/29/2001	5.2	7.5	1746-01-6	2,3,7,8-TCDD	N	0.26	0.13	ng/kg	U	1	0.130
HB-T-4-2	1/29/2001	5.2	7.5	51207-31-9	2,3,7,8-TCDF	N	0.166	0.083	ng/kg	U	0.1	0.008
HB-T-4-2	1/29/2001	5.2	7.5	39001-02-0	OCDF	N	0.359	0.1795	ng/kg	UJ	0.0003	0.0001
Sample Location TEQ = 0.3												

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-5-1	1/30/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	59.982	59.982	ng/kg	J	0.01	0.600
HB-T-5-1	1/30/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.433	0.2165	ng/kg	UJ	0.01	0.002
HB-T-5-1	1/30/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.282	1.282	ng/kg	J	0.1	0.128
HB-T-5-1	1/30/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	56.608	56.608	ng/kg	J	0.1	5.661
HB-T-5-1	1/30/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.597	8.597	ng/kg	J	0.1	0.860
HB-T-5-1	1/30/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	17.493	17.493	ng/kg	J	0.1	1.749
HB-T-5-1	1/30/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.418	2.418	ng/kg	J	0.1	0.242
HB-T-5-1	1/30/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.519	0.2595	ng/kg	UJ	0.1	0.026
HB-T-5-1	1/30/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	2.904	2.904	ng/kg		1	2.904
HB-T-5-1	1/30/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	16.127	16.127	ng/kg		0.03	0.484
HB-T-5-1	1/30/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.331	0.1655	ng/kg	U	1	0.166
HB-T-5-1	1/30/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	24.1	24.1	ng/kg		0.1	2.410
HB-T-5-1	1/30/2001	0	0.5	3268-87-9	OCDD	Y	705.376	705.376	ng/kg	J	0.0003	0.212
HB-T-5-1	1/30/2001	0	0.5	39001-02-0	OCDF	Y	158.594	158.594	ng/kg	J	0.0003	0.048
Sample Location TEQ =												15.5
HB-T-5-1	1/30/2001	0.5	1.5	40321-76-4	1,2,3,7,8-PECDD	N	0.992	0.496	ng/kg	U	1	0.496
HB-T-5-1	1/30/2001	0.5	1.5	1746-01-6	2,3,7,8-TCDD	N	1.431	0.7155	ng/kg	U	1	0.716
HB-T-5-1	1/30/2001	0.5	1.5	51207-31-9	2,3,7,8-TCDF	Y	5.11	5.11	ng/kg		0.1	0.511
Sample Location TEQ =												1.7
HB-T-5-1	1/30/2001	1.5	4.4	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	9.498	9.498	ng/kg		0.01	0.095
HB-T-5-1	1/30/2001	1.5	4.4	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.473	0.473	ng/kg	J	0.1	0.047
HB-T-5-1	1/30/2001	1.5	4.4	70648-26-9	1,2,3,4,7,8-HXCDF	Y	6.384	6.384	ng/kg	J	0.1	0.638
HB-T-5-1	1/30/2001	1.5	4.4	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.856	1.856	ng/kg	J	0.1	0.186
HB-T-5-1	1/30/2001	1.5	4.4	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.962	2.962	ng/kg	J	0.1	0.296
HB-T-5-1	1/30/2001	1.5	4.4	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.033	1.033	ng/kg	J	0.1	0.103
HB-T-5-1	1/30/2001	1.5	4.4	40321-76-4	1,2,3,7,8-PECDD	Y	0.736	0.736	ng/kg	J	1	0.736
HB-T-5-1	1/30/2001	1.5	4.4	57117-41-6	1,2,3,7,8-PECDF	Y	3.593	3.593	ng/kg		0.03	0.108
HB-T-5-1	1/30/2001	1.5	4.4	1746-01-6	2,3,7,8-TCDD	N	0.148	0.074	ng/kg	U	1	0.074
HB-T-5-1	1/30/2001	1.5	4.4	51207-31-9	2,3,7,8-TCDF	Y	7.49	7.49	ng/kg		0.1	0.749
HB-T-5-1	1/30/2001	1.5	4.4	3268-87-9	OCDD	Y	12.558	12.558	ng/kg		0.0003	0.004
HB-T-5-1	1/30/2001	1.5	4.4	39001-02-0	OCDF	Y	3.663	3.663	ng/kg		0.0003	0.001
Sample Location TEQ =												3.0

TABLE 2.19b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-T-5-1	1/30/2001	4.4	6.6	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.282	0.141	ng/kg	U	0.01	0.001
HB-T-5-1	1/30/2001	4.4	6.6	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.123	0.0615	ng/kg	U	0.1	0.006
HB-T-5-1	1/30/2001	4.4	6.6	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.133	0.0665	ng/kg	U	0.1	0.007
HB-T-5-1	1/30/2001	4.4	6.6	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.141	0.0705	ng/kg	U	0.1	0.007
HB-T-5-1	1/30/2001	4.4	6.6	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.155	0.0775	ng/kg	U	0.1	0.008
HB-T-5-1	1/30/2001	4.4	6.6	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.129	0.0645	ng/kg	U	0.1	0.006
HB-T-5-1	1/30/2001	4.4	6.6	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.176	0.088	ng/kg	U	0.1	0.009
HB-T-5-1	1/30/2001	4.4	6.6	40321-76-4	1,2,3,7,8-PECDD	N	0.149	0.0745	ng/kg	U	1	0.075
HB-T-5-1	1/30/2001	4.4	6.6	57117-41-6	1,2,3,7,8-PECDF	N	0.111	0.0555	ng/kg	U	0.03	0.002
HB-T-5-1	1/30/2001	4.4	6.6	1746-01-6	2,3,7,8-TCDD	N	0.148	0.074	ng/kg	U	1	0.074
HB-T-5-1	1/30/2001	4.4	6.6	51207-31-9	2,3,7,8-TCDF	N	0.12	0.06	ng/kg	U	0.1	0.006
HB-T-5-1	1/30/2001	4.4	6.6	39001-02-0	OCDF	N	0.344	0.172	ng/kg	U	0.0003	0.0001
Sample Location TEQ =											0.2	

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

EMPC = Estimated Maximum Possible Concentration

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223-241.

TABLE 2.19c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-H2	0	1.3	11/7/1996	0.23	mg/kg
Highly Chlorinated PCBs	HB-H3	0	1	11/7/1996	0.55	mg/kg
Highly Chlorinated PCBs	HB-H3	1	1.8	11/7/1996	0.2	mg/kg
Highly Chlorinated PCBs	HB-H4	0	1	11/7/1996	0.79	mg/kg
Highly Chlorinated PCBs	HB-H5	0	1	11/7/1996	1	mg/kg
Highly Chlorinated PCBs	HB-HBSED-16	0	0.5	6/2/2003	0.37	mg/kg
Highly Chlorinated PCBs	HB-HBSED-19	0	0.5	6/3/2003	0.21	mg/kg
Highly Chlorinated PCBs	HB-HBSED-19	0.5	1	6/3/2003	0.13	mg/kg
Highly Chlorinated PCBs	HB-T-1-1	0	0.5	1/24/2001	0.32	mg/kg
Highly Chlorinated PCBs	HB-T-1-2	0.5	1.5	1/24/2001	0.89	mg/kg
Highly Chlorinated PCBs	HB-T-1-2	1.5	2.5	1/24/2001	2	mg/kg
Highly Chlorinated PCBs	HB-T-1-2	3.4	4.4	1/24/2001	0.17	mg/kg
Highly Chlorinated PCBs	HB-T-2-1	0	0.5	1/25/2001	0.27	mg/kg
Highly Chlorinated PCBs	HB-T-2-2	0	0.5	1/25/2001	0.11	mg/kg
Highly Chlorinated PCBs	HB-T-2-3	0	0.5	1/25/2001	3.7	mg/kg
Highly Chlorinated PCBs	HB-T-2-3	0.5	1.5	1/25/2001	1.1	mg/kg
Highly Chlorinated PCBs	HB-T-2-3	1.5	2.5	1/25/2001	0.092	mg/kg
Highly Chlorinated PCBs	HB-T-3-1	0	0.5	1/26/2001	0.16	mg/kg
Highly Chlorinated PCBs	HB-T-3-2	0	0.5	1/26/2001	1.29	mg/kg
Highly Chlorinated PCBs	HB-T-3-3	0	0.5	1/26/2001	0.16	mg/kg
Highly Chlorinated PCBs	HB-T-4-1	0	0.5	1/29/2001	0.19	mg/kg
Highly Chlorinated PCBs	HB-T-4-2	0	0.5	1/29/2001	0.067	mg/kg
Highly Chlorinated PCBs	HB-T-4-2	0	1.5	1/29/2001	0.3	mg/kg
Highly Chlorinated PCBs	HB-T-4-2	1.5	3	1/29/2001	1.1	mg/kg
Highly Chlorinated PCBs	HB-T-4-3	0	0.5	1/29/2001	0.091	mg/kg
Highly Chlorinated PCBs	HB-T-5-1	0	0.5	1/30/2001	0.37	mg/kg
Highly Chlorinated PCBs	HB-T-5-2	0	0.5	1/31/2001	0.13	mg/kg
Less Chlorinated PCBs	HB-T-4-2	0	0.5	1/29/2001	0.12	mg/kg
Total PCBs	HB-H2	0	1.3	11/7/1996	0.23	mg/kg
Total PCBs	HB-H3	0	1	11/7/1996	0.55	mg/kg
Total PCBs	HB-H3	1	1.8	11/7/1996	0.2	mg/kg
Total PCBs	HB-H4	0	1	11/7/1996	0.79	mg/kg
Total PCBs	HB-H5	0	1	11/7/1996	1	mg/kg
Total PCBs	HB-HBSED-16	0	0.5	6/2/2003	0.37	mg/kg
Total PCBs	HB-HBSED-19	0	0.5	6/3/2003	0.21	mg/kg
Total PCBs	HB-HBSED-19	0.5	1	6/3/2003	0.13	mg/kg
Total PCBs	HB-T-1-1	0	0.5	1/24/2001	0.32	mg/kg
Total PCBs	HB-T-1-2	0.5	1.5	1/24/2001	0.89	mg/kg
Total PCBs	HB-T-1-2	1.5	2.5	1/24/2001	2	mg/kg
Total PCBs	HB-T-1-2	3.4	4.4	1/24/2001	0.17	mg/kg
Total PCBs	HB-T-2-1	0	0.5	1/25/2001	0.27	mg/kg

TABLE 2.19c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-T-2-2	0	0.5	1/25/2001	0.11	mg/kg
Total PCBs	HB-T-2-3	0	0.5	1/25/2001	3.7	mg/kg
Total PCBs	HB-T-2-3	0.5	1.5	1/25/2001	1.1	mg/kg
Total PCBs	HB-T-2-3	1.5	2.5	1/25/2001	0.092	mg/kg
Total PCBs	HB-T-3-1	0	0.5	1/26/2001	0.16	mg/kg
Total PCBs	HB-T-3-2	0	0.5	1/26/2001	1.29	mg/kg
Total PCBs	HB-T-3-3	0	0.5	1/26/2001	0.16	mg/kg
Total PCBs	HB-T-4-1	0	0.5	1/29/2001	0.19	mg/kg
Total PCBs	HB-T-4-2	0	0.5	1/29/2001	0.187	mg/kg
Total PCBs	HB-T-4-2	0	1.5	1/29/2001	0.3	mg/kg
Total PCBs	HB-T-4-2	1.5	3	1/29/2001	1.1	mg/kg
Total PCBs	HB-T-4-3	0	0.5	1/29/2001	0.091	mg/kg
Total PCBs	HB-T-5-1	0	0.5	1/30/2001	0.37	mg/kg
Total PCBs	HB-T-5-2	0	0.5	1/31/2001	0.13	mg/kg

Notes:

* Less Chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.19d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-CSXSED-1	11/14/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.022
HB-CSXSED-1	11/14/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.022
Total Chlordane =									ND
HB-CSXSED-2	11/14/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-CSXSED-2	11/14/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-H3	11/7/1996	0	1	57-74-9	CHLORDANE	N	U	mg/kg	0.04
HB-H3	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.021
Total Chlordane =									0.021
HB-H4	11/7/1996	0	1	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-H4	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0091
Total Chlordane =									0.0091
HB-H5	11/7/1996	0	1	57-74-9	CHLORDANE	Y	J	mg/kg	0.048
HB-H5	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.026
Total Chlordane =									0.074
HB-H6	11/7/1996	0	0.8	57-74-9	CHLORDANE	N	U	mg/kg	0.053
HB-H6	11/7/1996	0	0.8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.028
Total Chlordane =									0.028
HB-H7	11/7/1996	0	1	57-74-9	CHLORDANE	N	U	mg/kg	0.045
HB-H7	11/7/1996	0	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0053
Total Chlordane =									0.0053
HB-HBSED-14	11/14/2002	0	0.33	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-HBSED-14	11/14/2002	0	0.33	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-HBSED-15	11/14/2002	0	0.25	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-HBSED-15	11/14/2002	0	0.25	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-HBSED-16	6/2/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.015
HB-HBSED-16	6/2/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.015
Total Chlordane =									ND
HB-HBSED-19	6/3/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.014
HB-HBSED-19	6/3/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.014
Total Chlordane =									ND
HB-HBSED-19	6/3/2003	0.5	1	57-74-9	CHLORDANE	Y		mg/kg	0.022
HB-HBSED-19	6/3/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.022
Total Chlordane =									0.022
HB-HBSED-20	6/3/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.013
HB-HBSED-20	6/3/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.013
Total Chlordane =									ND
HB-S-1	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.025
HB-S-1	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.025
Total Chlordane =									ND

TABLE 2.19d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-S-2	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.043
HB-S-2	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.043
Total Chlordane =									ND
HB-T-1-1	1/24/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.041
HB-T-1-1	1/24/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.041
Total Chlordane =									ND
HB-T-1-2	1/24/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.041
HB-T-1-2	1/24/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.041
Total Chlordane =									ND
HB-T-1-2	1/24/2001	0.5	1.5	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-1-2	1/24/2001	0.5	1.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-1-2	1/24/2001	1.5	2.5	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-T-1-2	1/24/2001	1.5	2.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-T-1-2	1/24/2001	2.5	3.4	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-T-1-2	1/24/2001	2.5	3.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-T-1-2	1/24/2001	3.4	4.4	57-74-9	CHLORDANE	N	U	mg/kg	0.032
HB-T-1-2	1/24/2001	3.4	4.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.032
Total Chlordane =									ND
HB-T-1-2	1/24/2001	4.4	5.4	57-74-9	CHLORDANE	N	U	mg/kg	0.036
HB-T-1-2	1/24/2001	4.4	5.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.036
Total Chlordane =									ND
HB-T-1-2	1/24/2001	5.4	6.4	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-1-2	1/24/2001	5.4	6.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-1-2	1/24/2001	6.4	7.4	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-1-2	1/24/2001	6.4	7.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-1-2	1/24/2001	7.4	8.1	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-1-2	1/24/2001	7.4	8.1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-1-3	1/24/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-1-3	1/24/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-2-1	1/25/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-T-2-1	1/25/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-T-2-2	1/25/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-2-2	1/25/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND

TABLE 2.19d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-T-2-3	1/25/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-T-2-3	1/25/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-T-2-3	1/25/2001	0.5	1.5	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-T-2-3	1/25/2001	0.5	1.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-T-2-3	1/25/2001	1.5	2.5	57-74-9	CHLORDANE	N	U	mg/kg	0.036
HB-T-2-3	1/25/2001	1.5	2.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.036
Total Chlordane =									ND
HB-T-2-3	1/25/2001	2.5	3.4	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-2-3	1/25/2001	2.5	3.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-2-3	1/25/2001	3.4	4.4	57-74-9	CHLORDANE	N	U	mg/kg	0.023
HB-T-2-3	1/25/2001	3.4	4.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.023
Total Chlordane =									ND
HB-T-2-3	1/25/2001	4.4	5.4	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-T-2-3	1/25/2001	4.4	5.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-T-2-3	1/25/2001	5.4	6.4	57-74-9	CHLORDANE	N	U	mg/kg	0.0025
HB-T-2-3	1/25/2001	5.4	6.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0025
Total Chlordane =									ND
HB-T-2-3	1/25/2001	6.4	7.4	57-74-9	CHLORDANE	N	U	mg/kg	0.0029
HB-T-2-3	1/25/2001	6.4	7.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0029
Total Chlordane =									ND
HB-T-2-3	1/25/2001	7.4	8.4	57-74-9	CHLORDANE	N	U	mg/kg	0.003
HB-T-2-3	1/25/2001	7.4	8.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.003
Total Chlordane =									ND
HB-T-2-3	1/25/2001	8.4	9.4	57-74-9	CHLORDANE	N	U	mg/kg	0.0031
HB-T-2-3	1/25/2001	8.4	9.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0031
Total Chlordane =									ND
HB-T-2-3	1/25/2001	0.5	1.5	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-T-2-3	1/25/2001	0.5	1.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-T-3-1	1/26/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.025
HB-T-3-1	1/26/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.025
Total Chlordane =									ND
HB-T-3-2	1/26/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-T-3-2	1/26/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-T-3-2	1/26/2001	0.5	1.5	57-74-9	CHLORDANE	N	U	mg/kg	0.021
HB-T-3-2	1/26/2001	0.5	1.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.021
Total Chlordane =									ND

TABLE 2.19d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-T-3-2	1/26/2001	1.5	2.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-3-2	1/26/2001	1.5	2.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-3-2	1/26/2001	2.5	3.4	57-74-9	CHLORDANE	N	U	mg/kg	0.031
HB-T-3-2	1/26/2001	2.5	3.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.031
Total Chlordane =									ND
HB-T-3-2	1/26/2001	3.4	4.4	57-74-9	CHLORDANE	N	U	mg/kg	0.031
HB-T-3-2	1/26/2001	3.4	4.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.031
Total Chlordane =									ND
HB-T-3-2	1/26/2001	4.4	5.4	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-3-2	1/26/2001	4.4	5.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-3-2	1/26/2001	5.4	6.4	57-74-9	CHLORDANE	N	U	mg/kg	0.029
HB-T-3-2	1/26/2001	5.4	6.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-T-3-2	1/26/2001	6.4	7.4	57-74-9	CHLORDANE	N	U	mg/kg	0.029
HB-T-3-2	1/26/2001	6.4	7.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-T-3-2	1/26/2001	7.4	8.4	57-74-9	CHLORDANE	N	U	mg/kg	0.0029
HB-T-3-2	1/26/2001	7.4	8.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0029
Total Chlordane =									ND
HB-T-3-2	1/26/2001	8.4	9.2	57-74-9	CHLORDANE	N	U	mg/kg	0.0029
HB-T-3-2	1/26/2001	8.4	9.2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0029
Total Chlordane =									ND
HB-T-3-3	1/26/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.035
HB-T-3-3	1/26/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.035
Total Chlordane =									ND
HB-T-3-OIL	2/13/2001	4.1	6.86	57-74-9	CHLORDANE	N	UJ	mg/kg	5
HB-T-3-OIL	2/13/2001	4.1	6.86	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	5
Total Chlordane =									ND
HB-T-4-1	1/29/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-4-1	1/29/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-4-2	1/29/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-4-2	1/29/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-4-2	1/29/2001	0	1.5	57-74-9	CHLORDANE	N	U	mg/kg	0.024
HB-T-4-2	1/29/2001	0	1.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.024
Total Chlordane =									ND
HB-T-4-2	1/29/2001	1.5	3	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-T-4-2	1/29/2001	1.5	3	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND

TABLE 2.19d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-T-4-2	1/29/2001	3	5.2	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-4-2	1/29/2001	3	5.2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-4-2	1/29/2001	5.2	7.5	57-74-9	CHLORDANE	N	U	mg/kg	0.029
HB-T-4-2	1/29/2001	5.2	7.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-T-4-3	1/29/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-T-4-3	1/29/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-T-5-1	1/30/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.028
HB-T-5-1	1/30/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-T-5-1	1/30/2001	0.5	1.5	57-74-9	CHLORDANE	N	U	mg/kg	0.027
HB-T-5-1	1/30/2001	0.5	1.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.027
Total Chlordane =									ND
HB-T-5-1	1/30/2001	1.5	1.5	57-74-9	CHLORDANE	N	U	mg/kg	0.024
HB-T-5-1	1/30/2001	1.5	1.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.024
Total Chlordane =									ND
HB-T-5-1	1/30/2001	2.5	3.4	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-5-1	1/30/2001	2.5	3.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-5-1	1/30/2001	3.4	4.4	57-74-9	CHLORDANE	N	U	mg/kg	0.026
HB-T-5-1	1/30/2001	3.4	4.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.026
Total Chlordane =									ND
HB-T-5-1	1/30/2001	4.4	5.4	57-74-9	CHLORDANE	N	U	mg/kg	0.0028
HB-T-5-1	1/30/2001	4.4	5.4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0028
Total Chlordane =									ND
HB-T-5-1	1/30/2001	5.4	6.6	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-T-5-1	1/30/2001	5.4	6.6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-T-5-2	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.021
HB-T-5-2	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.021
Total Chlordane =									ND
HB-T-5-3	1/31/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.041
HB-T-5-3	1/31/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.041
Total Chlordane =									ND

TABLE 2.19e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-CSXSED-1	11/14/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.41	
HB-CSXSED-1	11/14/2002	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.28	
HB-CSXSED-1	11/14/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.69
HB-CSXSED-2	11/14/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	1.5	
HB-CSXSED-2	11/14/2002	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	1	
HB-CSXSED-2	11/14/2002	0	0.5	CALCULATED	TOTAL	Y		mg/kg		2.5
HB-H2	11/7/1996	0	1.3	1330-20-7	XYLENES, TOTAL	Y		mg/kg	14	14
HB-H3	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	1.2	1.2
HB-H3	11/7/1996	1	1.8	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	1	1
HB-H4	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.044	0.044
HB-H4	11/7/1996	1	2.2	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.02	0.02
HB-H5	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.007	0.007
HB-H5	11/7/1996	1	1.6	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.071	0.071
HB-H6	11/7/1996	0	0.8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	81	81
HB-H7	11/7/1996	0	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.89	0.89
HB-HBSED-14	11/14/2002	0	0.33	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0035	
HB-HBSED-14	11/14/2002	0	0.33	95-47-6	O-XYLENE	Y	J	mg/kg	0.0023	
HB-HBSED-14	11/14/2002	0	0.33	CALCULATED	TOTAL	Y	J	mg/kg		0.0058
HB-HBSED-15	11/14/2002	0	0.25	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.75	
HB-HBSED-15	11/14/2002	0	0.25	95-47-6	O-XYLENE	Y	J	mg/kg	0.38	
HB-HBSED-15	11/14/2002	0	0.25	CALCULATED	TOTAL	Y	J	mg/kg		1.13
HB-HBSED-16	6/2/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.19	
HB-HBSED-16	6/2/2003	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	0.085	
HB-HBSED-16	6/2/2003	0	0.5	CALCULATED	TOTAL	Y		mg/kg		0.275
HB-HBSED-19	6/3/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.35	
HB-HBSED-19	6/3/2003	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	1.6	
HB-HBSED-19	6/3/2003	0	0.5	CALCULATED	TOTAL	Y		mg/kg		1.95
HB-HBSED-19	6/3/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.2	
HB-HBSED-19	6/3/2003	0.5	1	95-47-6	O-XYLENE	Y		mg/kg	1.4	
HB-HBSED-19	6/3/2003	0.5	1	CALCULATED	TOTAL	Y		mg/kg		1.6
HB-HBSED-20	6/3/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.013	
HB-HBSED-20	6/3/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.0048	
HB-HBSED-20	6/3/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.0178
HB-S-1	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	12	
HB-S-1	1/31/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	4.1	
HB-S-1	1/31/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		16.1
HB-S-2	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0047	
HB-S-2	1/31/2001	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.016	
HB-S-2	1/31/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.0047
HB-T-1-1	1/24/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.033	
HB-T-1-1	1/24/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	0.06	
HB-T-1-1	1/24/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		0.093
HB-T-1-2	1/24/2001	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	4.8	
HB-T-1-2	1/24/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	4.8	
HB-T-1-2	1/24/2001	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		4.8
HB-T-1-2	1/24/2001	0.5	1.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	14	
HB-T-1-2	1/24/2001	0.5	1.5	95-47-6	O-XYLENE	Y		mg/kg	7.8	
HB-T-1-2	1/24/2001	0.5	1.5	CALCULATED	TOTAL	Y		mg/kg		21.8
HB-T-1-2	1/24/2001	1.5	2.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	5	
HB-T-1-2	1/24/2001	1.5	2.5	95-47-6	O-XYLENE	Y		mg/kg	4.8	
HB-T-1-2	1/24/2001	1.5	2.5	CALCULATED	TOTAL	Y		mg/kg		9.8
HB-T-1-2	1/24/2001	2.5	3.4	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.57	
HB-T-1-2	1/24/2001	2.5	3.4	95-47-6	O-XYLENE	Y	J	mg/kg	0.68	
HB-T-1-2	1/24/2001	2.5	3.4	CALCULATED	TOTAL	Y		mg/kg		1.25
HB-T-1-2	1/24/2001	3.4	4.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	1.8	
HB-T-1-2	1/24/2001	3.4	4.4	95-47-6	O-XYLENE	Y	J	mg/kg	0.91	

TABLE 2.19e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-T-1-2	1/24/2001	3.4	4.4	CALCULATED	TOTAL	Y		mg/kg		2.71
HB-T-1-2	1/24/2001	4.4	5.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.17	
HB-T-1-2	1/24/2001	4.4	5.4	95-47-6	O-XYLENE	Y		mg/kg	0.079	
HB-T-1-2	1/24/2001	4.4	5.4	CALCULATED	TOTAL	Y		mg/kg		0.249
HB-T-1-2	1/24/2001	5.4	6.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.048	
HB-T-1-2	1/24/2001	5.4	6.4	95-47-6	O-XYLENE	Y		mg/kg	0.027	
HB-T-1-2	1/24/2001	5.4	6.4	CALCULATED	TOTAL	Y		mg/kg		0.075
HB-T-1-2	1/24/2001	6.4	7.4	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0083	
HB-T-1-2	1/24/2001	6.4	7.4	95-47-6	O-XYLENE	Y	J	mg/kg	0.0085	
HB-T-1-2	1/24/2001	6.4	7.4	CALCULATED	TOTAL	Y		mg/kg		0.0168
HB-T-1-2	1/24/2001	7.4	8.1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0018	
HB-T-1-2	1/24/2001	7.4	8.1	95-47-6	O-XYLENE	Y	J	mg/kg	0.0022	
HB-T-1-2	1/24/2001	7.4	8.1	CALCULATED	TOTAL	Y		mg/kg		0.004
HB-T-1-3	1/24/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.029	
HB-T-1-3	1/24/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.045	
HB-T-1-3	1/24/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.029
HB-T-2-1	1/25/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.062	
HB-T-2-1	1/25/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	0.033	
HB-T-2-1	1/25/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		0.095
HB-T-2-2	1/25/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.3	
HB-T-2-2	1/25/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	1	
HB-T-2-2	1/25/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.3
HB-T-2-3	1/25/2001	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	1.1	
HB-T-2-3	1/25/2001	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	1.1	
HB-T-2-3	1/25/2001	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		1.1
HB-T-2-3	1/25/2001	0.5	1.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	1	
HB-T-2-3	1/25/2001	0.5	1.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.33	
HB-T-2-3	1/25/2001	0.5	1.5	CALCULATED	TOTAL		J	mg/kg		0.33
HB-T-2-3	1/25/2001	1.5	2.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.13	
HB-T-2-3	1/25/2001	1.5	2.5	95-47-6	O-XYLENE	Y		mg/kg	0.12	
HB-T-2-3	1/25/2001	1.5	2.5	CALCULATED	TOTAL	Y	0	mg/kg		0.25
HB-T-2-3	1/25/2001	2.5	3.4	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.035	
HB-T-2-3	1/25/2001	2.5	3.4	95-47-6	O-XYLENE	Y	J	mg/kg	0.044	
HB-T-2-3	1/25/2001	2.5	3.4	CALCULATED	TOTAL	Y	J	mg/kg		0.079
HB-T-2-3	1/25/2001	3.4	4.4	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0051	
HB-T-2-3	1/25/2001	3.4	4.4	95-47-6	O-XYLENE	Y		mg/kg	0.0091	
HB-T-2-3	1/25/2001	3.4	4.4	CALCULATED	TOTAL	Y		mg/kg		0.0142
HB-T-2-3	1/25/2001	4.4	5.4	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0089	
HB-T-2-3	1/25/2001	4.4	5.4	95-47-6	O-XYLENE	N	U	mg/kg	0.0089	
HB-T-2-3	1/25/2001			CALCULATED	TOTAL	N	U	mg/kg		0.0089
HB-T-2-3	1/25/2001	5.4	6.4	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0083	
HB-T-2-3	1/25/2001	5.4	6.4	95-47-6	O-XYLENE	N	U	mg/kg	0.0083	
HB-T-2-3	1/25/2001			CALCULATED	TOTAL	N	U	mg/kg		0.0083
HB-T-2-3	1/25/2001	6.4	7.4	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0089	
HB-T-2-3	1/25/2001	6.4	7.4	95-47-6	O-XYLENE	N	U	mg/kg	0.0089	
HB-T-2-3	1/25/2001			CALCULATED	TOTAL	N	U	mg/kg		0.0089
HB-T-2-3	1/25/2001	7.4	8.4	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0096	
HB-T-2-3	1/25/2001	7.4	8.4	95-47-6	O-XYLENE	N	U	mg/kg	0.0096	
HB-T-2-3	1/25/2001			CALCULATED	TOTAL	N	U	mg/kg		0.0096
HB-T-2-3	1/25/2001	8.4	9.4	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.01	
HB-T-2-3	1/25/2001	8.4	9.4	95-47-6	O-XYLENE	N	U	mg/kg	0.01	
HB-T-2-3	1/25/2001			CALCULATED	TOTAL	N	U	mg/kg		0.01
HB-T-2-3	1/25/2001	9.4	9.8	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.022	
HB-T-2-3	1/25/2001	9.4	9.8	95-47-6	O-XYLENE	N	U	mg/kg	0.022	
HB-T-2-3	1/25/2001			CALCULATED	TOTAL	N	U	mg/kg		0.022
HB-T-3-1	1/26/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.74	
HB-T-3-1	1/26/2001	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.88	
HB-T-3-1	1/26/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		1.62

TABLE 2.19e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-T-3-2	1/26/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.53	
HB-T-3-2	1/26/2001	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.67	
HB-T-3-2	1/26/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		1.2
HB-T-3-2	1/26/2001	0.5	1.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	3.7	
HB-T-3-2	1/26/2001	0.5	1.5	95-47-6	O-XYLENE	Y		mg/kg	4	
HB-T-3-2	1/26/2001	0.5	1.5	CALCULATED	TOTAL	Y		mg/kg		7.7
HB-T-3-2	1/26/2001	1.5	2.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	14	
HB-T-3-2	1/26/2001	1.5	2.5	95-47-6	O-XYLENE	Y		mg/kg	4.7	
HB-T-3-2	1/26/2001	1.5	2.5	CALCULATED	TOTAL	Y		mg/kg		18.7
HB-T-3-2	1/26/2001	2.5	3.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	16	
HB-T-3-2	1/26/2001	2.5	3.4	95-47-6	O-XYLENE	Y		mg/kg	5.5	
HB-T-3-2	1/26/2001	2.5	3.4	CALCULATED	TOTAL	Y		mg/kg		21.5
HB-T-3-2	1/26/2001	3.4	4.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	22	
HB-T-3-2	1/26/2001	3.4	4.4	95-47-6	O-XYLENE	Y	J	mg/kg	7.5	
HB-T-3-2	1/26/2001	3.4	4.4	CALCULATED	TOTAL	Y		mg/kg		29.5
HB-T-3-2	1/26/2001	4.4	5.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	170	
HB-T-3-2	1/26/2001	4.4	5.4	95-47-6	O-XYLENE	Y		mg/kg	57	
HB-T-3-2	1/26/2001	4.4	5.4	CALCULATED	TOTAL	Y		mg/kg		227
HB-T-3-2	1/26/2001	5.4	6.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	200	
HB-T-3-2	1/26/2001	5.4	6.4	95-47-6	O-XYLENE	Y		mg/kg	70	
HB-T-3-2	1/26/2001	5.4	6.4	CALCULATED	TOTAL	Y		mg/kg		270
HB-T-3-2	1/26/2001	6.4	7.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	7.6	
HB-T-3-2	1/26/2001	6.4	7.4	95-47-6	O-XYLENE	Y		mg/kg	2.5	
HB-T-3-2	1/26/2001	6.4	7.4	CALCULATED	TOTAL	Y		mg/kg		10.1
HB-T-3-2	1/26/2001	7.4	8.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.67	
HB-T-3-2	1/26/2001	7.4	8.4	95-47-6	O-XYLENE	Y		mg/kg	0.26	
HB-T-3-2	1/26/2001	7.4	8.4	CALCULATED	TOTAL	Y		mg/kg		0.93
HB-T-3-2	1/26/2001	8.4	9.2	XYLENES1314	XYLENES, M & P	Y		mg/kg	1.3	
HB-T-3-2	1/26/2001	8.4	9.2	95-47-6	O-XYLENE	Y		mg/kg	0.46	
HB-T-3-2	1/26/2001	8.4	9.2	CALCULATED	TOTAL	Y		mg/kg		1.76
HB-T-3-3	1/26/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	38	
HB-T-3-3	1/26/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	12	
HB-T-3-3	1/26/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		50
HB-T-3-OIL	2/13/2001	0	0	XYLENES1314	XYLENES, M & P	Y		mg/kg	7700	
HB-T-3-OIL	2/13/2001	0	0	95-47-6	O-XYLENE	Y		mg/kg	2800	
HB-T-3-OIL	2/13/2001	0	0	CALCULATED	TOTAL	Y		mg/kg		10500
HB-T-4-1	1/29/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	7.5	
HB-T-4-1	1/29/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	6.3	
HB-T-4-1	1/29/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		13.8
HB-T-4-2	1/29/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	38	
HB-T-4-2	1/29/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	15	
HB-T-4-2	1/29/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		53
HB-T-4-2	1/29/2001	0	1.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	30	
HB-T-4-2	1/29/2001	0	1.5	95-47-6	O-XYLENE	Y		mg/kg	11	
HB-T-4-2	1/29/2001	0	1.5	CALCULATED	TOTAL	Y		mg/kg		41
HB-T-4-2	1/29/2001	1.5	3	XYLENES1314	XYLENES, M & P	Y		mg/kg	36	
HB-T-4-2	1/29/2001	1.5	3	95-47-6	O-XYLENE	Y		mg/kg	12	
HB-T-4-2	1/29/2001	1.5	3	CALCULATED	TOTAL	Y		mg/kg		48
HB-T-4-2	1/29/2001	3	5.2	XYLENES1314	XYLENES, M & P	Y		mg/kg	20	
HB-T-4-2	1/29/2001	3	5.2	95-47-6	O-XYLENE	Y	J	mg/kg	6.6	
HB-T-4-2	1/29/2001	3	5.2	CALCULATED	TOTAL	Y		mg/kg		26.6
HB-T-4-2	1/29/2001	5.2	7.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	34	
HB-T-4-2	1/29/2001	5.2	7.5	95-47-6	O-XYLENE	Y	J	mg/kg	12	
HB-T-4-2	1/29/2001	5.2	7.5	CALCULATED	TOTAL	Y		mg/kg		46
HB-T-4-3	1/29/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	13	
HB-T-4-3	1/29/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	5.7	
HB-T-4-3	1/29/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		18.7
HB-T-5-1	1/30/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.023	

TABLE 2.19e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SUBSURFACE SEDIMENT (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-T-5-1	1/30/2001	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.013	
HB-T-5-1	1/30/2001	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.036
HB-T-5-1	1/30/2001	0.5	1.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.088	
HB-T-5-1	1/30/2001	0.5	1.5	95-47-6	O-XYLENE	Y		mg/kg	0.061	
HB-T-5-1	1/30/2001	0.5	1.5	CALCULATED	TOTAL	Y		mg/kg		0.149
HB-T-5-1	1/30/2001	1.5	2.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.31	
HB-T-5-1	1/30/2001	1.5	2.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.33	
HB-T-5-1	1/30/2001	1.5	2.5	CALCULATED	TOTAL	Y		mg/kg		0.64
HB-T-5-1	1/30/2001	2.5	3.4	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.32	
HB-T-5-1	1/30/2001	2.5	3.4	95-47-6	O-XYLENE	Y	J	mg/kg	0.37	
HB-T-5-1	1/30/2001	2.5	3.4	CALCULATED	TOTAL	Y		mg/kg		0.69
HB-T-5-1	1/30/2001	3.4	4.4	XYLENES1314	XYLENES, M & P	Y		mg/kg	3.9	
HB-T-5-1	1/30/2001	3.4	4.4	95-47-6	O-XYLENE	Y		mg/kg	1.6	
HB-T-5-1	1/30/2001	3.4	4.4	CALCULATED	TOTAL	Y		mg/kg		5.5
HB-T-5-1	1/30/2001	4.4	5.4	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	3.9	
HB-T-5-1	1/30/2001	4.4	5.4	95-47-6	O-XYLENE	Y	J	mg/kg	1.4	
HB-T-5-1	1/30/2001	4.4	5.4	CALCULATED	TOTAL	Y		mg/kg		5.3
HB-T-5-1	1/30/2001	5.4	6.6	XYLENES1314	XYLENES, M & P	Y		mg/kg	6.7	
HB-T-5-1	1/30/2001	5.4	6.6	95-47-6	O-XYLENE	Y	J	mg/kg	2.2	
HB-T-5-1	1/30/2001	5.4	6.6	CALCULATED	TOTAL	Y		mg/kg		8.9
HB-T-5-2	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	4.3	
HB-T-5-2	1/31/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	1.6	
HB-T-5-2	1/31/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		5.9
HB-T-5-3	1/31/2001	0	0.5	XYLENES1314	XYLENES, M & P	Y		mg/kg	240	
HB-T-5-3	1/31/2001	0	0.5	95-47-6	O-XYLENE	Y		mg/kg	74	
HB-T-5-3	1/31/2001	0	0.5	CALCULATED	TOTAL	Y		mg/kg		314

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.20a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE WATER
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
Harbor Brook Surface Water	METALS																	
	7429-90-5	ALUMINUM	0.0153 J	1.69 J	mg/L	HB-HBSW-07	6/14	0.0118-0.1	1.69E+00		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	N	BSL
	7440-36-0	ANTIMONY	0.0015	0.0016 J	mg/L	HB-HBSW-06	2/14	0.0014-0.06	1.60E-03		6.00E-03	1.46E-03	N	1.46E-03	nc	1.46E-03	Y	ASL
	7440-38-2	ARSENIC	0.0018	0.0018	mg/L	HB-HBSW-06	1/14	0.0016-0.01	1.80E-03		1.00E-02	4.46E-05	C	4.48E-05	ca	4.46E-05	Y	TOX
	7440-39-3	BARIIUM	0.0225	0.129	mg/L	HB-HBSW-07	14/14	-	1.29E-01		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	N	BSL
	7440-41-7	BERYLLIUM	0.00014	0.00038 J	mg/L	HB-HBSW-06	2/14	0.000076-0.005	3.80E-04		4.00E-03	7.30E-03	N	7.30E-03	nc	7.30E-03	N	BSL
	7440-43-9	CADMIUM	0.00016	0.00016	mg/L	HB-HBSW-06	1/14	0.00024-0.005	1.60E-04		5.00E-03	1.83E-03	N	1.82E-03	nc	1.82E-03	N	BSL
	7440-70-2	CALCIUM	56.8	284	mg/L	HB-HBSW-08	14/14	-	2.84E+02			NV	NV	NV	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	0.0037	0.006 J	mg/L	HB-HBSW-08	5/14	0.01-0.01	6.00E-03		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX
	7440-48-4	COBALT	0.00058	0.00058	mg/L	HB-HBSW-06	1/14	0.00093-0.05	5.80E-04			NV	7.30E-02	nc	7.30E-02	N	BSL	
	7440-50-8	COPPER	0.0017 J	0.0026 J	mg/L	HB-HBSW-08	3/14	0.001-0.02	2.60E-03		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	N	BSL
	57-12-5	CYANIDE	0.01	0.01	mg/L	HB-HBSW-06	1/14	0.01-0.01	1.00E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL
	7439-89-6	IRON	0.0844 J	12.28 J	mg/L	HB-HBSW-07	13/14	0.1-0.1	1.23E+01		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL
	7439-92-1	LEAD	0.0016 J	0.013	mg/L	HB-HBSW-07	3/14	0.00066-0.005	1.30E-02		1.50E-02	NV	NV	NV	1.50E-02	N	BSL	
	7439-95-4	MAGNESIUM	2.74	50.5	mg/L	HB-HBSW-08	14/14	-	5.05E+01			NV	NV	NV	NV	N	NUT	
	7439-96-5	MANGANESE	0.0166	0.0589	mg/L	HB-HBSW-07	13/14	0.01-0.01	5.89E-02		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	N	BSL
	7439-97-6	MERCURY ^b	0.00018	0.00018	mg/L	HB-HBSW-06	1/14	0.00018-0.0002	1.80E-04		2.00E-03	3.65E-04	N	3.65E-04	nc	3.65E-04	N	BSL
	7440-02-0	NICKEL	0.00079 J	0.0019	mg/L	HB-HBSW-06	5/14	0.04-0.04	1.90E-03			7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL
	7440-09-7	POTASSIUM	3 J	12.5	mg/L	HB-SEEP-3	14/14	-	1.25E+01			NV	NV	NV	NV	N	NUT	
	7782-49-2	SELENIUM	0.0024	0.0029 J	mg/L	HB-HBSW-06	2/14	0.0018-0.025	2.90E-03		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL
	7440-22-4	SILVER	0.00034	0.00034	mg/L	HB-HBSW-06	1/14	0.00073-0.01	3.40E-04		1.00E-01	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL
	7440-23-5	SODIUM	124	205	mg/L	HB-SEEP-3	14/14	-	2.05E+02			NV	NV	NV	NV	N	NUT	
	7440-28-0	THALLIUM	0.0038	0.0038	mg/L	HB-HBSW-06	1/14	0.0036-0.01	3.80E-03		2.00E-03	2.56E-04	N	2.41E-04	nc	2.41E-04	Y	ASL
	7440-62-2	VANADIUM	0.0003	0.00052 J	mg/L	HB-HBSW-06	2/14	0.00039-0.05	5.20E-04			3.65E-03	N	3.65E-03	nc	3.65E-03	N	BSL
	7440-66-6	ZINC	0.0082 J	0.0336	mg/L	HB-HBSW-07	3/14	0.0026-0.02	3.36E-02		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	SVOCS																	
	91-57-6	2-METHYLNAPHTHALENE	1 J	98	ug/L	HB-HBSW-06	5/13	9.3-11	9.80E+01			2.43E+00	N	NV	nc	2.43E+00	Y	ASL
	95-48-7	2-METHYLPHENOL	2 J	2 J	ug/L	HB-HBSW-06	1/13	9.3-11	2.00E+00			1.83E+02	N	1.82E+02	nc	1.82E+02	N	BSL
	83-32-9	ACENAPHTHENE	25	27	ug/L	HB-HBSW-06	2/13	9.3-11	2.70E+01			3.65E+01	N	3.65E+01	nc	3.65E+01	N	BSL
	208-96-8	ACENAPHTHYLENE	1.1 J	1.1 J	ug/L	HB-HBSW-06	1/13	9.3-11	1.10E+00			NV	NV	NV	NV	Y	NTX	
	56-55-3	BENZ(A)ANTHRACENE	1 J	1 J	ug/L	HB-HBSW-07	1/14	9.3-11	1.00E+00			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	50-32-8	BENZO(A)PYRENE	1.2 J	1.2 J	ug/L	HB-HBSW-07	1/14	9.3-11	1.20E+00		2.00E-01	3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	1.2 J	1.2 J	ug/L	HB-HBSW-07	1/14	9.3-11	1.20E+00			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1.2 J	7.8 J	ug/L	HB-HBSW-06	2/13	9.3-11	7.80E+00		6.00E+00	4.78E+00	C	4.80E+00	ca	4.78E+00	Y	ASL
	86-74-8	CARBAZOLE	2.4 J	2.4 J	ug/L	HB-HBSW-06	1/13	9.3-11	2.40E+00			3.35E+00	C	3.36E+00	ca	3.35E+00	N	BSL
	218-01-9	CHRYSENE	1.3 J	1.4 J	ug/L	HB-HBSW-07	2/14	9.3-11	1.40E+00			3.00E+00	C	9.21E+00	ca	3.00E+00	N	BSL
	117-84-0	DI-N-OCTYL PHTHALATE	8.6 J	8.6 J	ug/L	HB-HBSW-06	1/13	9.3-11	8.60E+00			NV	1.46E+02	nc	1.46E+02	N	BSL	
	206-44-0	FLUORANTHENE	2.7 J	3.2 J	ug/L	HB-HBSW-08	3/14	9.3-11	3.20E+00			1.46E+02	N	1.46E+02	nc	1.46E+02	N	BSL
	86-73-7	FLUORENE	4.8 J	12	ug/L	HB-HBSW-08	2/13	9.3-11	1.20E+01			2.43E+01	N	2.43E+01	nc	2.43E+01	N	BSL
	91-20-3	NAPHTHALENE	1 J	2200	ug/L	HB-HBSW-06	10/18	1-11	2.20E+03			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	85-01-8	PHENANTHRENE	2.1 J	24	ug/L	HB-HBSW-08	3/13	9.3-11	2.40E+01			NV	NV	NV	NV	Y	NTX	
	108-95-2	PHENOL	1.5 J	1.5 J	ug/L	HB-HBSW-06	1/13	9.3-11	1.50E+00			1.10E+03	N	1.09E+03	nc	1.09E+03	N	BSL
	129-00-0	PYRENE	2.3 J	4.4 J	ug/L	HB-HBSW-08	3/14	9.3-11	4.40E+00			1.83E+01	N	1.83E+01	nc	1.83E+01	N	BSL
	VOCs																	
	67-64-1	ACETONE	3 J	3 J	ug/L	HB-HBSW-07, HB-HBSW-08, HB-HBSW-09	3/13	10-20	3.00E+00			5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	71-43-2	BENZENE	0.1 J	4	ug/L	HB-HBSW-06	5/13	5-5	4.00E+00		5.00E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	156-59-2	CIS-1,2-DICHLOROETHENE	0.2 J	0.3 J	ug/L	HB-HBSW-09	4/13	5-5	3.00E-01		7.00E+01	6.08E+00	N	6.08E+00	nc	6.08E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.4 J	0.8	ug/L	HB-HBSW-06	2/13	0.5-5	8.00E-01		7.00E+02	1.34E+02	N	1.34E+02	nc	1.34E+02	N	BSL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.9	1	ug/L	HB-HBSW-06	2/4	0.5-0.5	1.00E+00			1.46E+00	N	1.23E+00	nc	1.23E+00	N	BSL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.3 J	0.4 J	ug/L	HB-HBSW-06	2/4	0.5-0.5	4.00E-01			NV	1.23E+00	nc	1.23E+00	N	BSL	

TABLE 2.20a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE WATER
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)		USEPA PRG for Tap Water (6)		Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
	100-42-5	STYRENE	0.3 J	0.3 J	ug/L	HB-HBSW-06, HB-HBSW-07	2/13	0.5-5	3.00E-01		1.00E+02	1.62E+02	N	1.64E+02	nc	1.62E+02	N	BSL
	108-88-3	TOLUENE	0.3 J	4	ug/L	HB-HBSW-06	7/13	5-5	4.00E+00		1.00E+03	2.27E+02	N	7.23E+01	nc	7.23E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	0.4 J	4	ug/L	HB-HBSW-06	7/13	5-5	4.00E+00		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) N/A - No background screening performed.
(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.
(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.20b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - HARBOR BROOK SURFACE WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HBSW-06	5/7/2001			1330-20-7	XYLENES, TOTAL	Y		ug/l	4	4
HB-HBSW-06	6/2/2003			XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.2	
HB-HBSW-06	6/2/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-06	6/2/2003	0	0	CALCULATED	TOTAL	Y	J	ug/l		1.2
HB-HBSW-06	9/9/2003			XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.6	
HB-HBSW-06	9/9/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-06	9/9/2003	0	0	CALCULATED	TOTAL	Y	J	ug/l		1.6
HB-HBSW-07	5/7/2001			1330-20-7	XYLENES, TOTAL	Y		ug/l	3	3
HB-HBSW-07	6/2/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-07	6/2/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-07	6/2/2003	0	0	CALCULATED	TOTAL	N	U	ug/l		5
HB-HBSW-07	9/9/2003			XYLENES1314	XYLENES, M & P	Y	J	ug/l	1.5	
HB-HBSW-07	9/9/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-07	9/9/2003	0	0	CALCULATED	TOTAL	Y	J	ug/l		1.5
HB-HBSW-08	5/7/2001			1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.4	0.4
HB-HBSW-08	6/3/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-08	6/3/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-08	6/3/2003	0	0	CALCULATED	TOTAL	N	U	ug/l		5
HB-HBSW-08	9/9/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-08	9/9/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-08	9/9/2003	0	0	CALCULATED	TOTAL	N	U	ug/l		5
HB-HBSW-09	5/7/2001			1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.4	0.4
HB-HBSW-09	6/3/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-09	6/3/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-09	6/3/2003	0	0	CALCULATED	TOTAL	N	U	ug/l		5
HB-HBSW-09	9/9/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-HBSW-09	9/9/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-HBSW-09	9/9/2003	0	0	CALCULATED	TOTAL	N	U	ug/l		5
HB-SEEP-3	6/12/2003			XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-SEEP-3	6/12/2003			95-47-6	O-XYLENE	N	U	ug/l	5	
HB-SEEP-3	6/12/2003	0	0	CALCULATED	TOTAL	N	U	ug/l		5

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.21a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- EAST FLUME SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-3 ft)^d

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)			
East Flume - Surface Sediment	DIOXIN/FURAN (8)																		
	1746-01-6	2,3,7,8-TCDD Equivalent	0.00002	0.000459	mg/kg	HB-LEF4	14/14	50-50	4.59E-04			1.91E-05	C	3.90E-06	ca	3.90E-06	Y	ASL	
	METALS																		
	7429-90-5	ALUMINUM	1280	6260 J	mg/kg	HB-UEF2	12/12	-	6.26E+03			7.82E+03	N	7.61E+03	nc	7.61E+03	N	BSL	
	7440-38-2	ARSENIC	8	22	mg/kg	HB-UEF7	12/12	-	2.20E+01			4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX	
	7440-39-3	BARIUM	131 J	247	mg/kg	S213S	12/12	-	2.47E+02			1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL	
	7440-41-7	BERYLLIUM	0.11	0.46 J	mg/kg	HB-UEF1	11/12	0.18-0.18	4.60E-01			1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL	
	7440-43-9	CADMIUM	0.73 J	1.6 J	mg/kg	HB-UEF7, HB-UEF1	9/22	0.91-2.5	1.60E+00			3.91E+00	N	3.70E+00	nc	3.70E+00	N	BSL	
	7440-70-2	CALCIUM	166000 J	355000	mg/kg	S210S	22/22	-	3.55E+05			NV	NV	NV	NV	N	NUT		
	7440-47-3	CHROMIUM ^a	8.1	44.1 J	mg/kg	HB-UEF7	22/22	-	4.41E+01			2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX	
	7440-48-4	COBALT	1.9	10.2	mg/kg	S213S	12/12	-	1.02E+01			NV	NV	9.03E+02	ca	9.03E+02	N	BSL	
	7440-50-8	COPPER	8.8	114 J	mg/kg	HB-UEF4	22/22	-	1.14E+02			3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL	
	57-12-5	CYANIDE	0.56	7	mg/kg	S211N	6/13	0.85-1	7.00E+00			2.04E+03	N	1.22E+02	nc	1.22E+02	N	BSL	
	7439-89-6	IRON	2520	15000 J	mg/kg	HB-UEF2	12/12	-	1.50E+04			7.15E+04	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	15.2 J	110	mg/kg	HB-UEF7	22/22	-	1.10E+02			NV	NV	4.00E+02	nc	4.00E+02	N	BSL	
	7439-95-4	MAGNESIUM	6160	25400	mg/kg	S211M	22/22	-	2.54E+04			NV	NV	NV	NV	N	NUT		
	7439-96-5	MANGANESE	181	315	mg/kg	HB-UEF1	12/12	-	3.15E+02			2.04E+03	N	1.76E+02	nc	1.76E+02	Y	ASL	
	7439-97-6	MERCURY ^b	0.81	7.5	mg/kg	S213N	26/26	-	7.50E+00			7.82E-01	N	6.11E-01	nc	6.11E-01	Y	ASL	
	7440-02-0	NICKEL	4.6	22.5	mg/kg	S212S	20/22	3.9-4.5	2.25E+01			2.04E+03	N	1.56E+02	nc	1.56E+02	N	BSL	
	7440-09-7	POTASSIUM	193	1010	mg/kg	S214N	12/12	-	1.01E+03			NV	NV	NV	NV	N	NUT		
	7440-22-4	SILVER	0.44 J	0.89 J	mg/kg	HB-UEF6	6/12	0.43-2.7	8.90E-01			5.11E+02	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-23-5	SODIUM	987	8240	mg/kg	S214N	22/22	-	8.24E+03			NV	NV	NV	NV	N	NUT		
	7440-62-2	VANADIUM	1.6	27.1 J	mg/kg	HB-UEF4	12/12	-	2.71E+01			1.02E+02	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	95.4 J	643 J	mg/kg	S213S	22/22	-	6.43E+02			3.07E+04	N	2.35E+03	nc	2.35E+03	N	BSL	
	PCBs																		
		HIGHLY CHLORINATED PCBs ^e		0.048	0.66	mg/kg	HB-UEF2	16/27	0.031-0.66	6.60E-01			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
		TOTAL PCBs ^f		0.048	0.66	mg/kg	HB-UEF2	16/27	0.031-0.66	6.60E-01			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
	PESTICIDES																		
	72-54-8	4,4'-DDD		0.034 J	0.034 J	mg/kg	HB-UEF1	1/17	0.0074-0.14	3.40E-02			1.19E+01	C	2.44E+00	ca	2.44E+00	N	BSL
	72-55-9	4,4'-DDE		0.0084 J	0.021 J	mg/kg	HB-UEF2	4/17	0.0074-0.14	2.10E-02			8.42E+00	C	1.72E+00	ca	1.72E+00	N	BSL
	57-74-9	TOTAL CHLORDANE ^g		0.015J	0.017J	mg/kg	HB-UEF6	2/16	0.0038-0.42	1.70E-02			1.82E+00	C	1.62E+00	ca	1.62E+00	N	BSL
	53494-70-5	ENDRIN KETONE		0.043 J	0.15	mg/kg	HB-UEF1	4/12	0.0074-0.14	1.50E-01			NV	NV	NV	NV	Y	NTX	
	1024-57-3	HEPTACHLOR EPOXIDE		0.063 J	0.063 J	mg/kg	HB-UEF2	1/17	0.0038-0.069	6.30E-02			3.14E-01	C	5.34E-02	ca	5.34E-02	Y	ASL
	72-43-5	METHOXYCHLOR		0.042 J	0.18 J	mg/kg	HB-UEF2	3/17	0.038-0.69	1.80E-01			5.11E+02	N	3.06E+01	nc	3.06E+01	N	BSL
	SVOCs																		
	95-94-3	1,2,4,5-TETRACHLOROBENZENE		0.009 J	0.15 J	mg/kg	S214M	10/10	-	1.50E-01			2.35E+01	N	1.83E+01	nc	1.83E+01	N	BSL
	90-12-0	1-METHYLNAPHTHALENE		0.007 J	4.6 J	mg/kg	S212N	5/5	-	4.60E+00			NV	NV	NV	NV	Y	NTX	
	91-57-6	2-METHYLNAPHTHALENE		0.071 J	39 J	mg/kg	HB-UEF7	6/12	0.11-160	3.90E+01			4.09E+02	N	NV	NV	4.09E+02	N	BSL
	34METPH	3&4-METHYLPHENOL ^h		0.13 J	16 J	mg/kg	HB-UEF6	4/7	6.7-180	1.60E+01			3.91E+01	N	3.06E+01	nc	3.06E+01	N	BSL
	106-47-8	4-CHLOROANILINE		0.11 J	0.11 J	mg/kg	HB-UEF1	1/12	0.11-180	1.10E-01			4.09E+02	N	2.44E+01	nc	2.44E+01	N	BSL
	106-44-5	4-METHYLPHENOL		0.2 J	0.25	mg/kg	S214N	2/5	0.11-0.33	2.50E-01			5.11E+02	N	3.06E+01	nc	3.06E+01	N	BSL
	83-32-9	ACENAPHTHENE		0.066 J	85 J	mg/kg	HB-UEF7	5/12	0.11-6.8	8.50E+01			6.13E+03	N	3.68E+02	nc	3.68E+02	N	BSL
	208-96-8	ACENAPHTHYLENE		0.061 J	0.14 J	mg/kg	S214N	3/12	0.11-180	1.40E-01			NV	NV	NV	NV	Y	NTX	
	120-12-7	ANTHRACENE		0.12 J	160 J	mg/kg	HB-UEF7	7/12	0.11-6.8	1.60E+02			3.07E+04	N	2.19E+03	nc	2.19E+03	N	BSL
	56-55-3	BENZ(A)ANTHRACENE		0.063 J	460 J	mg/kg	HB-UEF7	12/12	-	4.60E+02			3.92E+00	C	6.21E-01	ca	6.21E-01	Y	ASL
	50-32-8	BENZO(A)PYRENE		0.074 J	480 J	mg/kg	HB-UEF7	12/12	-	4.80E+02			3.92E-01	C	6.21E-02	ca	6.21E-02	Y	ASL

TABLE 2.21a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- EAST FLUME SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-3 ft)^d

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
	205-99-2	BENZO(B)FLUORANTHENE	0.1 J	720 J	mg/kg	HB-UEF7	12/12	-	7.20E+02			3.92E+00	C	6.21E-01	ca	6.21E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.04 J	280 J	mg/kg	HB-UEF7	12/12	-	2.80E+02			NV	C	NV	ca	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.096 J	270 J	mg/kg	HB-UEF7	12/12	-	2.70E+02			3.92E+01	C	6.21E+00	ca	6.21E+00	Y	ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHthalATE	0.24	290 J	mg/kg	HB-UEF6	12/12	-	2.90E+02			2.04E+02	C	3.47E+01	ca	3.47E+01	Y	ASL
	85-68-7	BUTYLBENZYL PHthalATE	0.24 J	20 J	mg/kg	HB-UEF7	2/12	0.11-160	2.00E+01			2.04E+04	N	1.22E+03	nc	1.22E+03	N	BSL
	86-74-8	CARBAZOLE	0.054 J	93 J	mg/kg	HB-UEF7	7/12	0.11-6.8	9.30E+01			1.43E+02	C	2.43E+01	ca	2.43E+01	Y	ASL
	218-01-9	CHRYSENE	0.1 J	650 J	mg/kg	HB-UEF7	12/12	-	6.50E+02			3.92E+02	C	6.21E+01	ca	6.21E+01	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	1.7 J	72 J	mg/kg	HB-UEF7	5/11	0.11-6.8	7.20E+01			3.92E-01	C	6.21E-02	ca	6.21E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.12 J	80 J	mg/kg	HB-UEF7	5/12	0.11-6.8	8.00E+01			1.02E+02	N	1.45E+01	nc	1.45E+01	Y	ASL
	117-84-0	DI-N-OCTYL PHthalATE	0.031 J	37 J	mg/kg	HB-UEF6	8/12	0.21-6.8	3.70E+01			NV	N	2.44E+02	nc	2.44E+02	N	BSL
	206-44-0	FLUORANTHENE	0.14	990	mg/kg	HB-UEF7	12/12	-	9.90E+02			4.09E+03	N	2.29E+02	nc	2.29E+02	Y	ASL
	86-73-7	FLUORENE	0.028 J	95 J	mg/kg	HB-UEF7	9/12	6.3-6.8	9.50E+01			4.09E+03	N	2.75E+02	nc	2.75E+02	N	BSL
	118-74-1	HEXACHLORO BENZENE	0.014 J	0.25 J	mg/kg	S213N	15/27	0.11-180	2.50E-01			1.79E+00	C	3.04E-01	ca	3.04E-01	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.041 J	230 J	mg/kg	HB-UEF7	12/12	-	2.30E+02			3.92E+00	C	6.21E-01	ca	6.21E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.024 J	94 J	mg/kg	HB-UEF7	13/17	0.0075-6.8	9.40E+01			2.04E+03	N	5.59E+00	nc	5.59E+00	Y	ASL
	544-76-3	N-HEXADACANE	0.52	0.83	mg/kg	S211N	2/2	-	8.30E-01			NV	N	NV	nc	NV	Y	NTX
	608-93-5	PENTACHLORO BENZENE	0.01 J	0.067 J	mg/kg	S212S	10/10	-	6.70E-02			6.26E+01	N	4.89E+01	nc	4.89E+01	N	BSL
	85-01-8	PHENANTHRENE	0.07 J	780	mg/kg	HB-UEF7	12/12	-	7.80E+02			NV	N	NV	nc	NV	Y	NTX
	108-95-2	PHENOL	0.068 J	3.3	mg/kg	S214N	4/12	0.11-180	3.30E+00			3.07E+04	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.17	1300 J	mg/kg	HB-UEF7	12/12	-	1.30E+03			3.07E+03	N	2.32E+02	nc	2.32E+02	Y	ASL
	VOCs																	
	87-61-6	1,2,3-TRICHLOROBENZENE	0.009 J	3.7 J	mg/kg	S214M	6/15	0.0048-0.023	3.70E+00			NV	N	NV	nc	NV	Y	NTX
	120-82-1	1,2,4-TRICHLOROBENZENE	0.071 J	1.8	mg/kg	S214N	5/20	0.0048-5.7	1.80E+00			1.02E+03	N	6.22E+00	nc	6.22E+00	N	BSL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.0067 J	0.047 J	mg/kg	HB-EF5	4/5	0.0048-0.0048	4.70E-02			NV	N	5.16E+01	nc	5.16E+01	N	BSL
	95-50-1	1,2-DICHLOROBENZENE	0.013 J	120	mg/kg	S214M	27/27	-	1.20E+02			9.20E+03	N	6.00E+01	nc	6.00E+01	Y	ASL
	540-59-0	1,2-DICHLOROETHENE (TOTAL)	0.002 J	0.003 J	mg/kg	HB-UEF4	2/12	0.019-5.6	3.00E-03			7.04E+02	N	NV	nc	7.04E+02	N	BSL
	108-70-3	1,3,5-TRICHLOROBENZENE	0.005 J	15	mg/kg	S214M	9/10	0.023-0.023	1.50E+01			NV	N	NV	nc	NV	Y	NTX
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.0025 J	0.045 J	mg/kg	HB-EF5	5/5	-	4.50E-02			NV	N	5.16E+00	nc	5.16E+00	N	BSL
	541-73-1	1,3-DICHLOROBENZENE	0.005 J	14	mg/kg	S214M	13/27	0.0048-180	1.40E+01			3.07E+02	N	5.31E+01	nc	5.31E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.015 J	160 J	mg/kg	HB-UEF7	27/27	-	1.60E+02			1.19E+02	C	3.45E+00	ca	3.45E+00	Y	ASL
	78-93-3	2-BUTANONE	0.007 J	0.041 J	mg/kg	HB-UEF4	7/12	0.023-5.6	4.10E-02			6.13E+04	N	2.23E+03	nc	2.23E+03	N	BSL
	95-49-8	2-CHLOROTOLUENE	0.0035 J	0.0035 J	mg/kg	HB-EF5	1/5	0.0048-0.0093	3.50E-03			1.56E+02	N	1.58E+01	nc	1.58E+01	N	BSL
	108-10-1	4-METHYL-2-PENTANONE	0.002 J	0.002 J	mg/kg	HB-UEF3, HB-UEF6	2/12	0.019-5.6	2.00E-03			NV	N	5.28E+02	nc	5.28E+02	N	BSL
	67-64-1	ACETONE	0.045 J	0.13 J	mg/kg	HB-UEF4	6/12	0.026-5.6	1.30E-01			9.20E+04	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.008 J	1.3 J	mg/kg	S214M	21/27	0.009-5.6	1.30E+00			5.20E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.005 J	0.041 J	mg/kg	HB-UEF5	7/12	0.023-5.6	4.10E-02			1.02E+04	N	3.55E+01	nc	3.55E+01	N	BSL
	108-90-7	CHLOROBENZENE	0.007 J	36	mg/kg	S214M	27/27	-	3.60E+01			2.04E+03	N	1.51E+01	nc	1.51E+01	Y	ASL
	74-87-3	CHLOROMETHANE	0.004 J	0.004 J	mg/kg	HB-UEF4	1/17	0.0097-5.6	4.00E-03			NV	NV	4.69E+00	nc	4.69E+00	N	BSL
	156-59-2	CIS-1,2-DICHLOROETHENE	0.002 J	0.003 J	mg/kg	HB-UEF4	2/12	0.0048-0.02	3.00E-03			1.02E+03	N	4.29E+00	nc	4.29E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.003 J	0.065	mg/kg	S214S	14/27	0.0048-5.7	6.50E-02			1.02E+04	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.0061 J	0.022 J	mg/kg	HB-EF5	2/5	0.0048-0.0093	2.20E-02			1.02E+04	N	5.72E+01	nc	5.72E+01	N	BSL

TABLE 2.21a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- EAST FLUME SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-3 ft)^d

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)
	99-87-6	P-ISOPROPYLTOLUENE	0.0023 J	0.016 J	mg/kg	HB-EF5	4/5	0.0048-0.0048	1.60E-02			NV	NV	NV	Y	NTX
	135-98-8	SEC-BUTYLBENZENE	0.0035 J	0.013 J	mg/kg	HB-EF5	3/5	0.0048-0.0093	1.30E-02			NV	2.20E+02	nc	N	BSL
	108-88-3	TOLUENE	0.003 J	4.6 J	mg/kg	S214M	23/27	0.0048-0.023	4.60E+00			8.18E+03	5.20E+01	nc	N	BSL
	79-01-6	TRICHLOROETHENE	0.005 J	0.005 J	mg/kg	S213S	1/17	0.0048-5.6	5.00E-03			7.15E+00	5.30E-02	ca	N	BSL
	1330-20-7	XYLENES, TOTAL	0.002 J	15	mg/kg	S214N	24/27	0.006-0.018	1.50E+01			2.04E+04	2.71E+01	nc	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(8) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.21b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = HB-UEF7 site used an estimated value for maximum detected concentration.
d = Sediment samples with start depths of 0 ft and end depths ranging from >1 to 3 ft were incorporated in the evaluation of surface sediment.
e = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on 1254.
f = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on 1254.
g = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
h = RBC and PRG values for 4-methylphenol utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
CUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-EF8	11/19/1997	0	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	N	100	50	ng/kg	U	0.01	0.5
HB-EF8	11/19/1997	0	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	1500	1500	ng/kg	J	0.01	15
HB-EF8	11/19/1997	0	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	80	40	ng/kg	U	0.01	0.4
HB-EF8	11/19/1997	0	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2600	2600	ng/kg	J	0.1	260
HB-EF8	11/19/1997	0	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	110	110	ng/kg	J	0.1	11
HB-EF8	11/19/1997	0	1	57653-85-7	1,2,3,6,7,8-HXCDD	N	70	35	ng/kg	U	0.1	3.5
HB-EF8	11/19/1997	0	1	57117-44-9	1,2,3,6,7,8-HXCDF	N	40	20	ng/kg	U	0.1	2
HB-EF8	11/19/1997	0	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	80	40	ng/kg	U	0.1	4
HB-EF8	11/19/1997	0	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	60	30	ng/kg	U	0.1	3
HB-EF8	11/19/1997	0	1	40321-76-4	1,2,3,7,8-PECDD	N	90	45	ng/kg	U	1	45
HB-EF8	11/19/1997	0	1	57117-41-6	1,2,3,7,8-PECDF	Y	170	170	ng/kg	J	0.03	5.1
HB-EF8	11/19/1997	0	1	1746-01-6	2,3,7,8-TCDD	N	50	25	ng/kg	U	1	25
HB-EF8	11/19/1997	0	1	51207-31-9	2,3,7,8-TCDF	Y	840	840	ng/kg	J	0.1	84
HB-EF8	11/19/1997	0	1	3268-87-9	OCDD	N	100	50	ng/kg	U	3E-04	0.015
HB-EF8	11/19/1997	0	1	39001-02-0	OCDF	Y	180	180	ng/kg	J	3E-04	0.054
Sample Location TEQ =												458.6
HB-LEF1	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	147	147	ng/kg	J	0.01	1.47
HB-LEF1	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	20.2	20.2	ng/kg	J	0.01	0.202
HB-LEF1	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	20.6	20.6	ng/kg	J	0.1	2.06
HB-LEF1	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	76	76	ng/kg	J	0.1	7.6
HB-LEF1	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	55.3	55.3	ng/kg	J	0.1	5.53
HB-LEF1	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	20	20	ng/kg	J	0.1	2
HB-LEF1	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	33.5	33.5	ng/kg	J	0.1	3.35
HB-LEF1	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	3.63	1.815	ng/kg	UJ	0.1	0.1815
HB-LEF1	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	22.1	22.1	ng/kg	J	1	22.1
HB-LEF1	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	40.3	40.3	ng/kg	J	0.03	1.209
HB-LEF1	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	4.78	4.78	ng/kg	J	1	4.78
HB-LEF1	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	108	108	ng/kg	J	0.1	10.8
HB-LEF1	9/29/1998	0	0.5	3268-87-9	OCDD	Y	2570	2570	ng/kg	J	3E-04	0.771
HB-LEF1	9/29/1998	0	0.5	39001-02-0	OCDF	Y	382	382	ng/kg	J	3E-04	0.1146
Sample Location TEQ =												62.2

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-LEF2	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	110	110	ng/kg	J	0.01	1.1
HB-LEF2	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	19.7	19.7	ng/kg	J	0.01	0.197
HB-LEF2	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	18.1	18.1	ng/kg	J	0.1	1.81
HB-LEF2	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	178	178	ng/kg	J	0.1	17.8
HB-LEF2	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	43.2	43.2	ng/kg	J	0.1	4.32
HB-LEF2	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	32.6	32.6	ng/kg	J	0.1	3.26
HB-LEF2	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	22.3	22.3	ng/kg	J	0.1	2.23
HB-LEF2	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	6.03	6.03	ng/kg	J	0.1	0.603
HB-LEF2	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	20.3	20.3	ng/kg	J	1	20.3
HB-LEF2	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	315	315	ng/kg	J	0.03	9.45
HB-LEF2	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	8.07	8.07	ng/kg	J	1	8.07
HB-LEF2	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1580	1580	ng/kg	J	0.1	158
HB-LEF2	9/29/1998	0	0.5	3268-87-9	OCDD	Y	1800	1800	ng/kg	J	3E-04	0.54
HB-LEF2	9/29/1998	0	0.5	39001-02-0	OCDF	Y	302	302	ng/kg	J	3E-04	0.0906
Sample Location TEQ =												227.8
HB-LEF3	9/29/1998	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	124	124	ng/kg	J	0.01	1.24
HB-LEF3	9/29/1998	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	45.7	45.7	ng/kg	J	0.01	0.457
HB-LEF3	9/29/1998	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	75.5	75.5	ng/kg	J	0.1	7.55
HB-LEF3	9/29/1998	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	267	267	ng/kg	J	0.1	26.7
HB-LEF3	9/29/1998	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	174	174	ng/kg	J	0.1	17.4
HB-LEF3	9/29/1998	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	52.3	52.3	ng/kg	J	0.1	5.23
HB-LEF3	9/29/1998	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	96	96	ng/kg	J	0.1	9.6
HB-LEF3	9/29/1998	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	11	11	ng/kg	J	0.1	1.1
HB-LEF3	9/29/1998	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	95.9	95.9	ng/kg	J	1	95.9
HB-LEF3	9/29/1998	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	159	159	ng/kg	J	0.03	4.77
HB-LEF3	9/29/1998	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	4.38	4.38	ng/kg	J	1	4.38
HB-LEF3	9/29/1998	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	449	449	ng/kg	J	0.1	44.9
HB-LEF3	9/29/1998	0.5	1	3268-87-9	OCDD	Y	888	888	ng/kg	J	3E-04	0.2664
HB-LEF3	9/29/1998	0.5	1	39001-02-0	OCDF	Y	531	531	ng/kg	J	3E-04	0.1593
Sample Location TEQ =												219.7

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-LEF4	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	33.7	33.7	ng/kg	J	0.01	0.337
HB-LEF4	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	6.39	6.39	ng/kg	J	0.01	0.0639
HB-LEF4	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.55	6.55	ng/kg	J	0.1	0.655
HB-LEF4	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	61.5	61.5	ng/kg	J	0.1	6.15
HB-LEF4	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	25	25	ng/kg	J	0.1	2.5
HB-LEF4	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	10.7	10.7	ng/kg	J	0.1	1.07
HB-LEF4	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	12.9	12.9	ng/kg	J	0.1	1.29
HB-LEF4	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.86	1.43	ng/kg	UJ	0.1	0.143
HB-LEF4	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	9.55	9.55	ng/kg	J	1	9.55
HB-LEF4	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	17.1	17.1	ng/kg	J	0.03	0.513
HB-LEF4	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	8.76	8.76	ng/kg	J	1	8.76
HB-LEF4	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	49.9	49.9	ng/kg	J	0.1	4.99
HB-LEF4	9/29/1998	0	0.5	3268-87-9	OCDD	Y	1150	1150	ng/kg	J	3E-04	0.345
HB-LEF4	9/29/1998	0	0.5	39001-02-0	OCDF	Y	56.5	56.5	ng/kg	J	3E-04	0.01695
Sample Location TEQ =												36.4
HB-LEF5	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	42.6	42.6	ng/kg	J	0.01	0.426
HB-LEF5	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.97	4.97	ng/kg	J	0.01	0.0497
HB-LEF5	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	5.69	5.69	ng/kg	J	0.1	0.569
HB-LEF5	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	28.3	28.3	ng/kg	J	0.1	2.83
HB-LEF5	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	18.4	18.4	ng/kg	J	0.1	1.84
HB-LEF5	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	8.74	8.74	ng/kg	J	0.1	0.874
HB-LEF5	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	9.92	9.92	ng/kg	J	0.1	0.992
HB-LEF5	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.01	1.01	ng/kg	J	0.1	0.101
HB-LEF5	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	9.03	9.03	ng/kg	J	1	9.03
HB-LEF5	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	15.3	15.3	ng/kg	J	0.03	0.459
HB-LEF5	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.2	2.2	ng/kg	J	1	2.2
HB-LEF5	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	37.4	37.4	ng/kg	J	0.1	3.74
HB-LEF5	9/29/1998	0	0.5	3268-87-9	OCDD	Y	896	896	ng/kg	J	3E-04	0.2688
HB-LEF5	9/29/1998	0	0.5	39001-02-0	OCDF	Y	74.4	74.4	ng/kg	J	3E-04	0.02232
Sample Location TEQ =												23.4

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-LEF5	9/29/1998	0.5	0.96	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	41.3	41.3	ng/kg	J	0.01	0.413
HB-LEF5	9/29/1998	0.5	0.96	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	13.5	13.5	ng/kg	J	0.01	0.135
HB-LEF5	9/29/1998	0.5	0.96	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.82	6.82	ng/kg	J	0.1	0.682
HB-LEF5	9/29/1998	0.5	0.96	70648-26-9	1,2,3,4,7,8-HXCDF	Y	62.9	62.9	ng/kg	J	0.1	6.29
HB-LEF5	9/29/1998	0.5	0.96	57653-85-7	1,2,3,6,7,8-HXCDD	Y	20.9	20.9	ng/kg	J	0.1	2.09
HB-LEF5	9/29/1998	0.5	0.96	57117-44-9	1,2,3,6,7,8-HXCDF	Y	14.8	14.8	ng/kg	J	0.1	1.48
HB-LEF5	9/29/1998	0.5	0.96	19408-74-3	1,2,3,7,8,9-HXCDD	Y	9.68	9.68	ng/kg	J	0.1	0.968
HB-LEF5	9/29/1998	0.5	0.96	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.61	0.805	ng/kg	UJ	0.1	0.0805
HB-LEF5	9/29/1998	0.5	0.96	40321-76-4	1,2,3,7,8-PECDD	Y	11	11	ng/kg	J	1	11
HB-LEF5	9/29/1998	0.5	0.96	57117-41-6	1,2,3,7,8-PECDF	Y	29.3	29.3	ng/kg	J	0.03	0.879
HB-LEF5	9/29/1998	0.5	0.96	1746-01-6	2,3,7,8-TCDD	Y	2.77	2.77	ng/kg	J	1	2.77
HB-LEF5	9/29/1998	0.5	0.96	51207-31-9	2,3,7,8-TCDF	Y	64.9	64.9	ng/kg	J	0.1	6.49
HB-LEF5	9/29/1998	0.5	0.96	3268-87-9	OCDD	Y	357	357	ng/kg	J	3E-04	0.1071
HB-LEF5	9/29/1998	0.5	0.96	39001-02-0	OCDF	Y	75.7	75.7	ng/kg	J	3E-04	0.02271
Sample Location TEQ =												33.4
HB-UEF1	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	155	155	ng/kg	U	0.01	1.55
HB-UEF1	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	15	15	ng/kg		0.01	0.15
HB-UEF1	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	9.91	9.91	ng/kg		0.1	0.991
HB-UEF1	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	15.7	15.7	ng/kg		0.1	1.57
HB-UEF1	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	24.6	24.6	ng/kg		0.1	2.46
HB-UEF1	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	8.2	8.2	ng/kg		0.1	0.82
HB-UEF1	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	19.3	19.3	ng/kg		0.1	1.93
HB-UEF1	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.16	0.58	ng/kg		0.1	0.058
HB-UEF1	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	5.9	5.9	ng/kg		1	5.9
HB-UEF1	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	6.79	6.79	ng/kg		0.03	0.2037
HB-UEF1	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.75	1.75	ng/kg		1	1.75
HB-UEF1	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	26.2	26.2	ng/kg		0.1	2.62
HB-UEF1	9/29/1998	0	0.5	3268-87-9	OCDD	Y	4420	4420	ng/kg		3E-04	1.326
HB-UEF1	9/29/1998	0	0.5	39001-02-0	OCDF	Y	380	380	ng/kg		3E-04	0.114
Sample Location TEQ =												21.4

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-UEF2	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	244	244	ng/kg	U	0.01	2.44
HB-UEF2	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	15	15	ng/kg		0.01	0.15
HB-UEF2	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	12.6	12.6	ng/kg		0.1	1.26
HB-UEF2	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	16.4	16.4	ng/kg		0.1	1.64
HB-UEF2	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	32.3	32.3	ng/kg		0.1	3.23
HB-UEF2	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	13.5	13.5	ng/kg		0.1	1.35
HB-UEF2	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	29.4	29.4	ng/kg		0.1	2.94
HB-UEF2	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.92	0.46	ng/kg		0.1	0.046
HB-UEF2	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	7.36	7.36	ng/kg		1	7.36
HB-UEF2	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	6.24	6.24	ng/kg		0.03	0.1872
HB-UEF2	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.85	2.85	ng/kg		1	2.85
HB-UEF2	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	26.5	26.5	ng/kg		0.1	2.65
HB-UEF2	9/29/1998	0	0.5	3268-87-9	OCDD	Y	7970	7970	ng/kg		3E-04	2.391
HB-UEF2	9/29/1998	0	0.5	39001-02-0	OCDF	Y	590	590	ng/kg	3E-04	0.177	
Sample Location TEQ =												28.7
HB-UEF3	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	217	217	ng/kg	U	0.01	2.17
HB-UEF3	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	14.7	14.7	ng/kg		0.01	0.147
HB-UEF3	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	12.8	12.8	ng/kg		0.1	1.28
HB-UEF3	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	13.1	13.1	ng/kg		0.1	1.31
HB-UEF3	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	31.9	31.9	ng/kg		0.1	3.19
HB-UEF3	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	10.4	10.4	ng/kg		0.1	1.04
HB-UEF3	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	29.1	29.1	ng/kg		0.1	2.91
HB-UEF3	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.1	0.55	ng/kg		0.1	0.055
HB-UEF3	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	6.92	6.92	ng/kg		1	6.92
HB-UEF3	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.14	4.14	ng/kg		0.03	0.1242
HB-UEF3	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.49	1.49	ng/kg		1	1.49
HB-UEF3	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	11.4	11.4	ng/kg		0.1	1.14
HB-UEF3	9/29/1998	0	0.5	3268-87-9	OCDD	Y	9820	9820	ng/kg		3E-04	2.946
HB-UEF3	9/29/1998	0	0.5	39001-02-0	OCDF	Y	597	597	ng/kg	3E-04	0.1791	
Sample Location TEQ =												24.9

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-UEF4	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	253	253	ng/kg	U	0.01	2.53
HB-UEF4	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	13	13	ng/kg		0.01	0.13
HB-UEF4	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	12.6	12.6	ng/kg		0.1	1.26
HB-UEF4	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	15.7	15.7	ng/kg		0.1	1.57
HB-UEF4	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	36.1	36.1	ng/kg		0.1	3.61
HB-UEF4	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	13.7	13.7	ng/kg		0.1	1.37
HB-UEF4	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	28.5	28.5	ng/kg		0.1	2.85
HB-UEF4	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.01	0.505	ng/kg		0.1	0.0505
HB-UEF4	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	7.84	7.84	ng/kg		1	7.84
HB-UEF4	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	5.77	5.77	ng/kg		0.03	0.1731
HB-UEF4	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.47	1.47	ng/kg		1	1.47
HB-UEF4	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	14.3	14.3	ng/kg		0.1	1.43
HB-UEF4	9/29/1998	0	0.5	3268-87-9	OCDD	Y	8920	8920	ng/kg		3E-04	2.676
HB-UEF4	9/29/1998	0	0.5	39001-02-0	OCDF	Y	586	586	ng/kg	3E-04	0.1758	
Sample Location TEQ =												27.1
HB-UEF5	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	215	215	ng/kg	U	0.01	2.15
HB-UEF5	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	11.3	11.3	ng/kg		0.01	0.113
HB-UEF5	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	13.7	13.7	ng/kg		0.1	1.37
HB-UEF5	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	14.8	14.8	ng/kg		0.1	1.48
HB-UEF5	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	32.4	32.4	ng/kg		0.1	3.24
HB-UEF5	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	12.5	12.5	ng/kg		0.1	1.25
HB-UEF5	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	27.1	27.1	ng/kg		0.1	2.71
HB-UEF5	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.2	1.1	ng/kg		0.1	0.11
HB-UEF5	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	7.55	7.55	ng/kg		1	7.55
HB-UEF5	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	5.61	5.61	ng/kg		0.03	0.1683
HB-UEF5	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.97	1.97	ng/kg		1	1.97
HB-UEF5	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	12.6	12.6	ng/kg		0.1	1.26
HB-UEF5	9/29/1998	0	0.5	3268-87-9	OCDD	Y	8760	8760	ng/kg		3E-04	2.628
HB-UEF5	9/29/1998	0	0.5	39001-02-0	OCDF	Y	496	496	ng/kg	3E-04	0.1488	
Sample Location TEQ =												26.1

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-UEF6	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	158	158	ng/kg	U	0.01	1.58
HB-UEF6	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.87	8.87	ng/kg		0.01	0.0887
HB-UEF6	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	9.66	9.66	ng/kg		0.1	0.966
HB-UEF6	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	11.5	11.5	ng/kg		0.1	1.15
HB-UEF6	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	25.9	25.9	ng/kg		0.1	2.59
HB-UEF6	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	8.49	8.49	ng/kg		0.1	0.849
HB-UEF6	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	22.5	22.5	ng/kg		0.1	2.25
HB-UEF6	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.57	0.785	ng/kg		0.1	0.0785
HB-UEF6	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	5.96	5.96	ng/kg		1	5.96
HB-UEF6	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.63	4.63	ng/kg		0.03	0.1389
HB-UEF6	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.28	1.28	ng/kg		1	1.28
HB-UEF6	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	13.9	13.9	ng/kg		0.1	1.39
HB-UEF6	9/29/1998	0	0.5	3268-87-9	OCDD	Y	8060	8060	ng/kg		3E-04	2.418
HB-UEF6	9/29/1998	0	0.5	39001-02-0	OCDF	Y	383	383	ng/kg		3E-04	0.1149
Sample Location TEQ =												20.9

TABLE 2.21b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-UEF7	9/29/1998	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	429	429	ng/kg	J	0.01	4.29
HB-UEF7	9/29/1998	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	125	125	ng/kg	J	0.01	1.25
HB-UEF7	9/29/1998	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.42	8.42	ng/kg	J	0.01	0.0842
HB-UEF7	9/29/1998	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	9.04	9.04	ng/kg	J	0.1	0.904
HB-UEF7	9/29/1998	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	13.7	13.7	ng/kg	J	0.1	1.37
HB-UEF7	9/29/1998	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	25.2	25.2	ng/kg	J	0.1	2.52
HB-UEF7	9/29/1998	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	8.03	8.03	ng/kg	J	0.1	0.803
HB-UEF7	9/29/1998	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	19.9	19.9	ng/kg	J	0.1	1.99
HB-UEF7	9/29/1998	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.51	1.51	ng/kg	J	0.1	0.151
HB-UEF7	9/29/1998	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	6.1	6.1	ng/kg	J	1	6.1
HB-UEF7	9/29/1998	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.66	4.66	ng/kg	J	0.03	0.1398
HB-UEF7	9/29/1998	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.56	2.56	ng/kg	J	1	2.56
HB-UEF7	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	41	41	ng/kg	J	0.1	4.1
HB-UEF7	9/29/1998	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	12.8	12.8	ng/kg	J	0.1	1.28
HB-UEF7	9/29/1998	0	0.5	3268-87-9	OCDD	Y	3430	3430	ng/kg	J	3E-04	1.029
HB-UEF7	9/29/1998	0	0.5	39001-02-0	OCDF	Y	352	352	ng/kg	J	3E-04	0.1056
Sample Location TEQ =											28.7	

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.21c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-EF7	0	2.25	10/2/1997	0.21	mg/kg
Highly Chlorinated PCBs	HB-UEF2	0	0.5	9/30/1998	0.66	mg/kg
Highly Chlorinated PCBs	HB-UEF3	0	0.5	9/30/1998	0.45	mg/kg
Highly Chlorinated PCBs	HB-UEF4	0	0.5	9/30/1998	0.21	mg/kg
Highly Chlorinated PCBs	HB-UEF5	0	0.5	9/30/1998	0.2	mg/kg
Highly Chlorinated PCBs	HB-UEF6	0	0.5	9/30/1998	0.22	mg/kg
Highly Chlorinated PCBs	HB-UEF7	0	0.5	9/30/1998	0.4	mg/kg
Highly Chlorinated PCBs	S210M	0	0	8/24/1993	0.074	mg/kg
Highly Chlorinated PCBs	S211M	0	0	8/24/1993	0.053	mg/kg
Highly Chlorinated PCBs	S211S	0	0	8/24/1993	0.104	mg/kg
Highly Chlorinated PCBs	S212M	0	0	8/24/1993	0.048	mg/kg
Highly Chlorinated PCBs	S212S	0	0	8/24/1993	0.3	mg/kg
Highly Chlorinated PCBs	S213M	0	0	8/24/1993	0.3	mg/kg
Highly Chlorinated PCBs	S213N	0	0	8/24/1993	0.194	mg/kg
Highly Chlorinated PCBs	S214M	0	0	8/24/1993	0.128	mg/kg
Highly Chlorinated PCBs	S214S	0	0	8/24/1993	0.164	mg/kg
Total PCBs	HB-EF7	0	2.25	10/2/1997	0.21	mg/kg
Total PCBs	HB-UEF2	0	0.5	9/30/1998	0.66	mg/kg
Total PCBs	HB-UEF3	0	0.5	9/30/1998	0.45	mg/kg
Total PCBs	HB-UEF4	0	0.5	9/30/1998	0.21	mg/kg
Total PCBs	HB-UEF5	0	0.5	9/30/1998	0.2	mg/kg
Total PCBs	HB-UEF6	0	0.5	9/30/1998	0.22	mg/kg
Total PCBs	HB-UEF7	0	0.5	9/30/1998	0.4	mg/kg
Total PCBs	S210M	0	0	8/24/1993	0.074	mg/kg
Total PCBs	S211M	0	0	8/24/1993	0.053	mg/kg
Total PCBs	S211S	0	0	8/24/1993	0.104	mg/kg
Total PCBs	S212M	0	0	8/24/1993	0.048	mg/kg
Total PCBs	S212S	0	0	8/24/1993	0.3	mg/kg
Total PCBs	S213M	0	0	8/24/1993	0.3	mg/kg
Total PCBs	S213N	0	0	8/24/1993	0.194	mg/kg
Total PCBs	S214M	0	0	8/24/1993	0.128	mg/kg
Total PCBs	S214S	0	0	8/24/1993	0.164	mg/kg

Notes:

* Highly chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.21d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-EF3	10/2/1997	0	3	57-74-9	CHLORDANE	N	UJ	mg/kg	0.074
Total Chlordane =									ND
HB-EF4	10/2/1997	0	1.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.37
Total Chlordane =									ND
HB-EF5	10/2/1997	0	2	57-74-9	CHLORDANE	N	UJ	mg/kg	0.42
Total Chlordane =									ND
HB-EF7	10/2/1997	0	2.25	57-74-9	CHLORDANE	N	U	mg/kg	0.062
Total Chlordane =									ND
HB-UEF1	9/30/1998	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.033
HB-UEF1	9/30/1998	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.033
Total Chlordane =									ND
HB-UEF2	9/30/1998	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.067
HB-UEF2	9/30/1998	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.067
Total Chlordane =									ND
HB-UEF3	9/30/1998	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.062
HB-UEF3	9/30/1998	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.062
Total Chlordane =									ND
HB-UEF4	9/30/1998	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.069
HB-UEF4	9/30/1998	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.015
Total Chlordane =									0.015
HB-UEF5	9/30/1998	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.069
HB-UEF5	9/30/1998	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.069
Total Chlordane =									ND
HB-UEF6	9/30/1998	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.057
HB-UEF6	9/30/1998	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.017
Total Chlordane =									0.017
HB-UEF7	9/30/1998	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.063
HB-UEF7	9/30/1998	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.063
Total Chlordane =									ND
S210N	8/24/1993	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0038
Total Chlordane =									ND
S211N	8/24/1993	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0075
Total Chlordane =									ND
S212N	8/24/1993	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.006
Total Chlordane =									ND
S213S	8/24/1993	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.004
Total Chlordane =									ND
S214N	8/24/1993	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0079
Total Chlordane =									ND

TABLE 2.21e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE SEDIMENT (0-3 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-EF1	10/2/1997	0	1.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.002	0.002
HB-EF3	10/2/1997	0	3	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.016	0.016
HB-EF4	10/2/1997	0	1.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0097	0.0097
HB-EF5	10/2/1997	0	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.093	0.093
HB-EF7	10/2/1997	0	2.25	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.014	0.014
HB-UEF1	9/30/1998	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.014	0.014
HB-UEF2	9/30/1998	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.019	0.019
HB-UEF3	9/30/1998	0	0.5	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.039	0.039
HB-UEF4	9/30/1998	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.065	0.065
HB-UEF5	9/30/1998	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.051	0.051
HB-UEF6	9/30/1998	0	0.5	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.037	0.037
HB-UEF7	9/30/1998	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.006	0.006
S210M	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.67	0.67
S210N	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.12	0.12
S210S	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.26	0.26
S211M	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.42	0.42
S211N	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.23	0.23
S211S	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.27	0.27
S212M	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y	U	mg/kg	0.091	0.091
S212N	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.036	0.018
S212S	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.023	0.0115
S213M	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.006	0.006
S213N	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.012	0.006
S213S	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.03	0.03
S214M	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	12	12
S214N	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	15	15
S214S	8/24/1993	0	0	1330-20-7	XYLENES, TOTAL	Y		mg/kg	1.5	1.5

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.22a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE WATER (OUTFALL)
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Surface Water (Outfall)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
East Flume Surface Water (Outfall)	METALS																	
	7429-90-5	ALUMINUM	0.38	0.38	mg/L	Outfall 015	1/2	0.1-0.1	3.80E-01		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	N	BSL
	7439-89-6	IRON	0.16	0.42	mg/L	Outfall 015	2/2	-	4.20E-01		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	7440-66-6	ZINC	0.053	0.069	mg/L	Outfall 015	2/2	-	6.90E-02		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL
	VOCs																	
	108-90-7	CHLOROBENZENE	0.23	0.36	ug/l	Outfall 015	2/2	-	3.60E-01		1.00E+02	8.96E+00	N	1.06E+01	nc	8.96E+00	N	BSL
	1330-20-7	XYLENES, TOTAL	0.18	0.31	ug/l	Outfall 015	2/2	0-0	3.10E-01		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	N	BSL
	OTHER																	
	25321-22-6	DICHLOROBENZENES ^a	4.8	9.68	ua/l	Outfall 015	2/2	-	9.68E+00		7.50E+01	2.8E-01	C	5.0E-01	ca	2.81E-01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) N/A - No background screening performed.
(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.
(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by
(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

a = Dichlorobenzene data from outfall 015 (east flume) are data that were collected as part of Honeywell's New York State Pollution Discharge Elimination System (SPDES) permit sampling program. As part of that program, Honeywell is responsible for reporting total dichlorobenzenes each month. The SPDES data constitutes the only water data available for the East Flume and was therefore utilized in the HHRA. The screening criterion for 1,4-dichlorobenzene was utilized as a conservative measure to address protection of human health.

TABLE 2.22b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - EAST FLUME SURFACE WATER (OUTFALL)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
Outfall 015	5/5/2003			1330-20-7	XYLENES, TOTAL	Y		ug/l	0.31	0.31
Outfall 015	8/5/2003			1330-20-7	XYLENES, TOTAL	Y		ug/l	0.18	0.18

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.23a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - I-690 STORM SEWER AND DRAINAGE DITCH SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)
I-690 Storm Sewer And Drainage Ditch Surface Sediment	DIOXIN/FURAN (8)															
	1746-01-6	2,3,7,8-TCDD Equivalent	0.0000002	0.00002	mg/kg	HB-HBSED-11	14/14		1.98E-05			4.26E-06	C	3.90E-06	ca	3.90E-06 Y ASL
	METALS															
	7429-90-5	ALUMINUM	1070 J	7300 J	mg/Kg	HB-HBSED-13	14/14	-	7.30E+03			7.82E+03	N	7.61E+03	nc	7.61E+03 N BSL
	7440-36-0	ANTIMONY	0.56 J	0.56 J	mg/Kg	HB-HBSED-12	1/14	0.23-22.2	5.60E-01			3.13E+00	N	3.13E+00	nc	3.13E+00 N BSL
	7440-38-2	ARSENIC	2.3 J	5.2 J	mg/Kg	HB-HBSED-12	12/14	3.7-3.7	5.20E+00			4.26E-01	C	3.90E-01	ca	3.90E-01 Y TOX
	7440-39-3	BARIUM	14.4 J	90.4 J	mg/Kg	HB-HBSED-12	14/14	-	9.04E+01			1.56E+03	N	5.37E+02	nc	5.37E+02 N BSL
	7440-41-7	BERYLLIUM	0.1 J	0.26 J	mg/Kg	HB-HBSED-13	3/14	0.58-1.9	2.60E-01			1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL
	7440-43-9	CADMIUM	0.61	1.2 J	mg/Kg	HB-HBSED-11	5/14	0.37-1.9	1.20E+00			3.91E+00	N	3.70E+00	nc	3.70E+00 N BSL
	7440-70-2	CALCIUM	154000	393000 J	mg/Kg	HB-HBSED-11	14/14	-	3.93E+05			NV	NV	NV	N	NUT
	7440-47-3	CHROMIUM ^a	9.4 J	534 J	mg/Kg	HB-HBSED-12	14/14	-	5.34E+02			2.35E+01	N	3.01E+00	nc	3.01E+00 Y TOX
	7440-48-4	COBALT	2 J	6.8	mg/Kg	HB-DR-72	3/14	1.2-18.5	6.80E+00			NV	N	9.03E+02	ca	9.03E+02 N BSL
	7440-50-8	COPPER	12.6 J	99.5 J	mg/Kg	HB-DR-69	14/14	-	9.95E+01			3.13E+02	N	3.13E+02	nc	3.13E+02 N BSL
	57-12-5	CYANIDE	1.5 J	15.6 J	mg/Kg	HB-HBSED-13	11/14	0.82-2.65	1.56E+01			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	7439-89-6	IRON	1580 J	21300	mg/Kg	HB-DR-69	14/14	-	2.13E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL
	7439-92-1	LEAD	5.9 J	145 J	mg/Kg	HB-HBSED-12	14/14	-	1.45E+02			NV	N	4.00E+02	nc	4.00E+02 N BSL
	7439-95-4	MAGNESIUM	6940	52900 J	mg/Kg	HB-HBSED-13	14/14	-	5.29E+04			NV	N	NV	N	NUT
	7439-96-5	MANGANESE	56 J	365	mg/Kg	HB-DR-70	14/14	-	3.65E+02			1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL
	7439-97-6	MERCURY ^b	0.04	0.75 J	mg/kg	HB-HBSED-12	14/14	-	7.50E-01			7.82E-01	N	6.11E-01	nc	6.11E-01 Y ASL
	7440-02-0	NICKEL	4.6 J	24.8	mg/Kg	HB-DR-70	10/14	8.6-14.8	2.48E+01			1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL
	7440-09-7	POTASSIUM	260 J	801	mg/Kg	HB-DR-69	9/14	415-742	8.01E+02			NV	N	NV	N	NUT
	7782-49-2	SELENIUM	0.4 J	0.59 J	mg/Kg	HB-HBSED-12	3/14	0.58-3.7	5.90E-01			3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-23-5	SODIUM	193	2590 J	mg/Kg	HB-HBSED-13	14/14	-	2.59E+03			NV	N	NV	N	NUT
	7440-62-2	VANADIUM	8.3 J	24	mg/Kg	HB-DR-72	10/14	10.8-18.5	2.40E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL
	7440-66-6	ZINC	48.9 J	844	mg/Kg	HB-DR-72	14/14	-	8.44E+02			2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL
PCBs																
		HIGHLY CHLORINATED PBCs ^c	0.05	0.56	mg/kg	HB-HBSED-11	4/14	0.03-0.26	5.60E-01			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
		TOTAL PCBs ^d	0.05	0.56	mg/kg	HB-HBSED-11	4/14	0.03-0.26	5.60E-01			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
PESTICIDES																
	72-54-8	4,4'-DDD	0.007 J	0.007 J	mg/kg	HB-HBSED-13	1/14	0.008-0.065	7.00E-03			2.66E+00	C	2.44E+00	ca	2.44E+00 N BSL
	57-74-9	TOTAL CHLORDANE ^e	0.004 J	0.005 J	mg/kg	HB-HBSED-12	2/14	0.004-0.033	5.00E-03			1.82E+00	C	1.62E+00	ca	1.62E+00 N BSL
	72-55-9	4,4'-DDE	0.005 J	0.005 J	mg/kg	HB-HBSED-13	1/14	0.008-0.065	5.00E-03			1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	50-29-3	4,4'-DDT	0.008 J	0.008 J	mg/kg	HB-HBSED-11	1/14	0.005-0.065	8.00E-03			1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
SVOCs																
	105-67-9	2,4-DIMETHYLPHENOL	0.16 J	0.52 J	mg/kg	HB-HBSED-12	2/14	0.55-11	5.20E-01			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	91-57-6	2-METHYLNAPHTHALENE	0.28 J	45 J	mg/kg	HB-HBSED-12	7/14	1.2-4.9	4.50E+01			3.13E+01	N	NV	nc	3.13E+01 Y ASL
	95-48-7	2-METHYLPHENOL	0.2 J	0.75 J	mg/kg	HB-HBSED-11	3/14	0.55-11	7.50E-01			3.91E+02	N	3.06E+02	nc	3.06E+02 N BSL
	34METPH	3&4-METHYLPHENOL ^f	0.28 J	2 J	mg/kg	HB-HBSED-12	5/14	0.55-11	2.00E+00			3.91E+01	N	3.06E+01	nc	3.06E+01 N BSL
	83-32-9	ACENAPHTHENE	0.15 J	12 J	mg/kg	HB-HBSED-12	7/14	1.2-4.9	1.20E+01			4.69E+02	N	3.68E+02	nc	3.68E+02 N BSL
	208-96-8	ACENAPHTHYLENE	0.095 J	11 J	mg/kg	HB-HBSED-12	7/14	1.2-4.9	1.10E+01			NV	N	NV	Y	NTX
	120-12-7	ANTHRACENE	0.12 J	2 J	mg/kg	HB-HBSED-12	7/14	0.6-7.3	2.00E+00			2.35E+03	N	2.19E+03	nc	2.19E+03 N BSL
	56-55-3	BENZ(A)ANTHRACENE	0.086 J	2 J	mg/kg	HB-DR-69	8/14	1.2-11	2.00E+00			2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	50-32-8	BENZO(A)PYRENE	0.078 J	2 J	mg/kg	HB-DR-69	10/14	1.2-5.2	2.00E+00			2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.11 J	2.1 J	mg/kg	HB-DR-69	10/14	1.2-5.2	2.10E+00			2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.19 J	1.8 J	mg/kg	HB-DR-69	6/14	0.76-11	1.80E+00			NV	N	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.15 J	1.9 J	mg/kg	HB-DR-69	9/14	0.76-5.2	1.90E+00			2.20E+00	C	6.21E+00	ca	2.20E+00 N BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.72 J	11	mg/kg	HB-DR-69	6/14	0.55-8.3	1.10E+01			4.56E+01	C	3.47E+01	ca	3.47E+01 N BSL
	86-74-8	CARBAZOLE	0.12 J	14 J	mg/kg	HB-HBSED-12	10/14	0.49-4	1.40E+01			3.19E+01	C	2.43E+01	ca	2.43E+01 N BSL
	218-01-9	CHRYSENE	0.1 J	2.8 J	mg/kg	HB-DR-69	11/14	1.2-2.2	2.80E+00			2.20E+01	C	6.21E+01	ca	2.20E+01 N BSL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.068 J	0.096 J	mg/kg	HB-HBSED-12	2/14	0.76-11	9.60E-02			2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL
	132-64-9	DIBENZOFURAN	0.15 J	13 J	mg/kg	HB-HBSED-12	6/14	1.2-11	1.30E+01			7.82E+00	N	1.45E+01	nc	7.82E+00 Y ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.5 J	1.1 J	mg/kg	HB-DR-69	2/14	0.55-11	1.10E+00			7.82E+02	N	6.11E+02	nc	6.11E+02 N BSL
	117-84-0	DI-N-OCTYL PHTHALATE	0.088 J	0.088 J	mg/kg	HB-HBSED-12	1/14	0.55-11	8.80E-02			NV	N	2.44E+02	nc	2.44E+02 N BSL
	206-44-0	FLUORANTHENE	0.17 J	9.1 J	mg/kg	HB-HBSED-12	11/14	1.3-4.2	9.10E+00			3.13E+02	N	2.29E+02	nc	2.29E+02 N BSL
	86-73-7	FLUORENE	0.2 J	13 J	mg/kg	HB-HBSED-12	7/14	1.2-4.9	1.30E+01			3.13E+02	N	2.75E+02	nc	2.75E+02 N BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.17 J	1.5 J	mg/kg	HB-DR-69	5/14	0.76-11	1.50E+00			2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	91-20-3	NAPHTHALENE	0.21	150 J	mg/kg	HB-HBSED-12	10/17	1.2-4.9	1.50E+02			1.56E+02	N	5.59E+00	nc	5.59E+00 Y ASL

TABLE 2.23a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - I-690 STORM SEWER AND DRAINAGE DITCH SURFACE SEDIMENT
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment (0-1 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
	85-01-8	PHENANTHRENE	0.57 J	18 J	mg/kg	HB-HBSED-12	11/14	1.2-2.6	1.80E+01			NV	NV	NV	Y	NTX		
	108-95-2	PHENOL	0.47 J	2.4 J	mg/kg	HB-HBSED-12	6/14	0.55-4.9	2.40E+00			2.35E+03	N	1.83E+03	N	BSL		
	129-00-0	PYRENE	0.18 J	6.8 J	mg/kg	HB-HBSED-12	11/14	1.2-3.3	6.80E+00			2.35E+02	N	2.32E+02	N	BSL		
	VOCs																	
	78-93-3	2-BUTANONE	0.0059 J	0.06 J	mg/kg	HB-DR-69	5/14	0.037-11	6.00E-02			4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	108-10-1	4-METHYL-2-PENTANONE	0.0019 J	0.018 J	mg/kg	HB-DR-69	3/14	0.012-5.7	1.80E-02			NV		5.28E+02	nc	5.28E+02	N	BSL
	67-64-1	ACETONE	0.018 J	0.16 J	mg/kg	HB-HBSED-11	11/14	5.5-11	1.60E-01			7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.0015 J	2 J	mg/kg	HB-HBSED-11	9/14	0.0061-0.02	2.00E+00			1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.0017 J	0.0067 J	mg/kg	HB-HBSED-13	3/11	0.0064-2.9	6.70E-03			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL
	124-48-1	CHLORODIBROMOMETHANE	0.0075	0.0075	mg/kg	HB-DR-69	1/14	0.0061-2.8	7.50E-03			7.60E+00	C	1.11E+00	ca	1.11E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.0045 J	1.4 J	mg/kg	HB-HBSED-12	9/14	0.0061-0.02	1.40E+00			7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.029 J	0.029 J	mg/kg	HB-HBSED-11	1/3	0.011-2.8	2.90E-02			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.0075	0.0075	mg/kg	HB-DR-69	1/14	0.0061-5.7	7.50E-03			8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL
	103-65-1	N-PROPYLBENZENE	0.024 J	0.024 J	mg/kg	HB-HBSED-11	1/3	0.011-2.8	2.40E-02			NV		2.40E+01	nc	2.40E+01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.004 J	0.017 J	mg/kg	HB-HBSED-11	2/3	2.8-2.8	1.70E-02			NV		NV		NV	Y	NTX
	100-42-5	STYRENE	0.0017 J	2.5 J	mg/kg	HB-HBSED-12	11/14	0.019-2.4	2.50E+00			1.56E+03	N	1.70E+02	nc	1.70E+02	N	BSL
	108-88-3	TOLUENE	0.0071 J	5.5 J	mg/kg	HB-HBSED-12	10/14	0.0061-0.019	5.50E+00			6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	0.0086 J	17.6 J	mg/kg	HB-HBSED-12	10/14	0.0061-0.019	1.76E+01			1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value.
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(8) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.23b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
f = RBC and PRG values for 4-methylphenol (CAA# 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.23b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-DR-69	6/5/2003	0	0	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	19.613	19.613	ng/kg	EMPC	0.01	0.196
HB-DR-69	6/5/2003	0	0	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.709	0.709	ng/kg		0.01	0.007
HB-DR-69	6/5/2003	0	0	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.636	0.636	ng/kg		0.1	0.064
HB-DR-69	6/5/2003	0	0	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.942	2.942	ng/kg		0.1	0.294
HB-DR-69	6/5/2003	0	0	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.676	3.676	ng/kg	J	0.1	0.368
HB-DR-69	6/5/2003	0	0	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.612	1.612	ng/kg		0.1	0.161
HB-DR-69	6/5/2003	0	0	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.499	2.499	ng/kg		0.1	0.250
HB-DR-69	6/5/2003	0	0	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg		0.1	0.125
HB-DR-69	6/5/2003	0	0	40321-76-4	1,2,3,7,8-PECDD	Y	1.003	1.003	ng/kg	J	1	1.003
HB-DR-69	6/5/2003	0	0	57117-41-6	1,2,3,7,8-PECDF	Y	1.275	1.275	ng/kg	J	0.03	0.038
HB-DR-69	6/5/2003	0	0	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-DR-69	6/5/2003	0	0	51207-31-9	2,3,7,8-TCDF	Y	2.565	2.565	ng/kg		0.1	0.257
HB-DR-69	6/5/2003	0	0	3268-87-9	OCDD	Y	803.457	803.457	ng/kg	J	0.0003	0.241
HB-DR-69	6/5/2003	0	0	39001-02-0	OCDF	Y	37.661	37.661	ng/kg	J	0.0003	0.011
Sample Location TEQ = 3.5												
HB-DR-69	9/11/2003	0	0	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	4.043	4.043	ng/kg		0.01	0.040
HB-DR-69	9/11/2003	0	0	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	U	0.01	0.013
HB-DR-69	9/11/2003	0	0	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-69	9/11/2003	0	0	70648-26-9	1,2,3,4,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-69	9/11/2003	0	0	57653-85-7	1,2,3,6,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-69	9/11/2003	0	0	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-69	9/11/2003	0	0	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-69	9/11/2003	0	0	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-69	9/11/2003	0	0	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-DR-69	9/11/2003	0	0	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	U	0.03	0.038
HB-DR-69	9/11/2003	0	0	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-DR-69	9/11/2003	0	0	51207-31-9	2,3,7,8-TCDF	N	1	0.5	ng/kg	U	0.1	0.050
HB-DR-69	9/11/2003	0	0	3268-87-9	OCDD	Y	203.944	203.944	ng/kg	J	0.0003	0.061
HB-DR-69	9/11/2003	0	0	39001-02-0	OCDF	Y	7.485	7.485	ng/kg	J	0.0003	0.002
Sample Location TEQ = 2.7												

TABLE 2.23b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-DR-70	6/5/2003	0	0	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	130.936	130.936	ng/kg		0.01	1.309
HB-DR-70	6/5/2003	0	0	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	10.41	10.41	ng/kg		0.01	0.104
HB-DR-70	6/5/2003	0	0	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.644	1.644	ng/kg	J	0.1	0.164
HB-DR-70	6/5/2003	0	0	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.32	3.32	ng/kg		0.1	0.332
HB-DR-70	6/5/2003	0	0	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.115	7.115	ng/kg		0.1	0.712
HB-DR-70	6/5/2003	0	0	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.029	2.029	ng/kg	J	0.1	0.203
HB-DR-70	6/5/2003	0	0	19408-74-3	1,2,3,7,8,9-HXCDD	Y	4.48	4.48	ng/kg		0.1	0.448
HB-DR-70	6/5/2003	0	0	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.931	0.931	ng/kg	J	0.1	0.093
HB-DR-70	6/5/2003	0	0	40321-76-4	1,2,3,7,8-PECDD	Y	0.848	0.848	ng/kg	J	1	0.848
HB-DR-70	6/5/2003	0	0	57117-41-6	1,2,3,7,8-PECDF	Y	0.665	0.665	ng/kg	J	0.03	0.020
HB-DR-70	6/5/2003	0	0	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-DR-70	6/5/2003	0	0	51207-31-9	2,3,7,8-TCDF	Y	0.845	0.845	ng/kg	J	0.1	0.085
HB-DR-70	6/5/2003	0	0	3268-87-9	OCDD	Y	7563.653	7563.653	ng/kg	J	0.0003	2.269
HB-DR-70	6/5/2003	0	0	39001-02-0	OCDF	Y	1208.058	1208.058	ng/kg	J	0.0003	0.362
Sample Location TEQ = 7.4												
HB-DR-70	9/11/2003	0	0	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	4.588	4.588	ng/kg		0.01	0.046
HB-DR-70	9/11/2003	0	0	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	U	0.01	0.013
HB-DR-70	9/11/2003	0	0	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-70	9/11/2003	0	0	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.938	0.938	ng/kg	J	0.1	0.094
HB-DR-70	9/11/2003	0	0	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.479	1.479	ng/kg	EMPC	0.1	0.148
HB-DR-70	9/11/2003	0	0	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-70	9/11/2003	0	0	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.312	1.312	ng/kg	J	0.1	0.131
HB-DR-70	9/11/2003	0	0	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-DR-70	9/11/2003	0	0	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-DR-70	9/11/2003	0	0	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	U	0.03	0.038
HB-DR-70	9/11/2003	0	0	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-DR-70	9/11/2003	0	0	51207-31-9	2,3,7,8-TCDF	N	1	0.5	ng/kg	U	0.1	0.050
HB-DR-70	9/11/2003	0	0	3268-87-9	OCDD	Y	219.561	219.561	ng/kg	J	0.0003	0.066
HB-DR-70	9/11/2003	0	0	39001-02-0	OCDF	Y	6.72	6.72	ng/kg	J	0.0003	0.002
Sample Location TEQ = 2.7												

TABLE 2.23b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-DR-72	6/5/2003	0	0	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	20.782	20.782	ng/kg		0.01	0.208
HB-DR-72	6/5/2003	0	0	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	U	0.01	0.013
HB-DR-72	6/5/2003	0	0	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.78	1.78	ng/kg	J	0.1	0.178
HB-DR-72	6/5/2003	0	0	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.979	2.979	ng/kg		0.1	0.298
HB-DR-72	6/5/2003	0	0	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.219	5.219	ng/kg		0.1	0.522
HB-DR-72	6/5/2003	0	0	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.824	1.824	ng/kg	J	0.1	0.182
HB-DR-72	6/5/2003	0	0	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.048	5.048	ng/kg		0.1	0.505
HB-DR-72	6/5/2003	0	0	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.521	0.521	ng/kg	EMPC	0.1	0.052
HB-DR-72	6/5/2003	0	0	40321-76-4	1,2,3,7,8-PECDD	Y	1.234	1.234	ng/kg	J	1	1.234
HB-DR-72	6/5/2003	0	0	57117-41-6	1,2,3,7,8-PECDF	Y	0.812	0.812	ng/kg	J	0.03	0.024
HB-DR-72	6/5/2003	0	0	1746-01-6	2,3,7,8-TCDD	Y	0.275	0.275	ng/kg	EMPC	1	0.275
HB-DR-72	6/5/2003	0	0	51207-31-9	2,3,7,8-TCDF	N	1	0.5	ng/kg	U	0.1	0.050
HB-DR-72	6/5/2003	0	0	3268-87-9	OCDD	Y	570.143	570.143	ng/kg		0.0003	0.171
HB-DR-72	6/5/2003	0	0	39001-02-0	OCDF	Y	42.936	42.936	ng/kg		0.0003	0.013
Sample Location TEQ =												3.7
HB-HBSED-11	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	34.4	34.4	ng/kg	J	0.01	0.344
HB-HBSED-11	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	5	2.5	ng/kg	UJ	0.01	0.025
HB-HBSED-11	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-11	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-11	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-11	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-11	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-11	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-11	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	5	2.5	ng/kg	UJ	1	2.500
HB-HBSED-11	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	5	2.5	ng/kg	UJ	0.03	0.075
HB-HBSED-11	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-HBSED-11	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	4.39	4.39	ng/kg	J	0.1	0.439
HB-HBSED-11	5/11/2001	0	0.5	3268-87-9	OCDD	Y	982	982	ng/kg	J	0.0003	0.295
HB-HBSED-11	5/11/2001	0	0.5	39001-02-0	OCDF	Y	101	101	ng/kg	J	0.0003	0.030
Sample Location TEQ =												5.7

TABLE 2.23b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-11	6/3/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.977	8.977	ng/kg	J	0.01	0.090
HB-HBSED-11	6/3/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.527	0.2635	ng/kg	UJ	0.01	0.003
HB-HBSED-11	6/3/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.398	0.199	ng/kg	UJ	0.1	0.020
HB-HBSED-11	6/3/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.802	0.802	ng/kg	J	0.1	0.080
HB-HBSED-11	6/3/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.216	1.216	ng/kg	J	0.1	0.122
HB-HBSED-11	6/3/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.296	0.148	ng/kg	UJ	0.1	0.015
HB-HBSED-11	6/3/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	0.668	0.668	ng/kg	EMPC	0.1	0.067
HB-HBSED-11	6/3/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.296	0.148	ng/kg	UJ	0.1	0.015
HB-HBSED-11	6/3/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.252	0.126	ng/kg	UJ	1	0.126
HB-HBSED-11	6/3/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.338	0.169	ng/kg	UJ	0.03	0.005
HB-HBSED-11	6/3/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.345	0.1725	ng/kg	UJ	1	0.173
HB-HBSED-11	6/3/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.972	0.972	ng/kg	J	0.1	0.097
HB-HBSED-11	6/3/2003	0	0.5	3268-87-9	OCDD	Y	177.8	177.8	ng/kg	J	0.0003	0.053
HB-HBSED-11	6/3/2003	0	0.5	39001-02-0	OCDF	Y	17.484	17.484	ng/kg	J	0.0003	0.005
Sample Location TEQ =												0.9
HB-HBSED-11	6/3/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	27.995	27.995	ng/kg	J	0.01	0.280
HB-HBSED-11	6/3/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.003	1.003	ng/kg	J	0.01	0.010
HB-HBSED-11	6/3/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.98	3.98	ng/kg	J	0.1	0.398
HB-HBSED-11	6/3/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	17.568	17.568	ng/kg	J	0.1	1.757
HB-HBSED-11	6/3/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	25.854	25.854	ng/kg	J	0.1	2.585
HB-HBSED-11	6/3/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.643	0.3215	ng/kg	UJ	0.1	0.032
HB-HBSED-11	6/3/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	10.56	10.56	ng/kg	J	1	10.560
HB-HBSED-11	6/3/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	0.661	0.661	ng/kg	J	0.03	0.020
HB-HBSED-11	6/3/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	2.955	2.955	ng/kg	J	1	2.955
HB-HBSED-11	6/3/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	2.11	2.11	ng/kg	J	0.1	0.211
HB-HBSED-11	6/3/2003	0.5	1	3268-87-9	OCDD	Y	3163.388	3163.388	ng/kg	J	0.0003	0.949
HB-HBSED-11	6/3/2003	0.5	1	39001-02-0	OCDF	Y	64.704	64.704	ng/kg	J	0.0003	0.019
Sample Location TEQ =												19.8

TABLE 2.23b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-12	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	36.1	36.1	ng/kg	J	0.01	0.361
HB-HBSED-12	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	5	2.5	ng/kg	UJ	0.01	0.025
HB-HBSED-12	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-12	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-12	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.15	7.15	ng/kg	J	0.1	0.715
HB-HBSED-12	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-12	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.45	6.45	ng/kg	J	0.1	0.645
HB-HBSED-12	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-12	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	5	2.5	ng/kg	UJ	1	2.500
HB-HBSED-12	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	5	2.5	ng/kg	UJ	0.03	0.075
HB-HBSED-12	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-HBSED-12	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	5.11	5.11	ng/kg	J	0.1	0.511
HB-HBSED-12	5/11/2001	0	0.5	3268-87-9	OCDD	Y	1580	1580	ng/kg	J	0.0003	0.474
Sample Location TEQ =												6.8
HB-HBSED-12	6/3/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	7.002	7.002	ng/kg	J	0.01	0.070
HB-HBSED-12	6/3/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.869	0.4345	ng/kg	UJ	0.01	0.004
HB-HBSED-12	6/3/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.657	0.3285	ng/kg	UJ	0.1	0.033
HB-HBSED-12	6/3/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.003	1.003	ng/kg	J	0.1	0.100
HB-HBSED-12	6/3/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.505	0.2525	ng/kg	UJ	0.1	0.025
HB-HBSED-12	6/3/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.692	0.346	ng/kg	UJ	0.1	0.035
HB-HBSED-12	6/3/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.516	0.258	ng/kg	UJ	1	0.258
HB-HBSED-12	6/3/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.274	0.137	ng/kg	UJ	0.03	0.004
HB-HBSED-12	6/3/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.422	0.211	ng/kg	UJ	1	0.211
HB-HBSED-12	6/3/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.38	1.38	ng/kg	J	0.1	0.138
HB-HBSED-12	6/3/2003	0	0.5	3268-87-9	OCDD	Y	243.797	243.797	ng/kg	J	0.0003	0.073
HB-HBSED-12	6/3/2003	0	0.5	39001-02-0	OCDF	Y	13.736	13.736	ng/kg	J	0.0003	0.004
Sample Location TEQ =												1.0

TABLE 2.23b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-12	6/3/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	82.489	82.489	ng/kg	EMPC	0.01	0.825
HB-HBSED-12	6/3/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.682	4.682	ng/kg		0.01	0.047
HB-HBSED-12	6/3/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	1.994	1.994	ng/kg		0.1	0.199
HB-HBSED-12	6/3/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	18.479	18.479	ng/kg		0.1	1.848
HB-HBSED-12	6/3/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	14.53	14.53	ng/kg		0.1	1.453
HB-HBSED-12	6/3/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	6.371	6.371	ng/kg		0.1	0.637
HB-HBSED-12	6/3/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	8.598	8.598	ng/kg	U	0.1	0.860
HB-HBSED-12	6/3/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.514	0.757	ng/kg		0.1	0.076
HB-HBSED-12	6/3/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	1.42	1.42	ng/kg	J	1	1.420
HB-HBSED-12	6/3/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	5.346	5.346	ng/kg	U	0.03	0.160
HB-HBSED-12	6/3/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.406	0.203	ng/kg		1	0.203
HB-HBSED-12	6/3/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	5.89	5.89	ng/kg	J	0.1	0.589
HB-HBSED-12	6/3/2003	0.5	1	3268-87-9	OCDD	Y	3536.853	3536.853	ng/kg		0.0003	1.061
HB-HBSED-12	6/3/2003	0.5	1	39001-02-0	OCDF	Y	93.16	93.16	ng/kg		0.0003	0.028
Sample Location TEQ = 9.4												
HB-HBSED-13	5/11/2001	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	38.2	38.2	ng/kg	J	0.01	0.382
HB-HBSED-13	5/11/2001	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	5	2.5	ng/kg	UJ	0.01	0.025
HB-HBSED-13	5/11/2001	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-13	5/11/2001	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	5.44	5.44	ng/kg	J	0.1	0.544
HB-HBSED-13	5/11/2001	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	12.1	12.1	ng/kg	J	0.1	1.210
HB-HBSED-13	5/11/2001	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-13	5/11/2001	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	12.1	12.1	ng/kg	J	0.1	1.210
HB-HBSED-13	5/11/2001	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	5	2.5	ng/kg	UJ	0.1	0.250
HB-HBSED-13	5/11/2001	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	5	2.5	ng/kg	UJ	1	2.500
HB-HBSED-13	5/11/2001	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	5	2.5	ng/kg	UJ	0.03	0.075
HB-HBSED-13	5/11/2001	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.38	1.38	ng/kg	J	1	1.380
HB-HBSED-13	5/11/2001	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	8.34	8.34	ng/kg	J	0.1	0.834
HB-HBSED-13	5/11/2001	0	0.5	3268-87-9	OCDD	Y	713	713	ng/kg	J	0.0003	0.214
HB-HBSED-13	5/11/2001	0	0.5	39001-02-0	OCDF	Y	91.8	91.8	ng/kg	J	0.0003	0.028
Sample Location TEQ = 9.2												

TABLE 2.23b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-13	6/2/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	N	0.429	0.2145	ng/kg	UJ	0.01	0.002
HB-HBSED-13	6/2/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.631	0.3155	ng/kg	UJ	0.01	0.003
HB-HBSED-13	6/2/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.246	0.123	ng/kg	UJ	0.1	0.012
HB-HBSED-13	6/2/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	0.302	0.151	ng/kg	UJ	0.1	0.015
HB-HBSED-13	6/2/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.23	0.115	ng/kg	UJ	0.1	0.012
HB-HBSED-13	6/2/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.429	0.2145	ng/kg	UJ	0.1	0.021
HB-HBSED-13	6/2/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.236	0.118	ng/kg	UJ	0.1	0.012
HB-HBSED-13	6/2/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.385	0.1925	ng/kg	UJ	0.1	0.019
HB-HBSED-13	6/2/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.322	0.161	ng/kg	UJ	1	0.161
HB-HBSED-13	6/2/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.29	0.145	ng/kg	UJ	0.03	0.004
HB-HBSED-13	6/2/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.267	0.1335	ng/kg	UJ	1	0.134
HB-HBSED-13	6/2/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	N	0.229	0.1145	ng/kg	UJ	0.1	0.011
HB-HBSED-13	6/2/2003	0	0.5	39001-02-0	OCDF	N	1.36	0.68	ng/kg	UJ	0.0003	0.000
Sample Location TEQ =												0.4
HB-HBSED-13	6/3/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	N	0.215	0.1075	ng/kg	UJ	0.01	0.001
HB-HBSED-13	6/3/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.317	0.1585	ng/kg	UJ	0.01	0.002
HB-HBSED-13	6/3/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.225	0.1125	ng/kg	UJ	0.1	0.011
HB-HBSED-13	6/3/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	N	0.21	0.105	ng/kg	UJ	0.1	0.011
HB-HBSED-13	6/3/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.169	0.0845	ng/kg	UJ	0.1	0.008
HB-HBSED-13	6/3/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.216	0.108	ng/kg	UJ	0.1	0.011
HB-HBSED-13	6/3/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.21	0.105	ng/kg	UJ	0.1	0.011
HB-HBSED-13	6/3/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	0.175	0.0875	ng/kg	UJ	1	0.088
HB-HBSED-13	6/3/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	0.156	0.078	ng/kg	UJ	0.03	0.002
HB-HBSED-13	6/3/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.149	0.0745	ng/kg	UJ	1	0.075
HB-HBSED-13	6/3/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	N	0.175	0.0875	ng/kg	UJ	0.1	0.009
Sample Location TEQ =												0.2

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

EMPC = Estimated Maximum Possible Concentration

N/A = not applicable, J = estimated value, U = not detected, EMPC = estimated maximum possible concentration

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.23c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - INTERSTATE 690 DRAINAGE DITCH SURFACE SEDIMENT

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-HBSED-11	0	0.5	5/8/2001	0.09	mg/kg
Highly Chlorinated PCBs	HB-HBSED-11	0.5	1	6/2/2003	0.56	mg/kg
Highly Chlorinated PCBs	HB-HBSED-12	0	0.5	5/8/2001	0.05	mg/kg
Highly Chlorinated PCBs	HB-HBSED-13	0	0.5	5/8/2001	0.07	mg/kg
Total PCBs	HB-HBSED-11	0	0.5	5/8/2001	0.09	mg/kg
Total PCBs	HB-HBSED-11	0.5	1	6/2/2003	0.56	mg/kg
Total PCBs	HB-HBSED-12	0	0.5	5/8/2001	0.05	mg/kg
Total PCBs	HB-HBSED-13	0	0.5	5/8/2001	0.07	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

· (0-1 ft)

TABLE 2.23d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - I-690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-DR-69	6/5/2003	0	0	57-74-9	CHLORDANE	N	U	mg/kg	0.013
HB-DR-69	6/5/2003	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.013
Total Chlordane =									ND
HB-DR-69	9/11/2003	0	0	57-74-9	CHLORDANE	N	U	mg/kg	0.024
HB-DR-69	9/11/2003	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.024
Total Chlordane =									ND
HB-DR-70	6/5/2003	0	0	57-74-9	CHLORDANE	N	U	mg/kg	0.011
HB-DR-70	6/5/2003	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.011
Total Chlordane =									ND
HB-DR-70	9/11/2003	0	0	57-74-9	CHLORDANE	N	U	mg/kg	0.021
HB-DR-70	9/11/2003	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.021
Total Chlordane =									ND
HB-DR-72	6/5/2003	0	0	57-74-9	CHLORDANE	N	U	mg/kg	0.011
HB-DR-72	6/5/2003	0	0	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.011
Total Chlordane =									ND
HB-HBSED-11	5/8/2001	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.004
HB-HBSED-11	5/8/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.004
Total Chlordane =									ND
HB-HBSED-11	6/2/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.022
HB-HBSED-11	6/2/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.022
Total Chlordane =									ND
HB-HBSED-11	6/2/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.019
HB-HBSED-11	6/2/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.019
Total Chlordane =									ND
HB-HBSED-12	5/8/2001	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.004
HB-HBSED-12	5/8/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.005
Total Chlordane =									0.005
HB-HBSED-12	6/2/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.019
HB-HBSED-12	6/2/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.019
Total Chlordane =									ND
HB-HBSED-12	6/2/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.019
HB-HBSED-12	6/2/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.019
Total Chlordane =									ND
HB-HBSED-13	5/8/2001	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.003
HB-HBSED-13	5/8/2001	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.004
Total Chlordane =									0.004
HB-HBSED-13	6/2/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.032
HB-HBSED-13	6/2/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.032
Total Chlordane =									ND
HB-HBSED-13	6/2/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.033
HB-HBSED-13	6/2/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.033
Total Chlordane =									ND

TABLE 2.23e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - I-690 DRAINAGE DITCH SURFACE SEDIMENT (0-1 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-DR-69	6/5/2003	0	0	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.011	
HB-DR-69	6/5/2003	0	0	95-47-6	O-XYLENE	Y	J	mg/kg	0.0051	
HB-DR-69	6/5/2003	0	0	CALCULATED	TOTAL	Y	J	mg/kg		0.0161
HB-DR-69	9/11/2003	0	0	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0061	
HB-DR-69	9/11/2003	0	0	95-47-6	O-XYLENE	Y	J	mg/kg	0.004	
HB-DR-69	9/11/2003	0	0	CALCULATED	TOTAL	Y	J	mg/kg		0.0101
HB-DR-70	6/5/2003	0	0	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.0064	
HB-DR-70	6/5/2003	0	0	95-47-6	O-XYLENE	N	UJ	mg/kg	0.0064	
HB-DR-70	6/5/2003	0	0	CALCULATED	TOTAL	N	UJ	mg/kg		0.0064
HB-DR-70	9/11/2003	0	0	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0061	
HB-DR-70	9/11/2003	0	0	95-47-6	O-XYLENE	N	U	mg/kg	0.0061	
HB-DR-70	9/11/2003	0	0	CALCULATED	TOTAL	N	U	mg/kg		0.0061
HB-DR-72	6/5/2003	0	0	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0063	
HB-DR-72	6/5/2003	0	0	95-47-6	O-XYLENE	N		mg/kg	0.0063	
HB-DR-72	6/5/2003	0	0	CALCULATED	TOTAL	N		mg/kg		0.0063
HB-HBSED-11	5/8/2001	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	4.5	4.5
HB-HBSED-11	6/2/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	5.6	
HB-HBSED-11	6/2/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	1.9	
HB-HBSED-11	6/2/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		7.5
HB-HBSED-11	6/2/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	4	
HB-HBSED-11	6/2/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	1.7	
HB-HBSED-11	6/2/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		5.7
HB-HBSED-12	5/8/2001	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	9.1	9.1
HB-HBSED-12	6/2/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	11	
HB-HBSED-12	6/2/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	3.5	
HB-HBSED-12	6/2/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		14.5
HB-HBSED-12	6/2/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	13	
HB-HBSED-12	6/2/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	4.6	
HB-HBSED-12	6/2/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		17.6
HB-HBSED-13	5/8/2001	0	0.5	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.12	0.12
HB-HBSED-13	6/2/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.019	
HB-HBSED-13	6/2/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.019	
HB-HBSED-13	6/2/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.019
HB-HBSED-13	6/2/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0043	
HB-HBSED-13	6/2/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.0043	
HB-HBSED-13	6/2/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0086

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.24a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - I-690 DRAINAGE SEWER AND DITCH SURFACE WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
I-690 Drainage Sewer and Ditch Surface	METALS																		
	7429-90-5	ALUMINUM	0.11 J	1.3 J	mg/L	HB-DR-69	3/7	0.0532-0.1	1.30E+00		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	N	BSL	
	7440-39-3	BARIUM	0.0303	0.0966 J	mg/L	HB-HBSW-12	7/7	-	9.66E-02		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	N	BSL	
	7440-41-7	BERYLLIUM	0.0001 J	0.0001 J	mg/L	HB-HBSW-12	1/7	0.000076-0.005	1.00E-04		4.00E-03	7.30E-03	N	7.30E-03	nc	7.30E-03	N	BSL	
	7440-70-2	CALCIUM	25.8	542	mg/L	HB-HBSW-12	7/7	-	5.42E+02			NV	NV			NV	N	NUT	
	7440-47-3	CHROMIUM ^a	0.007 J	0.0155	mg/L	HB-DR-69	4/7	0.01-0.01	1.55E-02		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX	
	7440-50-8	COPPER	0.0072 J	0.0309	mg/L	HB-DR-69	4/7	0.02-0.02	3.09E-02		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	N	BSL	
	57-12-5	CYANIDE	0.044	0.065	mg/L	HB-HBSW-12	2/7	0.01-0.01	6.50E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL	
	7439-89-6	IRON	0.0112 J	3.13 J	mg/L	HB-DR-69	4/7	0.1-0.1	3.13E+00		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL	
	7439-92-1	LEAD	0.0044	0.0256	mg/L	HB-DR-72	5/7	0.005-0.005	2.56E-02		1.50E-02	NV	NV			1.50E-02	Y	ASL	
	7439-95-4	MAGNESIUM	0.227 J	3.85	mg/L	HB-HBSW-11	7/7	-	3.85E+00			NV	NV			NV	N	NUT	
	7439-96-5	MANGANESE	0.0655	0.152	mg/L	HB-DR-69	2/7	0.0014-0.01	1.52E-01		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	Y	ASL	
	7439-97-6	MERCURY ^b	0.000025	0.00048	mg/L	HB-HBSW-12	4/10	0.00018 - 0.0002	4.80E-04		2.00E-03	3.65E-04	N	3.65E-04	nc	3.65E-04	Y	ASL	
	7440-02-0	NICKEL	0.002 J	0.0024 J	mg/L	HB-HBSW-12	2/7	0.04-0.04	2.40E-03			7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL	
	7440-09-7	POTASSIUM	2.01	16.7	mg/L	HB-HBSW-12	6/7	2-2	1.67E+01			NV	NV			NV	N	NUT	
	7782-49-2	SELENIUM	0.0031 J	0.0031 J	mg/L	HB-HBSW-12	1/7	0.0018-0.01	3.10E-03		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL	
	7440-23-5	SODIUM	37.7	662	mg/L	HB-HBSW-11	7/7	-	6.62E+02			NV	NV			NV	N	NUT	
	7440-62-2	VANADIUM	0.0023 J	0.0037 J	mg/L	HB-HBSW-13	2/7	0.05-0.05	3.70E-03			3.65E-03	N	3.65E-03	nc	3.65E-03	Y	ASL	
	7440-66-6	ZINC	0.679	1.41	mg/L	HB-DR-72	2/7	0.0029-0.02	1.41E+00		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	Y	ASL	
	PESTICIDES																		
	72-54-8	4,4'-DDD		0.21	0.21	ug/L	HB-HBSW-13	1/7	0.1-0.2	2.10E-01			2.79E-01	C	2.80E-01	ca	2.79E-01	N	BSL
	SVOCs																		
	105-67-9	2,4-DIMETHYLPHENOL		2.5 J	11 J	ug/L	HB-HBSW-12	3/7	9.3-220	1.10E+01			7.30E+01	N	7.30E+01	nc	7.30E+01	N	BSL
	91-57-6	2-METHYLNAPHTHALENE		19 J	160 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	1.60E+02			NV	NV			NV	Y	NTX
	95-48-7	2-METHYLPHENOL		8.9 J	97 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	9.70E+01			1.83E+02	N	1.82E+02	nc	1.82E+02	N	BSL
	34METPH	3&4-METHYLPHENOL ^c		7 J	210 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	2.10E+02			1.83E+01	N	1.82E+01	nc	1.82E+01	Y	ASL
	83-32-9	ACENAPHTHENE		3.4 J	25 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	2.50E+01			3.65E+01	N	3.65E+01	nc	3.65E+01	N	BSL
	208-96-8	ACENAPHTHYLENE		2.2 J	11 J	ug/L	HB-HBSW-12	4/7	9.3-220	1.10E+01			NV	NV			NV	Y	NTX
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE		2.8 J	3 J	ug/L	HB-DR-69	2/7	20-220	3.00E+00		6.00E+00	4.78E+00	C	4.80E+00	ca	4.78E+00	N	BSL
	86-74-8	CARBAZOLE		5 J	30 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	3.00E+01			3.35E+00	C	3.36E+00	ca	3.35E+00	Y	ASL
	132-64-9	DIBENZOFURAN		4.1 J	35 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	3.50E+01			3.65E+00	N	1.22E+00	nc	1.22E+00	Y	ASL
	206-44-0	FLUORANTHENE		1 J	1 J	ug/L	HB-DR-72	1/7	9.4-220	1.00E+00			1.46E+02	N	1.46E+02	nc	1.46E+02	N	BSL
	86-73-7	FLUORENE		2.2 J	27 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	2.70E+01			2.43E+01	N	2.43E+01	nc	2.43E+01	Y	ASL
	91-20-3	NAPHTHALENE		160	1400	ug/L	HB-HBSW-12	6/8	9.3-9.4	1.40E+03			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	85-01-8	PHENANTHRENE		2.5 J	27 J	ug/L	HB-HBSW-12	5/7	9.3-9.4	2.70E+01			NV	NV			NV	Y	NTX
	108-95-2	PHENOL		17 J	700	ug/L	HB-HBSW-12	5/7	9.3-9.4	7.00E+02			1.10E+03	N	1.09E+03	nc	1.09E+03	N	BSL
	VOCs																		
	95-63-6	1,2,4-TRIMETHYLBENZENE		16	67	ug/L	HB-HBSW-12	2/2	-	6.70E+01			1.46E+00	N	1.23E+00	nc	1.23E+00	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE		6 J	26	ug/L	HB-HBSW-12	2/2	-	2.60E+01			NV	NV			1.23E+00	Y	ASL
	78-93-3	2-BUTANONE		1 J	2 J	ug/L	HB-HBSW-12	2/7	10-250	2.00E+00			6.97E+02	N	6.97E+02	nc	6.97E+02	N	BSL
	67-64-1	ACETONE		6.6 J	17 J	ug/L	HB-HBSW-12	5/7	250-250	1.70E+01			5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	71-43-2	BENZENE		9.6	130	ug/L	HB-HBSW-12	5/7	5-5	1.30E+02		5.00E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	100-41-4	ETHYLBENZENE		2.9 J	21	ug/L	HB-HBSW-12	5/7	5-5	2.10E+01			7.00E+02	N	1.34E+02	nc	1.34E+02	N	BSL
	100-42-5	STYRENE		3.1 J	22	ug/L	HB-HBSW-12	5/7	5-5	2.20E+01			1.62E+02	N	1.64E+02	nc	1.62E+02	N	BSL
	108-88-3	TOLUENE		28	270	ug/L	HB-HBSW-12	5/7	5-5	2.70E+02			1.00E+03	N	7.23E+01	nc	7.23E+01	Y	ASL
	1330-20-7	XYLENES, TOTAL		37	300	ug/L	HB-HBSW-12	5/7	5-5	3.00E+02		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) N/A - No background screening performed.
(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.
(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = RBC and PRG values for 4-methylphenol (CAS# 106445) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and not screened in
NV: No Value
PRG: Preliminary Remediation Goals
RBC: Risk Based Concentration
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.24b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - I-690 DRAINAGE SEWER AND DITCH SURFACE WATER

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-DR-69	6/5/2003	---	---	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-DR-69	6/5/2003	---	---	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-DR-69	6/5/2003	---	---	CALCULATED	TOTAL	N	U	ug/l		5
HB-DR-72	6/5/2003	---	---	XYLENES1314	XYLENES, M & P	N	U	ug/l	5	
HB-DR-72	6/5/2003	---	---	95-47-6	O-XYLENE	N	U	ug/l	5	
HB-DR-72	6/5/2003	---	---	CALCULATED	TOTAL	N	U	ug/l		5
HB-HBSW-11	6/2/2003	---	---	XYLENES1314	XYLENES, M & P	Y		ug/l	28	
HB-HBSW-11	6/2/2003	---	---	95-47-6	O-XYLENE	Y		ug/l	11	
HB-HBSW-11	6/2/2003	---	---	CALCULATED	TOTAL	Y		ug/l		39
HB-HBSW-12	5/8/2001	---	---	1330-20-7	XYLENES, TOTAL	Y		ug/l	300	300
HB-HBSW-12	6/2/2003	---	---	XYLENES1314	XYLENES, M & P	Y		ug/l	140	
HB-HBSW-12	6/2/2003	---	---	95-47-6	O-XYLENE	Y		ug/l	53	
HB-HBSW-12	6/2/2003	---	---	CALCULATED	TOTAL	Y		ug/l		193
HB-HBSW-13	5/8/2001	---	---	1330-20-7	XYLENES, TOTAL	Y		ug/l	77	77
HB-HBSW-13	6/2/2003	---	---	XYLENES1314	XYLENES, M & P	Y		ug/l	25	
HB-HBSW-13	6/2/2003	---	---	95-47-6	O-XYLENE	Y		ug/l	12	
HB-HBSW-13	6/2/2003	---	---	CALCULATED	TOTAL	Y		ug/l		37

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.25a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #1 SURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
DSA #1 Surface Soil	DIOXIN/FURAN (9)																		
	1746-01-6	2,3,7,8 TCDD Equivalent	0.00003	0.000036	mg/Kg	HB-SS-01	2/2		3.64E-05			4.26E-06	C	3.90E-06	ca	3.90E-06	Y	ASL	
	METALS																		
	7429-90-5	ALUMINUM	3240	3320	mg/Kg	HBSS01	2/2	-	3.32E+03			7.82E+03	N	7.61E+03	nc	7.61E+03	N	BSL	
	7440-38-2	ARSENIC	13.7 J	14.2 J	mg/Kg	HBSS01	2/2	-	1.42E+01		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX	
	7440-39-3	BARIUM	106	119	mg/Kg	HBSS01	2/2	-	1.19E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL	
	7440-43-9	CADMIUM	0.83	0.87	mg/Kg	HBSS01	2/2	-	8.70E-01		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	N	BSL	
	7440-70-2	CALCIUM	246000	279000	mg/Kg	HBSS01	2/2	-	2.79E+05			NV	NV	NV	NV	N	NUT		
	7440-47-3	CHROMIUM ^a	29.6	34.5	mg/Kg	HBSS01	2/2	-	3.45E+01			2.35E+01	N	3.01E+01	ca	2.35E+01	Y	TOX	
	7440-50-8	COPPER	229	240	mg/Kg	HBSS01	2/2	-	2.40E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL	
	7439-89-6	IRON	14400	14800	mg/Kg	HBSS01	2/2	-	1.48E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	107 J	109 J	mg/Kg	HBSS01	2/2	-	1.09E+02			NV	4.00E+02	nc	4.00E+02	N	BSL		
	7439-95-4	MAGNESIUM	4730	4790	mg/Kg	HBSS01	2/2	-	4.79E+03			NV	NV	NV	NV	N	NUT		
	7439-96-5	MANGANESE	295 J	302 J	mg/Kg	HBSS01	2/2	-	3.02E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL	
	7439-97-6	MERCURY ^b	1.5	9	mg/kg	HBDSA#1NWTP	4/4	-	9.00E+00			2.35E+00	N	2.35E+00	nc	2.35E+00	Y	ASL	
	22967-92-6	METHYL MERCURY	0.00323	0.00323	mg/kg	HBSS01	1/1	-	3.23E+00			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL	
	7440-02-0	NICKEL	25.3	26.9	mg/Kg	HBSS01	2/2	-	2.69E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL	
	7440-09-7	POTASSIUM	444	489	mg/Kg	HBSS01	2/2	-	4.89E+02			NV	NV	NV	NV	N	NUT		
	7782-49-2	SELENIUM	1.6 J	1.6 J	mg/Kg	HBSS01	1/2	0.74-0.74	1.60E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-23-5	SODIUM	938	1170	mg/Kg	HBSS01	2/2	-	1.17E+03			NV	NV	NV	NV	N	NUT		
	7440-62-2	VANADIUM	13.2	15.7	mg/Kg	HBSS01	2/2	-	1.57E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	122	133	mg/Kg	HBSS01	2/2	-	1.33E+02		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL	
	PCBs																		
		HIGHLY CHLORINATED PCBs ^c		0.18	2	mg/kg	HBDSA#1NETP	4/5	4-4	2.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
		TOTAL PCBs ^d		0.18	2	mg/kg	HBDSA#1NETP	4/5	4-4	2.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
	SVOCs																		
	56-55-3	BENZ(A)ANTHRACENE		0.34 J	0.36 J	mg/kg	HBSS01	2/2	-	3.60E-01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	50-32-8	BENZO(A)PYRENE		0.42 J	0.44 J	mg/kg	HBSS01	2/2	-	4.40E-01		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE		0.37 J	0.39 J	mg/kg	HBSS01	2/2	-	3.90E-01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE		0.3 J	0.32 J	mg/kg	HBSS01	2/2	-	3.20E-01		1.00E+02	NV	NV	NV	NV	Y	NTX	
	207-08-9	BENZO(K)FLUORANTHENE		0.33 J	0.39 J	mg/kg	HBSS01	2/2	-	3.90E-01		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00	N	BSL
	218-01-9	CHRYSENE		0.45 J	0.45 J	mg/kg	HBSS01	2/2	-	4.50E-01		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	N	BSL
	206-44-0	FLUORANTHENE		0.71 J	0.75 J	mg/kg	HBSS01	2/2	-	7.50E-01		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	118-74-1	HEXACHLOROBENZENE		0.04	0.83	mg/kg	HBDSA#1NWTP	2/5	0.0016-2.6	8.30E-01		3.30E-01	3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL
	193-39-5	INDENO(1,2,3CD)PYRENE		0.26 J	0.29 J	mg/kg	HBSS01	2/2	-	2.90E-01		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE		0.0021 J	0.35 J	mg/kg	HBDSA#1NWTP	2/5	0.0031-2.6	3.50E-01		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	N	BSL
	85-01-8	PHENANTHRENE		0.45 J	0.48 J	mg/kg	HBSS01	2/2	-	4.80E-01		1.00E+02	NV	NV	NV	NV	Y	NTX	
	129-00-0	PYRENE		0.55 J	0.55 J	mg/kg	HBSS01	2/2	-	5.50E-01		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	75-09-2	METHYLENE CHLORIDE		0.0017 J	0.0023 J	mg/kg	HBDSA#1NETP	2/5	0.0085-0.75	2.30E-03		5.10E+01	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL
	1330-20-7	XYLENES, TOTAL		0.12 J	0.12 J	mg/kg	HBDSA#1NWTP	1/4	0.00155-0.012	1.20E-01		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

TABLE 2.25a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #1 SURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
	VOCs															
	87-61-6	1,2,3TRICHLOROBENZENE	8.3	8.3	mg/kg	HBDSA#1NWTP	1/2	0.0031-0.0031	8.30E+00			NV	NV	NV	Y	NTX
	120-82-1	1,2,4TRICHLOROBENZENE	0.0022 J	35	mg/kg	HBDSA#1NWTP	2/5	0.0031-2.6	3.50E+01			NV	6.22E+00	nc	6.22E+00	Y
	95-63-6	1,2,4TRIMETHYLBENZENE	0.0019 J	0.0019 J	mg/kg	HBDSA#1SETP	1/3	0.0031-0.38	1.90E-03	4.70E+01	7.82E+01	N	5.16E+00	nc	5.16E+00	N
	95-50-1	1,2DICHLOROBENZENE	0.0076	22	mg/kg	HBDSA#1NWTP	5/5	-	2.20E+01	1.00E+02	7.04E+02	N	6.00E+02	na	6.00E+02	N
	541-73-1	1,3DICHLOROBENZENE	0.0026 J	0.7	mg/kg	HBDSA#1NWTP	2/5	0.0031-2.6	7.00E-01	1.70E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	N
	106-46-7	1,4DICHLOROBENZENE	0.0095 J	52	mg/kg	HBDSA#1NWTP	5/5	-	5.20E+01	9.80E+00	2.66E+01	C	3.45E+00	ca	3.45E+00	Y
	108-90-7	CHLOROBENZENE	0.0016 J	0.96	mg/kg	HBDSA#1NWTP	2/5	0.0031-0.012	9.60E-01	1.00E+02	1.56E+02	N	1.51E+01	nc	1.51E+01	N
	135-98-8	SEC BUTYLBENZENE	0.0022 J	0.0022 J	mg/kg	HBDSA#1SETP	1/3	0.0031-0.38	2.20E-03	1.00E+02	NV	2.20E+02	na	2.20E+02	N	BSL

Footnotes:

- (1) J = estimated value; N = tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
(5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC
(6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL = Above Screening Level; TOX = Class A Carcinogen; NTX = No Toxicity Information. Deletion Rationale: BSL = Below Screening Level
(9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.25b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for mercury compounds utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency
DSA #1: Dredge Soil Area #1

TABLE 2.25b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #1 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SS-01	12/3/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	125.298	125.298	ng/kg		0.01	1.253
HB-SS-01	12/3/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	36.846	36.846	ng/kg		0.01	0.368
HB-SS-01	12/3/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	3.836	3.836	ng/kg		0.01	0.038
HB-SS-01	12/3/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.961	3.961	ng/kg		0.1	0.396
HB-SS-01	12/3/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	36.599	36.599	ng/kg		0.1	3.660
HB-SS-01	12/3/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	9.264	9.264	ng/kg		0.1	0.926
HB-SS-01	12/3/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	18.422	18.422	ng/kg		0.1	1.842
HB-SS-01	12/3/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	8.404	8.404	ng/kg		0.1	0.840
HB-SS-01	12/3/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	4.007	4.007	ng/kg		0.1	0.401
HB-SS-01	12/3/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	5.518	5.518	ng/kg		1	5.518
HB-SS-01	12/3/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	51.548	51.548	ng/kg		0.03	1.546
HB-SS-01	12/3/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	3.992	3.992	ng/kg		1	3.992
HB-SS-01	12/3/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	153.9	153.9	ng/kg	J	0.1	15.390
HB-SS-01	12/3/2002	0	0.5	3268-87-9	OCDD	Y	795.567	795.567	ng/kg	J	0.0003	0.239
HB-SS-01	12/3/2002	0	0.5	39001-02-0	OCDF	Y	48.338	48.338	ng/kg	J	0.0003	0.015
Sample Location TEQ =												36.4
HB-SS-01	12/3/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	89.983	89.983	ng/kg		0.01	0.900
HB-SS-01	12/3/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	26.482	26.482	ng/kg		0.01	0.265
HB-SS-01	12/3/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.817	2.817	ng/kg		0.01	0.028
HB-SS-01	12/3/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.303	3.303	ng/kg		0.1	0.330
HB-SS-01	12/3/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	28.961	28.961	ng/kg		0.1	2.896
HB-SS-01	12/3/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.971	7.971	ng/kg		0.1	0.797
HB-SS-01	12/3/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	14.363	14.363	ng/kg		0.1	1.436
HB-SS-01	12/3/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.64	6.64	ng/kg		0.1	0.664
HB-SS-01	12/3/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	3.032	3.032	ng/kg		0.1	0.303
HB-SS-01	12/3/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	4.496	4.496	ng/kg		1	4.496
HB-SS-01	12/3/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	38.955	38.955	ng/kg		0.03	1.169
HB-SS-01	12/3/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	2.936	2.936	ng/kg		1	2.936
HB-SS-01	12/3/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	121.9	121.9	ng/kg	J	0.1	12.190
HB-SS-01	12/3/2002	0.5	1	3268-87-9	OCDD	Y	533.328	533.328	ng/kg	J	0.0003	0.160
HB-SS-01	12/3/2002	0.5	1	39001-02-0	OCDF	Y	34.961	34.961	ng/kg	J	0.0003	0.010
Sample Location TEQ =												28.6

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable, J = estimated value

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.25c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DSA #1 SURFACE SOIL (0-2 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-DNA#1NETP	0	2	10/22/1997	2	mg/kg
Highly Chlorinated PCBs	HB-DNA#1SETP	0	2	10/22/1997	0.18	mg/kg
Highly Chlorinated PCBs	HB-SS-01	0	0.5	12/3/2002	0.33	mg/kg
Highly Chlorinated PCBs	HB-SS-01	0.5	1	12/3/2002	0.41	mg/kg
Total PCBs	HB-DNA#1NETP	0	2	10/22/1997	2	mg/kg
Total PCBs	HB-DNA#1SETP	0	2	10/22/1997	0.18	mg/kg
Total PCBs	HB-SS-01	0	0.5	12/3/2002	0.33	mg/kg
Total PCBs	HB-SS-01	0.5	1	12/3/2002	0.41	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.25d
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #1 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-DSA#1NETP	10/22/1997	0	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0031	0.00155
HB-DSA#1NWTP	10/22/1997	0	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.12	0.12
HB-SS-01	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0085	
HB-SS-01	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0085	
HB-SS-01	12/3/2002	0	0.5	1330-20-7	TOTAL	N	U	mg/kg		0.0085
HB-SS-01	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.012	
HB-SS-01	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.012	
HB-SS-01	12/3/2002	0.5	1	1330-20-7	TOTAL	N	U	mg/kg		0.012

Notes:

a - Total Xylene value utilized in the risk assessment.

Table 2.26a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #1 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil
(0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
DSA #1 Subsurface Soil	DIOXIN/FURAN (9)															
	1746-01-6	2,3,7,8 TCDD Equivalent	0.00003	0.00004	mg/Kg	HB-SS-01	2/2		3.64E-05			4.26E-06	C	3.90E-06	ca	3.90E-06 Y ASL
	METALS															
	7429-90-5	ALUMINUM	3240	9600	mg/Kg	HB-DSA#1NWTP	7/7	-	9.60E+03			7.82E+03	N	7.61E+03	nc	7.61E+03 Y ASL
	7440-39-3	BARIUM	80	1700 J	mg/Kg	HB-TP-44	7/7	-	1.70E+03		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02 Y ASL
	7440-43-9	CADMIUM	0.83	3.1	mg/Kg	HB-TP-44	4/7	1-1	3.10E+00		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00 N BSL
	7440-70-2	CALCIUM	40000	310000	mg/Kg	HB-TP-44	7/7	-	3.10E+05			NV		NV		N NUT
	7440-47-3	CHROMIUM ^a	13 J	260 J	mg/Kg	HB-DSA#1NETP	7/7	-	2.60E+02			2.35E+01	N	3.01E+00	nc	3.01E+00 Y TOX
	7440-48-4	COBALT	5.9 J	9	mg/Kg	HB-DSA#1NETP	2/7	6-9	9.00E+00			NV		9.03E+01	nc	9.03E+01 N BSL
	7440-50-8	COPPER	55	240	mg/Kg	HB-SS-01	7/7	-	2.40E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02 N BSL
	57-12-5	CYANIDE	3.8	12	mg/Kg	HB-DSA#1NWTP	3/7	0.6-1.56	1.20E+01			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	7439-89-6	IRON	8600	20000	mg/Kg	HB-DSA#1NWTP	7/7	-	2.00E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL
	7439-92-1	LEAD	6.1	240	mg/Kg	HB-DSA#1NETP	7/7	-	2.40E+02			NV		4.00E+02	nc	4.00E+02 N BSL
	7439-95-4	MAGNESIUM	4730	24000 J	mg/Kg	HB-DSA#1NETP	7/7	-	2.40E+04			NV		NV		N NUT
	7439-96-5	MANGANESE	150 J	390 J	mg/Kg	HB-DSA#1NETP	7/7	-	3.90E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL
	7439-97-6	MERCURY ^a	0.2	97	mg/kg	HB-DSA#1NWTP	8/8	-	9.70E+01			2.35E+00	N	2.35E+00	nc	2.35E+00 Y ASL
	22967-92-6	METHYL MERCURY	3.23	3.23	ug/kg	HBSS01	1/1	-	3.23E+00			7.82E+02	N	6.11E+02	nc	6.11E+02 N BSL
	7440-02-0	NICKEL	18	49	mg/Kg	HB-DSA#1NWTP	7/7	-	4.90E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL
	7440-09-7	POTASSIUM	444	2900 J	mg/Kg	HB-DSA#1NWTP	6/7	900-900	2.90E+03			NV		NV		N NUT
	7782-49-2	SELENIUM	0.73 J	2.1	mg/Kg	HB-DSA#1NWTP	5/7	0.7-0.74	2.10E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-23-5	SODIUM	600 J	3800	mg/Kg	HB-TP-44	7/7	-	3.80E+03			NV		NV		N NUT
	7440-62-2	VANADIUM	11	28	mg/Kg	HB-DSA#1NWTP	7/7	-	2.80E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL
	7440-66-6	ZINC	36	133	mg/Kg	HB-SS-01	7/7	-	1.33E+02		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL
	PCBs															
		LESS CHLORINATED PCBs ^c	0.035	2.9	mg/kg	HB-DSA#1NWTP	3/10	0.019-4	2.90E+00			5.48E-01	N	3.93E-01	nc	3.93E-01 Y ASL
		HIGHLY CHLORINATED PCBs ^d	0.051	2	mg/kg	HB-DSA#1NETP	8/10	0.0533-4	2.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
		TOTAL PCBs ^e	0.086	2.4	mg/kg	HB-DSA#1NWTP	8/10	0.0533-4	2.90E+00			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
	SVOCs															
	91-57-6	2-METHYLNAPHTHALENE	2 J	13 J	mg/kg	HB-DSA#1NETP	3/7	0.38-26	1.30E+01			3.13E+01	N	NV		3.13E+01 N BSL
	208-96-8	ACENAPHTHYLENE	3.6 J	3.6 J	mg/kg	HB-DSA#1NETP	1/7	0.38-41	3.60E+00		1.00E+02	NV		NV		N NTX
	120-12-7	ANTHRACENE	6.6 J	6.6 J	mg/kg	HB-DSA#1NETP	1/7	0.38-41	6.60E+00		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03 N BSL
	56-55-3	BENZ(A)ANTHRACENE	0.34 J	5.9 J	mg/kg	HB-DSA#1NETP	4/7	0.38-41	5.90E+00		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	50-32-8	BENZO(A)PYRENE	0.42 J	4.9 J	mg/kg	HB-DSA#1NETP	3/6	0.38-41	4.90E+00		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.37 J	5.2 J	mg/kg	HB-DSA#1NETP	4/6	0.38-26	5.20E+00		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	207-08-9	BENZO(K)FLUORANTHENE	0.33 J	1.8 J	mg/kg	HB-DSA#1NETP	3/6	0.38-41	1.80E+00		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00 N BSL
	218-01-9	CHRYSENE	0.45 J	6.8 J	mg/kg	HB-DSA#1NETP	4/7	0.38-41	6.80E+00		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01 N BSL
	132-64-9	DIBENZOFURAN	5.2 J	5.2 J	mg/kg	HB-DSA#1NETP	1/7	0.38-41	5.20E+00		1.40E+01	7.82E+00	N	1.45E+01	nc	7.82E+00 N BSL
	206-44-0	FLUORANTHENE	0.71 J	12 J	mg/kg	HB-DSA#1NETP	6/7	0.38-0.38	1.20E+01		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02 N BSL
	118-74-1	HEXACHLOROBENZENE	0.04	13 J	mg/kg	HB-DSA#1NWTP	4/10	0.0016-26	1.30E+01		3.30E-01	3.99E-01	C	3.04E-01	ca	3.04E-01 Y ASL
	91-20-3	NAPHTHALENE	0.0021 J	73 J	mg/kg	HB-DSA#1NETP	6/11	0.0031-7	7.30E+01		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00 Y ASL
	85-01-8	PHENANTHRENE	0.45 J	28 J	mg/kg	HB-DSA#1NETP	5/7	0.38-41	2.80E+01		1.00E+02	NV		NV		Y NTX
	129-00-0	PYRENE	0.55 J	24 J	mg/kg	HB-DSA#1NETP	6/7	0.38-0.38	2.40E+01		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02 N BSL

Table 2.26a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #1 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil
(0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
	VOCs															
	87-61-6	1,2,3-TRICHLOROBENZENE	8.3	8.3	mg/kg	HB-DSA#1NWTP	1/3	0.0031-1.1	8.30E+00			NV		NV	Y	NTX
	120-82-1	1,2,4-TRICHLOROBENZENE	0.0022 J	350 J	mg/kg	HB-DSA#1NWTP	6/10	0.0031-2.6	3.50E+02			7.82E+01	N	6.22E+00	Y	ASL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.0019 J	19	mg/kg	HB-DSA#1NETP	2/4	0.0031-0.38	1.90E+01		4.70E+01	NV		5.16E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	0.0076	2700 J	mg/kg	HB-DSA#1NWTP	10/10	-	2.70E+03		1.00E+02	7.04E+02	N	6.00E+01	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	13	13	mg/kg	HB-DSA#1NETP	1/4	0.0031-0.38	1.30E+01		4.70E+01	NV		2.13E+00	Y	ASL
	541-73-1	1,3-DICHLOROBENZENE	0.0026 J	25	mg/kg	HB-DSA#1NETP	7/10	0.0031-2.6	2.50E+01		1.70E+01	2.35E+01	N	5.31E+01	Y	ASL
	106-46-7	1,4-DICHLOROBENZENE	0.0095 J	3400 J	mg/kg	HB-DSA#1NWTP	10/10	-	3.40E+03		9.80E+00	2.66E+01	C	3.45E+00	Y	ASL
	67-64-1	ACETONE	0.015	0.015	mg/kg	HB-DSA#1NETP	1/6	0.034-3.1	1.50E-02		1.00E+02	7.04E+03	N	1.41E+03	N	BSL
	71-43-2	BENZENE	0.63 J	0.71	mg/kg	HB-TP-44	2/10	0.0029-0.78	7.10E-01		2.90E+00	1.16E+01	C	6.43E-01	Y	TOX
	108-90-7	CHLOROBENZENE	0.0016 J	17	mg/kg	HB-DSA#1NETP	7/10	0.0031-0.012	1.70E+01		1.00E+02	1.56E+02	N	1.51E+01	Y	ASL
	100-41-4	ETHYLBENZENE	0.24 J	0.85 J	mg/kg	HB-DSA#1NETP	2/10	0.0029-0.78	8.50E-01		3.00E+01	7.82E+02	N	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE ^d	0.16 J	2.3	mg/kg	HB-DSA#1NETP	2/5	0.0031-0.38	2.30E+00			7.82E+02	N	5.72E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.0017 J	0.0023 J	mg/kg	HB-DSA#1NETP	2/10	0.0057-2.3	2.30E-03		5.10E+01	8.52E+01	C	9.11E+00	N	BSL
	103-65-1	N-PROPYLBENZENE	0.83 J	0.83 J	mg/kg	HB-DSA#1NETP	1/4	0.0031-0.38	8.30E-01		1.00E+02	NV		2.40E+01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	2.5	2.5	mg/kg	HB-DSA#1NETP	1/4	0.0031-0.38	2.50E+00			NV		NV	Y	NTX
	135-98-8	SEC-BUTYLBENZENE	0.0022 J	18	mg/kg	HB-DSA#1NETP	2/4	0.0031-0.38	1.80E+01		1.00E+02	NV		2.20E+01	N	BSL
	98-06-6	TERT-BUTYLBENZENE	1 J	1 J	mg/kg	HB-DSA#1NETP	1/4	0.0031-0.38	1.00E+00		1.00E+02	NV		3.90E+01	N	BSL
	108-88-3	TOLUENE	0.39 J	0.76 J	mg/kg	HB-DSA#1NETP	2/10	0.0029-0.78	7.60E-01		1.00E+02	6.26E+02	N	5.20E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	0.12 J	8.7	mg/kg	HB-DSA#1NETP	5/9	0.00145-0.012	8.70E+00		1.00E+02	1.56E+03	N	2.71E+01	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(8) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.26b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for mercury compounds utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
f = RBC and PRG values for isopropylbenzene are referred to as cumene (CAS # 98-82-8).

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency
DSA #1: Dredge Spoil Area #1
SCO = Soil Cleanup Objective

TABLE 2.26b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #1 SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SS-01	12/3/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	125.298	125.298	ng/kg		0.01	1.253
HB-SS-01	12/3/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	36.846	36.846	ng/kg		0.01	0.368
HB-SS-01	12/3/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	3.836	3.836	ng/kg		0.01	0.038
HB-SS-01	12/3/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.961	3.961	ng/kg		0.1	0.396
HB-SS-01	12/3/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	36.599	36.599	ng/kg		0.1	3.660
HB-SS-01	12/3/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	9.264	9.264	ng/kg		0.1	0.926
HB-SS-01	12/3/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	18.422	18.422	ng/kg		0.1	1.842
HB-SS-01	12/3/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	8.404	8.404	ng/kg		0.1	0.840
HB-SS-01	12/3/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	4.007	4.007	ng/kg		0.1	0.401
HB-SS-01	12/3/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	5.518	5.518	ng/kg		1	5.518
HB-SS-01	12/3/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	51.548	51.548	ng/kg		0.03	1.546
HB-SS-01	12/3/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	3.992	3.992	ng/kg		1	3.992
HB-SS-01	12/3/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	153.9	153.9	ng/kg	J	0.1	15.390
HB-SS-01	12/3/2002	0	0.5	3268-87-9	OCDD	Y	795.567	795.567	ng/kg	J	0.0003	0.239
HB-SS-01	12/3/2002	0	0.5	39001-02-0	OCDF	Y	48.338	48.338	ng/kg	J	0.0003	0.015
Sample Location TEQ = 36.4												
HB-SS-01	12/3/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	89.983	89.983	ng/kg		0.01	0.900
HB-SS-01	12/3/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	26.482	26.482	ng/kg		0.01	0.265
HB-SS-01	12/3/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.817	2.817	ng/kg		0.01	0.028
HB-SS-01	12/3/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.303	3.303	ng/kg		0.1	0.330
HB-SS-01	12/3/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	28.961	28.961	ng/kg		0.1	2.896
HB-SS-01	12/3/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.971	7.971	ng/kg		0.1	0.797
HB-SS-01	12/3/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	14.363	14.363	ng/kg		0.1	1.436
HB-SS-01	12/3/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.64	6.64	ng/kg		0.1	0.664
HB-SS-01	12/3/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	3.032	3.032	ng/kg		0.1	0.303
HB-SS-01	12/3/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	4.496	4.496	ng/kg		1	4.496
HB-SS-01	12/3/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	38.955	38.955	ng/kg		0.03	1.169
HB-SS-01	12/3/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	2.936	2.936	ng/kg		1	2.936
HB-SS-01	12/3/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	121.9	121.9	ng/kg	J	0.1	12.190
HB-SS-01	12/3/2002	0.5	1	3268-87-9	OCDD	Y	533.328	533.328	ng/kg	J	0.0003	0.160
HB-SS-01	12/3/2002	0.5	1	39001-02-0	OCDF	Y	34.961	34.961	ng/kg	J	0.0003	0.010
Sample Location TEQ = 28.6												

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable, J = estimated value

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223-241.

TABLE 2.26c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #1 SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-DSEA#1NETP	0	2	10/22/1997	2	mg/kg
Highly Chlorinated PCBs	HB-DSEA#1NETP	2	5	10/22/1997	0.051	mg/kg
Highly Chlorinated PCBs	HB-DSEA#1NETP	6	6	10/22/1997	0.67	mg/kg
Highly Chlorinated PCBs	HB-DSEA#1NWTP	3	4	10/22/1997	1.6	mg/kg
Highly Chlorinated PCBs	HB-DSEA#1SETP	0	2	10/22/1997	0.18	mg/kg
Highly Chlorinated PCBs	HB-SS-01	0	0.5	12/3/2002	0.33	mg/kg
Highly Chlorinated PCBs	HB-SS-01	0.5	1	12/3/2002	0.41	mg/kg
Highly Chlorinated PCBs	HB-TP-44	5	6	11/16/2006	0.294	mg/kg
Less Chlorinated PCBs	HB-DSEA#1NETP	2	5	10/22/1997	0.035	mg/kg
Less Chlorinated PCBs	HB-DSEA#1NETP	6	6	10/22/1997	1	mg/kg
Less Chlorinated PCBs	HB-DSEA#1NWTP	3	4	10/22/1997	2.9	mg/kg
Total PCBs	HB-DSEA#1NETP	0	2	10/22/1997	2	mg/kg
Total PCBs	HB-DSEA#1NETP	2	5	10/22/1997	0.086	mg/kg
Total PCBs	HB-DSEA#1NETP	6	6	10/22/1997	1.67	mg/kg
Total PCBs	HB-DSEA#1NWTP	3	4	10/22/1997	4.5	mg/kg
Total PCBs	HB-DSEA#1SETP	0	2	10/22/1997	0.18	mg/kg
Total PCBs	HB-SS-01	0	0.5	12/3/2002	0.33	mg/kg
Total PCBs	HB-SS-01	0.5	1	12/3/2002	0.41	mg/kg
Total PCBs	HB-TP-44	5	6	11/16/2006	0.294	mg/kg

Notes:

* Less Chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.26d
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #1 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-DSA#1NETP	10/22/1997	0	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0031	0.00155
HB-DSA#1NETP	10/22/1997	2	5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0029	0.00145
HB-DSA#1NETP	10/22/1997	6	6	1330-20-7	XYLENES, TOTAL	Y		mg/kg	8.7	8.7
HB-DSA#1NWTP	10/22/1997	0	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.12	0.12
HB-DSA#1NWTP	10/22/1997	3	4	1330-20-7	XYLENES, TOTAL	Y		mg/kg	7.6	7.6
HB-DSA#1NWTP	10/22/1997	5	5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.25	0.25
HB-TP-44	11/16/2006	5	6	1330-20-7	XYLENES, TOTAL	Y		mg/kg	1.8	1.8
HB-SS-01	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0085	
HB-SS-01	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0085	
HB-SS-01	12/3/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0085
HB-SS-01	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.012	
HB-SS-01	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.012	
HB-SS-01	12/3/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.012

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.27a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE DREDGE SPOIL AREA #2 SURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
DSA #2 Surface Soil	DIOXIN/FURAN (9)																	
	1746-01-6	2,3,7,8 TCDD Equivalent	0.0000025	0.0000027	mg/Kg	HB-SS-03	2/2		2.71E-06			4.26E-06	C	3.90E-06	ca	3.90E-06	N	BSL
	METALS																	
	7429-90-5	ALUMINUM	6470	11600	mg/Kg	HB-SS-03	4/4	-	1.16E+04			7.82E+03	N	7.61E+03	nc	7.61E+03	Y	ASL
	7440-36-0	ANTIMONY	0.29 J	0.54 J	mg/Kg	HB-HB-01D	2/4	6.58-6.63	5.40E-01			3.13E+00	N	3.13E+00	nc	3.13E+00	N	BSL
	7440-38-2	ARSENIC	3.4 J	13.8	mg/Kg	HB-HB-01D	4/4	-	1.38E+01		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX
	7440-39-3	BARIUM	60.6	203 J	mg/Kg	HB-HB-01D	4/4	-	2.03E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL
	7440-41-7	BERYLLIUM	0.44 J	0.52 J	mg/Kg	HB-HB-01D	2/4	0.55-0.55	5.20E-01		1.40E+01	1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL
	7440-43-9	CADMIUM	1.1	1.1	mg/Kg	HB-HB-01D	1/4	0.029-0.55	1.10E+00		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	N	BSL
	7440-70-2	CALCIUM	46900	166000	mg/Kg	HB-HB-01D	4/4	-	1.66E+05			NV	NV	NV	N	NUT		
	7440-47-3	CHROMIUM ^a	17.7 J	33.7 J	mg/Kg	HB-HB-01D	4/4	-	3.37E+01			2.35E+01	N	3.01E+01	ca	2.35E+01	Y	TOX
	7440-48-4	COBALT	6.6 J	12.2	mg/Kg	HB-SS-03	4/4	-	1.22E+01			NV	NV	9.03E-02	ca	9.03E-02	N	BSL
	7440-50-8	COPPER	24.1 J	74.4 J	mg/Kg	HB-HB-01D	4/4	-	7.44E+01		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL
	57-12-5	CYANIDE	0.68	0.68	mg/Kg	HB-HB-01D	1/4	0.51-1.16	6.80E-01			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL
	7439-89-6	IRON	16500	19500 J	mg/Kg	HB-HB-01D	4/4	-	1.95E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL
	7439-92-1	LEAD	6.1 J	99.7 J	mg/Kg	HB-HB-01D	4/4	-	9.97E+01			NV	NV	4.00E+02	nc	4.00E+02	N	BSL
	7439-95-4	MAGNESIUM	10300	36500	mg/Kg	HB-SS-03	4/4	-	3.65E+04			NV	NV	NV	N	NUT		
	7439-96-5	MANGANESE	268 J	395 J	mg/Kg	HB-SS-03	4/4	-	3.95E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL
	7439-97-6	MERCURY ^b	2.1 J	27.4 J	mg/kg	HB-HB-01D	2/4	0.04-0.04	2.74E+01			7.82E-01	N	6.11E-01	nc	6.11E-01	Y	ASL
	7440-02-0	NICKEL	23.3 J	42.9 J	mg/Kg	HB-HB-01D	4/4	-	4.29E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL
	7440-09-7	POTASSIUM	1250 J	2500 J	mg/Kg	HB-HB-01S	4/4	-	2.50E+03			NV	NV	NV	N	NUT		
	7782-49-2	SELENIUM	0.46 J	1.7 J	mg/Kg	HB-HB-01D	2/4	0.55-0.55	1.70E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL
	7440-22-4	SILVER	0.2 J	0.2 J	mg/Kg	HB-HB-01D	1/4	0.077-1.1	2.00E-01		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL
	7440-23-5	SODIUM	81.9	926	mg/Kg	HB-HB-01D	4/4	-	9.26E+02			NV	NV	NV	N	NUT		
	7440-62-2	VANADIUM	15.4 J	17.6	mg/Kg	HB-SS-03	4/4	-	1.76E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL
	7440-66-6	ZINC	34.2	76.1	mg/Kg	HB-HB-01D	4/4	-	7.61E+01		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL
	PCBs																	
		LESS CHLORINATED PCBs ^c	0.3	2	mg/Kg	HB-HB-01D	2/4	0.077-0.077	2.00E+00			5.48E-01	N	3.93E+00	ca	5.48E-01	Y	ASL
		HIGHLY CHLORINATED PCBs ^d	0.4	3	mg/Kg	HB-HB-01D	2/4	0.077-0.9	3.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
		TOTAL PCBs ^e	0.7	5	mg/kg	HB-HB-01D	2/4	0.077-0.9	5.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
	PESTICIDES																	
	57-74-9	TOTAL CHLORDANE ^f	0.001 J	0.1 J	mg/kg	HB-HB-01D	2/4	0.0039-0.004	1.00E-01			1.82E+00	C	1.62E+00	ca	1.62E+00	N	BSL
	SVOCs																	
	91-57-6	2-METHYLNAPHTHALENE	0.38 J	0.38 J	mg/kg	HB-HB-01D	1/4	0.34-0.38	3.80E-01			3.13E+01	N	NV	NV	3.13E+01	N	BSL
	208-96-8	ACENAPHTHYLENE	0.037 J	0.39 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	3.90E-01		1.00E+02	NV	NV	NV	NV	Y	NTX	
	56-55-3	BENZ(A)ANTHRACENE	0.05 J	0.58 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	5.80E-01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	50-32-8	BENZO(A)PYRENE	0.061 J	1.3 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	1.30E+00		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.15 J	3.4 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	3.40E+00		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.084 J	1.3 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	1.30E+00		1.00E+02	NV	NV	NV	NV	Y	NTX	
	207-08-9	BENZO(K)FLUORANTHENE	0.046 J	0.66 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	6.60E-01		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00	N	BSL
	65-85-0	BENZOIC ACID	0.091 J	0.091 J	mg/kg	HB-HB-01S	1/2	18-18	9.10E-02			3.13E+04	N	1.00E+04	nc	1.00E+04	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.45	0.45	mg/kg	HB-SS-03	1/4	0.34-3.5	4.50E-01			4.56E+01	C	3.47E+01	ca	3.47E+01	N	BSL
	218-01-9	CHRYSENE	0.065 J	0.79 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	7.90E-01		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	N	BSL
	206-44-0	FLUORANTHENE	0.093 J	0.92 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	9.20E-01		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	118-74-1	HEXACHLOROBENZENE	0.45	11 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	1.10E+01		3.30E-01	3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.082 J	1.3 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	1.30E+00		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.088 J	1.9 J	mg/kg	HB-HB-01D	2/6	0.005-0.38	1.90E+00		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	N	BSL
85-01-8	PHENANTHRENE	0.075 J	0.075 J	mg/kg	HB-HB-01S	1/4	0.38-3.5	7.50E-02		1.00E+02	NV	NV	NV	NV	Y	NTX		
108-95-2	PHENOL	0.044 J	0.044 J	mg/kg	HB-HB-01S	1/4	0.38-3.5	4.40E-02		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL	
129-00-0	PYRENE	0.08 J	2.2 J	mg/kg	HB-HB-01D	2/4	0.38-0.38	2.20E+00		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL	

TABLE 2.27a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE DREDGE SPOIL AREA #2 SURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	VOCs																	
	87-61-6	1,2,3-TRICHLOROBENZENE	0.057	0.057	mg/kg	HB-HB-01D	1/2	0.005-0.005	5.70E-02			NV	NV	NV	Y	NTX		
	120-82-1	1,2,4-TRICHLOROBENZENE	0.004 J	53 J	mg/kg	HB-HB-01D	4/6	0.38-0.38	5.30E+01			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.003 J	0.003 J	mg/kg	HB-HB-01D	1/2	0.003-0.003	3.00E-03	4.70E+01		NV	N	5.16E+00	nc	5.16E+00	N	BSL
	95-50-1	1,2-DICHLOROBENZENE	0.025 J	34 J	mg/kg	HB-HB-01D	4/6	0.38-0.38	3.40E+01	1.00E+02		7.04E+02	N	6.00E+01	nc	6.00E+01	N	BSL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.002 J	0.002 J	mg/kg	HB-HB-01D	1/2	0.003-0.003	2.00E-03	4.70E+01		NV	N	2.13E+00	nc	2.13E+00	N	BSL
	541-73-1	1,3-DICHLOROBENZENE	0.026	0.86 J	mg/kg	HB-HB-01D	3/6	0.003-0.38	8.60E-01	1.70E+01		2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.028 J	16 J	mg/kg	HB-HB-01D	4/6	0.38-0.38	1.60E+01	9.80E+00		2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL
	71-43-2	BENZENE	0.002 J	0.027	mg/kg	HB-HB-01D	2/4	0.0057-0.0058	2.70E-02	2.90E+00		1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	108-90-7	CHLOROBENZENE	0.011	0.37	mg/kg	HB-HB-01D	2/4	0.0057-0.0058	3.70E-01	1.00E+02		1.56E+02	N	1.51E+01	nc	1.51E+01	N	BSL
	100-41-4	ETHYLBENZENE	0.003 J	0.003 J	mg/kg	HB-HB-01D	1/4	0.003-0.0058	3.00E-03	3.00E+01		7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.001 J	0.001 J	mg/kg	HB-HB-01D	1/2	0.003-0.003	1.00E-03			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
	135-98-8	SEC-BUTYLBENZENE	0.002 J	0.002 J	mg/kg	HB-HB-01D	1/2	0.003-0.003	2.00E-03	1.00E+02		NV	N	2.20E+01	nc	2.20E+01	N	BSL
	108-88-3	TOLUENE	0.001 J	0.014	mg/kg	HB-HB-01D	2/4	0.0057-0.0058	1.40E-02	1.00E+02		6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL
	79-01-6	TRICHLOROETHENE	0.001 J	0.001 J	mg/kg	HB-HB-01D	1/4	0.003-0.0058	1.00E-03	1.00E+01		1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL
	1330-20-7	XYLENES, TOTAL	0.002 J	0.037	mg/kg	HB-HB-01D	2/4	0.0057-0.0058	3.70E-02	1.00E+02		1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

- (1) J = estimated value; N = tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
(5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL Above Screening Level; TOX Class A Carcinogen; NTX No Toxicity Information. Deletion Rationale: BSL Below Screening Level
(9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.27b.
= Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
f = RBC value for chlordanes (CAS# 57749) and PRG value for technical chlordanes (CAS# 12789-03-6) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency
DSA #1: Dredge Soil Area #1

TABLE 2.27b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #2 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SS-03	12/3/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	4.36	4.36	ng/kg	EMPC	0.01	0.044
HB-SS-03	12/3/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	0.537	0.537	ng/kg		0.01	0.005
HB-SS-03	12/3/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg		U	0.01
HB-SS-03	12/3/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-SS-03	12/3/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	U	0.03	0.038
HB-SS-03	12/3/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-SS-03	12/3/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1	1	ng/kg		0.1	0.100
HB-SS-03	12/3/2002	0	0.5	3268-87-9	OCDD	Y	21.764	21.764	ng/kg	J	0.0003	0.007
HB-SS-03	12/3/2002	0	0.5	39001-02-0	OCDF	N	5	2.5	ng/kg	UJ	0.0003	0.001
Sample Location TEQ = 2.7												
HB-SS-03	12/3/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	2.613	2.613	ng/kg	EMPC	0.01	0.026
HB-SS-03	12/3/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	0.543	0.543	ng/kg		0.01	0.005
HB-SS-03	12/3/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg		U	0.01
HB-SS-03	12/3/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.465	0.465	ng/kg	J	0.1	0.047
HB-SS-03	12/3/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.42	0.42	ng/kg	EMPC	0.1	0.042
HB-SS-03	12/3/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-SS-03	12/3/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	U	0.03	0.038
HB-SS-03	12/3/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-SS-03	12/3/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	1	1	ng/kg		0.1	0.100
HB-SS-03	12/3/2002	0.5	1	3268-87-9	OCDD	Y	9.932	9.932	ng/kg	J	0.0003	0.003
HB-SS-03	12/3/2002	0.5	1	39001-02-0	OCDF	N	5	2.5	ng/kg	UJ	0.0003	0.001
Sample Location TEQ = 2.5												

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

EMPC = Estimated Maximum Possible Concentration

N/A = not applicable, J = estimated value, U = non detect, EMPC = estimated maximum possible concentration

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.27c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #2 SURFACE SOIL (0-2 ft)

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-HB-01D	0	0.17	7/21/2000	3	mg/kg
Highly Chlorinated PCBs	HB-HB-01S	0	0.17	7/25/2000	0.4	mg/kg
Less Chlorinated PCBs	HB-HB-01D	0	0.17	7/21/2000	2	mg/kg
Less Chlorinated PCBs	HB-HB-01S	0	0.17	7/25/2000	0.3	mg/kg
Total PCBs	HB-HB-01D	0	0.17	7/21/2000	5	mg/kg
Total PCBs	HB-HB-01S	0	0.17	7/25/2000	0.7	mg/kg

Notes:

* Less Chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.27d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DSA #2 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-01D	7/21/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.09
HB-HB-01D	7/21/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.1
Total Chlordane =									0.1
HB-HB-01S	7/25/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-HB-01S	7/25/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.001
Total Chlordane =									0.001
HB-SS-03	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0039
HB-SS-03	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0039
Total Chlordane =									ND
HB-SS-03	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.004
HB-SS-03	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.004
Total Chlordane =									ND

TABLE 2.27e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #2 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-01D	7/21/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.037	0.037
HB-HB-01S	7/25/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.002	0.002
HB-SS-03	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0057	
HB-SS-03	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0057	
HB-SS-03	12/3/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0057
HB-SS-03	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0058	
HB-SS-03	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0058	
HB-SS-03	12/3/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0058

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.28a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #2 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
DSA #2 Subsurface Soil	DIOXIN/FURAN (9)															
	1746-01-6	2,3,7,8 TCDD Equivalent	0.0000025	0.0000027	mg/kg	HB-SS-03	2/2		2.71E-06			4.26E-06	C	3.90E-06	ca	3.90E-06 N BSL
	METALS															
	7429-90-5	ALUMINUM	3300	11600	mg/kg	HB-SS-03	9/9	-	1.16E+04			7.82E+03	N	7.61E+03	nc	7.61E+03 Y ASL
	7440-36-0	ANTIMONY	0.29 J	1.1 J	mg/kg	HB-TP-53	7/9	6.58-6.63	1.10E+00			3.13E+00	N	3.13E+00	nc	3.13E+00 N BSL
DSA #2 Subsurface Soil	7440-38-2	ARSENIC	3.4 J	55.4	mg/kg	HB-TP-03B	9/9	-	5.54E+01	1.60E+01		4.26E-01	C	3.90E-01	ca	3.90E-01 Y TOX
	7440-39-3	BARIUM	60.6	1300 J	mg/kg	HB-TP-53	9/9	-	1.30E+03	3.50E+02		1.56E+03	N	5.37E+02	nc	5.37E+02 Y ASL
	7440-41-7	BERYLLIUM	0.35 J	0.9	mg/kg	HB-HB-01S	7/9	0.55-0.55	9.00E-01	1.40E+01		1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL
	7440-43-9	CADMIUM	0.18 J	1.5	mg/kg	HB-TP-03B	6/9	0.029-0.55	1.50E+00	2.50E+00		3.91E+00	N	3.70E+00	nc	3.70E+00 N BSL
	7440-70-2	CALCIUM	46900	300000	mg/kg	HB-TP-53	9/9	-	3.00E+05			NV	NV	NV	N	NUT
	7440-47-3	CHROMIUM ^a	6.7 J	33.7 J	mg/kg	HB-HB-01D	9/9	-	3.37E+01			2.35E+01	N	3.01E+00	nc	3.01E+00 Y TOX
	7440-48-4	COBALT	1.6 J	12.2	mg/kg	HB-SS-03	9/9	-	1.22E+01			NV	NV	9.03E+01	nc	9.03E+01 N BSL
	7440-50-8	COPPER	24.1 J	106 J	mg/kg	HB-TP-03A	9/9	-	1.06E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02 N BSL
	57-12-5	CYANIDE	0.68	22.7	mg/kg	HB-TP-03C	5/9	0.51-1.16	2.27E-01			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	7439-89-6	IRON	5800	19500 J	mg/kg	HB-HB-01D	9/9	-	1.95E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL
	7439-92-1	LEAD	6.1 J	180 J	mg/kg	HB-TP-53	9/9	-	1.80E+02			NV	NV	4.00E+02	nc	4.00E+02 N BSL
	7439-95-4	MAGNESIUM	5910	36500	mg/kg	HB-SS-03	9/9	-	3.65E+04			NV	NV	NV	N	NUT
	7439-96-5	MANGANESE	114 J	395 J	mg/kg	HB-SS-03	9/9	-	3.95E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL
	7439-97-6	MERCURY ^b	2.1 J	48.1 J	mg/kg	HB-HB-01S	9/11	0.04-0.04	4.81E+01			7.82E-01	N	6.11E-01	nc	6.11E-01 Y ASL
	7440-02-0	NICKEL	6.6 J	42.9 J	mg/kg	HB-HB-01D	9/9	-	4.29E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL
	2023-69-5	POTASSIUM	220 J	2500 J	mg/kg	HB-HB-01S	8/9	7-7	2.50E+03			NV	NV	NV	N	NUT
	7782-49-2	SELENIUM	0.4 J	4.7 J	mg/kg	HB-TP-03B	7/9	0.55-0.55	4.70E+00	3.60E+01		3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-22-4	SILVER	0.13 J	0.55 J	mg/kg	HB-TP-53	4/9	0.077-1.1	5.50E-01	3.60E+01		3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-23-5	SODIUM	81.9	2200	mg/kg	HB-HB-01S	9/9	-	2.20E+03			NV	NV	NV	N	NUT
	7440-28-0	THALLIUM	0.95 J	0.95 J	mg/kg	HB-TP-03B	1/9	0.5-3.5	9.50E-01			7.15E+01	N	6.75E+01	nc	6.75E+01 N BSL
	7440-62-2	YANADIUM	8.4 J	22.3 J	mg/kg	HB-HB-01S	9/9	-	2.23E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL
	7440-66-6	ZINC	34.2	80	mg/kg	HB-TP-03A	9/9	-	8.00E+01		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL
	PESTICIDES															
	1031-07-8	ENDOSULFAN SULFATE ^c	0.3 J	0.3 J	mg/kg	HB-TP-03B	1/11	0.003-0.54	3.00E-01		2.40E+01	7.15E+01	N	3.69E+03	nc	7.15E+01 N BSL
	57-74-9	TOTAL CHLORDANE ^d	0.001 J	0.1 J	mg/kg	HB-HB-01D	2/11	0.0039-2.7	1.00E-01			1.82E+00	C	1.62E+00	ca	1.62E+00 N BSL
	PCBs															
		LESS CHLORINATED PCBs ^e	0.052	3	mg/kg	HB-DSA#2TP1	6/9	0.03-0.54	3.00E+00			5.48E-01	N	3.93E-01	nc	3.93E-01 Y ASL
		HIGHLY CHLORINATED PCBs ^f	0.07	3	mg/kg	HB-HB-01D	6/9	0.03-0.54	3.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
		TOTAL PCBs ^g	0.212	5	mg/kg	HB-HB-01D	6/9	0.03-0.54	3.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01 Y ASL
	SVOCs															
	91-57-6	2-METHYLNAPHTHALENE	0.38 J	1900 J	mg/kg	HB-TP-03B	5/10	0.19-57	1.90E+03			3.13E+01	N	NV	nc	3.13E+01 Y ASL
	83-32-9	ACENAPHTHENE	2.7 J	120 J	mg/kg	HB-TP-03B	3/10	0.34-57	1.20E+02		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02 N BSL
	208-96-8	ACENAPHTHYLENE	0.037 J	38 J	mg/kg	HB-TP-03B	4/10	0.38-57	3.80E+01		1.00E+02	NV	NV	NV	Y	NTX
	120-12-7	ANTHRACENE	1.4 J	260 J	mg/kg	HB-TP-03B	4/10	0.18-57	2.60E+02		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03 N BSL
	56-55-3	BENZ(A)ANTHRACENE	0.05 J	150 J	mg/kg	HB-TP-03B	6/10	0.3-57	1.50E+02		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	50-32-8	BENZO(A)PYRENE	0.061 J	68 J	mg/kg	HB-TP-03B	6/10	0.27-57	6.80E+01		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.15 J	110 J	mg/kg	HB-TP-03B	6/10	0.38-57	1.10E+02		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.084 J	12 J	mg/kg	HB-TP-03A	5/10	0.085-340	1.20E+01		1.00E+02	NV	NV	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.046 J	38 J	mg/kg	HB-TP-03B	6/10	0.38-57	3.80E+01		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00 Y ASL
	65-85-0	BENZOIC ACID	0.091 J	0.091 J	mg/kg	HB-HB-01S	1/7	10-1700	9.10E-02			3.13E+04	N	1.00E+04	nc	1.00E+04 N BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.45	0.45	mg/kg	HB-SS-03	1/10	0.34-340	4.50E-01			4.56E+01	C	3.47E+01	ca	3.47E+01 N BSL
	86-74-8	CARBAZOLE	0.22 J	43 J	mg/kg	HB-TP-03B	2/9	0.34-57	4.30E+01			7.15E+01	N	8.62E+01	ca	7.15E+01 N BSL
	218-01-9	CHRYSENE	0.065 J	130 J	mg/kg	HB-TP-03B	6/10	0.3-57	1.30E+02		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01 Y ASL
	132-64-9	DIBENZOFURAN	0.4 J	320 J	mg/kg	HB-TP-03B	3/10	0.074-57	3.20E+02		5.90E+01	7.15E+01	N	1.56E+03	nc	7.15E+01 Y ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	1.7 J	5.2 J	mg/kg	HB-TP-03A	3/10	0.34-340	5.20E+00		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL
	206-44-0	FLUORANTHENE	0.093 J	400 J	mg/kg	HB-TP-03B	7/10	0.38-57	4.00E+02		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02 Y ASL
	86-73-7	FLUORENE	4.5 J	270 J	mg/kg	HB-TP-03B	3/10	0.12-57	2.70E+02		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02 N BSL
	118-74-1	HEXACHLOROBENZENE	0.019	11 J	mg/kg	HB-HB-01D	4/12	0.13-340	1.10E+01		3.30E-01	3.99E-01	C	3.04E-01	ca	3.04E-01 Y ASL

TABLE 2.28a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #2 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.082 J	13 J	mg/kg	HB-TP-03A	5/10	0.07-340	1.30E+01		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.088 J	21000 J	mg/kg	HB-TP-03B	12/18	0.005-57	2.10E+04		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	85-01-8	PHENANTHRENE	0.075 J	1000 J	mg/kg	HB-TP-03B	5/10	0.38-57	1.00E+03		1.00E+02	NV	NV	NV	Y	NTX		
	108-95-2	PHENOL	0.044 J	0.044 J	mg/kg	HB-HB-01S	1/10	0.38-340	4.40E-02		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.08 J	240 J	mg/kg	HB-TP-03B	7/10	0.38-57	2.40E+02		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	Y	ASL
	VOCs																	
	87-61-6	1,2,3-TRICHLOROBENZENE	0.057	16	mg/kg	HB-DSA#2TP1	3/8	0.005-2600	1.60E+01			NV	NV	NV	Y	NTX		
	120-82-1	1,2,4-TRICHLOROBENZENE	0.004 J	120 J	mg/kg	HB-DSA#2TP1	10/17	0.38-2600	1.20E+02			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-63-6	1,2,4-TRIMETHYLBENZENE	0.003 J	250 J	mg/kg	HB-TP-03B	7/8	0.003-0.003	2.50E+02		4.70E+01	NV	N	5.16E+00	nc	5.16E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	0.025 J	920	mg/kg	HB-HB-01S	13/18	0.38-1300	9.20E+02		1.00E+02	7.04E+02	N	6.00E+01	nc	6.00E+01	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	0.002 J	150 J	mg/kg	HB-TP-03B	6/8	0.003-0.56	1.50E+02		4.70E+01	NV	N	2.13E+00	nc	2.13E+00	Y	ASL
	541-73-1	1,3-DICHLOROBENZENE	0.017	41	mg/kg	HB-TP-53	9/18	0.003-1300	4.10E+01		1.70E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	Y	ASL
	106-46-7	1,4-DICHLOROBENZENE	0.028 J	280 J	mg/kg	HB-DSA#2TP1	15/18	0.38-340	2.80E+02		9.80E+00	2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL
	78-93-3	2-BUTANONE	0.01 J	0.01 J	mg/kg	HB-TP-03C	1/9	0.01-5100	1.00E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	71-43-2	BENZENE	0.002 J	190 J	mg/kg	HB-TP-03B	7/11	0.0057-5.1	1.90E+02		2.90E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	108-86-1	BROMOBENZENE	0.092 J	0.092 J	mg/kg	HB-HB-01S	1/8	0.003-1300	9.20E-02			NV	N	2.78E+00	nc	2.78E+00	N	BSL
	74-83-9	BROMOMETHANE	0.76 J	0.76 J	mg/kg	HB-DSA#2TP2	1/11	0.005-2600	7.60E-01			1.10E+01	N	3.90E-01	nc	3.90E-01	Y	ASL
	104-51-8	BUTYLBENZENE	0.02	0.52 J	mg/kg	HB-HB-01S	2/8	0.003-1300	5.20E-01		1.00E+02	NV	N	2.40E+01	nc	2.40E+01	N	BSL
	108-90-7	CHLOROBENZENE	0.011	79	mg/kg	HB-HB-01S	7/11	0.0057-1300	7.90E+01		1.00E+02	1.56E+02	N	1.51E+01	nc	1.51E+01	Y	ASL
	74-87-3	CHLOROMETHANE	0.97 J	0.97 J	mg/kg	HB-DSA#2TP2	1/11	0.005-2600	9.70E-01			NV	N	4.69E+00	nc	4.69E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.003 J	5.9 J	mg/kg	HB-TP-03A	5/11	0.003-1300	5.90E+00		3.00E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE ^h	0.001 J	4.9 J	mg/kg	HB-TP-03A	6/9	0.003-1300	4.90E+00			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL
	108-87-2	METHYLCYCLOHEXANE	0.76 J	0.76 J	mg/kg	HB-TP-53	1/1	-	7.60E-01			NV	N	2.59E+02	nc	2.59E+02	N	BSL
	103-65-1	N-PROPYLBENZENE	0.025	0.58 J	mg/kg	HB-HB-01S	2/8	0.003-1300	5.80E-01		1.00E+02	NV	N	2.40E+01	nc	2.40E+01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	0.045	1.7 J	mg/kg	HB-HB-01S	3/8	0.003-1300	1.70E+00			NV	N	NV		NV	Y	NTX
	135-98-8	SEC-BUTYLBENZENE	0.002 J	14	mg/kg	HB-HB-01S	5/8	0.003-1300	1.40E+01		1.00E+02	NV	N	2.20E+01	nc	2.20E+01	N	BSL
	100-42-5	STYRENE	0.038	0.19 J	mg/kg	HB-HB-01S	2/11	0.003-1300	1.90E-01			1.56E+03	N	1.70E+02	nc	1.70E+02	N	BSL
	98-06-6	TERT-BUTYLBENZENE	0.051	1.1 J	mg/kg	HB-DSA#2TP1	4/8	0.003-1300	1.10E+00		1.00E+02	NV	N	3.90E+01	nc	3.90E+01	N	BSL
	127-18-4	TETRACHLOROETHENE	0.33 J	0.33 J	mg/kg	HB-DSA#2TP2	1/11	0.003-1300	3.30E-01		5.50E+00	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL
	108-88-3	TOLUENE	0.001 J	450 J	mg/kg	HB-TP-03B	6/11	0.0057-5.1	4.50E+02		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	Y	ASL
	79-01-6	TRICHLOROETHENE	0.001 J	0.001 J	mg/kg	HB-HB-01D	1/11	0.003-1300	1.00E-03		1.00E+01	1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL
	1330-20-7	XYLENES, TOTAL	0.002 J	8.60E+02	mg/kg	HB-TP-03B	9/11	0.0057-0.0058	8.60E+02		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	Y	ASL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
 - (9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.28b.
- = Compound detected in 100% of samples.
- NA = Not applicable, minimum and maximum values are calculated.
- a = RBC and PRG values for chromium VI utilized.
- b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.
- c = RBC and PRG values for Endosulfan (CAS #115297) utilized.
- d = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
- e = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
- f = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
- g = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
- h = RBC and PRG values for isopropylbenzene are referred to as cumene (CAS # 98-82-8).

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency
DSA #2: Dredge Spoil Area #2

TABLE 2.28b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #2 SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-SS-03	12/3/2002	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	4.36	4.36	ng/kg		0.01	0.044
HB-SS-03	12/3/2002	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	0.537	0.537	ng/kg	EMPC	0.01	0.005
HB-SS-03	12/3/2002	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	U	0.01	0.013
HB-SS-03	12/3/2002	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-SS-03	12/3/2002	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	U	0.03	0.038
HB-SS-03	12/3/2002	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-SS-03	12/3/2002	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1	1	ng/kg		0.1	0.100
HB-SS-03	12/3/2002	0	0.5	3268-87-9	OCDD	Y	21.764	21.764	ng/kg	J	0.0003	0.007
HB-SS-03	12/3/2002	0	0.5	39001-02-0	OCDF	N	5	2.5	ng/kg	UJ	0.0003	0.001
Sample Location TEQ =												2.7
HB-SS-03	12/3/2002	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	2.613	2.613	ng/kg		0.01	0.026
HB-SS-03	12/3/2002	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	0.543	0.543	ng/kg		0.01	0.005
HB-SS-03	12/3/2002	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	2.5	1.25	ng/kg	U	0.01	0.013
HB-SS-03	12/3/2002	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.465	0.465	ng/kg	J	0.1	0.047
HB-SS-03	12/3/2002	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.42	0.42	ng/kg	EMPC	0.1	0.042
HB-SS-03	12/3/2002	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-SS-03	12/3/2002	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	U	1	1.250
HB-SS-03	12/3/2002	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	2.5	1.25	ng/kg	U	0.03	0.038
HB-SS-03	12/3/2002	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-SS-03	12/3/2002	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	1	1	ng/kg		0.1	0.100
HB-SS-03	12/3/2002	0.5	1	3268-87-9	OCDD	Y	9.932	9.932	ng/kg	J	0.0003	0.003
HB-SS-03	12/3/2002	0.5	1	39001-02-0	OCDF	N	5	2.5	ng/kg	UJ	0.0003	0.001
Sample Location TEQ =												2.5

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable, J = estimated value, U = non detect, EMPC = estimated maximum possible concentration

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.28c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #2 SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-DSA#2TP1	5	5	9/19/1997	1.5	mg/kg
Highly Chlorinated PCBs	HB-DSA#2TP2	5	5	9/19/1997	0.16	mg/kg
Highly Chlorinated PCBs	HB-HB-01D	0	0.17	7/21/2000	3	mg/kg
Highly Chlorinated PCBs	HB-HB-01S	0	0.17	7/25/2000	0.4	mg/kg
Highly Chlorinated PCBs	HB-HB-01S	8	10	7/25/2000	0.07	mg/kg
Highly Chlorinated PCBs	HB-TP-53	3	4	11/14/2006	0.499	mg/kg
Less Chlorinated PCBs	HB-DSA#2TP1	5	5	9/19/1997	3	mg/kg
Less Chlorinated PCBs	HB-DSA#2TP2	5	5	9/19/1997	0.052	mg/kg
Less Chlorinated PCBs	HB-HB-01D	0	0.17	7/21/2000	2	mg/kg
Less Chlorinated PCBs	HB-HB-01S	0	0.17	7/25/2000	0.3	mg/kg
Less Chlorinated PCBs	HB-HB-01S	8	10	7/25/2000	0.3	mg/kg
Less Chlorinated PCBs	HB-TP-53	3	4	11/14/2006	0.994	mg/kg
Total PCBs	HB-DSA#2TP1	5	5	9/19/1997	5.58	mg/kg
Total PCBs	HB-DSA#2TP2	5	5	9/19/1997	0.272	mg/kg
Total PCBs	HB-HB-01D	0	0.17	7/21/2000	9.5	mg/kg
Total PCBs	HB-HB-01S	0	0.17	7/25/2000	1.7	mg/kg
Total PCBs	HB-HB-01S	8	10	7/25/2000	0.52	mg/kg
Total PCBs	HB-TP-53	3	4	11/14/2006	2.081	mg/kg

Notes:

* Less Chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.28d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DSA #2 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-DSA#2TP1	9/19/1997	5	5	57-74-9	CHLORDANE	N	U	mg/kg	2.7
Total Chlordane =									ND
HB-DSA#2TP2	9/19/1997	5	5	57-74-9	CHLORDANE	N	U	mg/kg	0.15
Total Chlordane =									ND
HB-HB-01D	7/21/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.09
HB-HB-01D	7/21/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.1
Total Chlordane =									0.1
HB-HB-01S	7/25/2000	0	0.17	57-74-9	CHLORDANE	N	U	mg/kg	0.002
HB-HB-01S	7/25/2000	0	0.17	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.001
Total Chlordane =									0.001
HB-HB-01S	7/25/2000	8	10	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-HB-01S	7/25/2000	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-SS-03	12/3/2002	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0039
HB-SS-03	12/3/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0039
Total Chlordane =									ND
HB-SS-03	12/3/2002	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.004
HB-SS-03	12/3/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.004
Total Chlordane =									ND
HB-TP-03A	11/14/2006	6.5	6.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.1
HB-TP-03A	11/14/2006	6.5	6.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.1
Total Chlordane =									ND
HB-TP-03B	11/14/2006	9.5	9.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.2
HB-TP-03B	11/14/2006	9.5	9.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.2
Total Chlordane =									ND
HB-TP-03C	11/14/2006	9	9	57-74-9	CHLORDANE	N	UJ	mg/kg	0.04
HB-TP-03C	11/14/2006	9	9	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.04
Total Chlordane =									ND
HB-TP-53	11/14/2006	3	4	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-TP-53	11/14/2006	3	4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND

TABLE 2.28e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #2 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-DSA#2TP1	9/19/1997	5	5	1330-20-7	XYLENES, TOTAL	Y		mg/kg	8.8	8.8
HB-DSA#2TP2	9/19/1997	5	5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.19	0.19
HB-HB-01D	7/21/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.037	0.037
HB-HB-01S	7/25/2000	0	0.17	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.002	0.002
HB-HB-01S	7/25/2000	8	10	1330-20-7	XYLENES, TOTAL	Y		mg/kg	21	21
HB-TP-03A	7/19/2000	6.5	6.5	1330-20-7	XYLENES, TOTAL	Y		mg/kg	170	170
HB-TP-03B	7/19/2000	9.5	9.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	860	860
HB-TP-03C	7/19/2000	9	9	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.93	0.93
HB-TP-53	11/14/2006	3	4	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.52	0.52
HB-SS-03	12/3/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0057	
HB-SS-03	12/3/2002	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0057	
HB-SS-03	12/3/2002	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0057
HB-SS-03	12/3/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0058	
HB-SS-03	12/3/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0058	
HB-SS-03	12/3/2002	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0058

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.29a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #2 SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)	
DSA #2 Shallow Ground Water	METALS																
	7429-90-5	ALUMINUM	0.05 J	0.801	mg/L	HB-HB-01S	4/4	-	8.01E-01		2.00 E-01	3.65E+00	N	3.65E+00	nc	BSL	
	7440-36-0	ANTIMONY	0.0016 J	0.0016 J	mg/L	HB-HB-01S	1/4	0.06-0.06	1.60E-03		6.00 E-03	1.46E-03	N	1.46E-03	nc	ASL	
	7440-38-2	ARSENIC	0.0039 J	0.0039 J	mg/L	HB-HB-01S	1/4	0.01-0.01	3.90E-03		1.00 E-02	4.46E-05	C	4.48E-05	ca	TOX	
	7440-39-3	BARIUM	0.063	0.663	mg/L	HB-HB-01S	4/4	-	6.63E-01		2.00 E+00	7.30E-01	N	2.55E-01	nc	ASL	
	7440-70-2	CALCIUM	310	752	mg/L	HB-HB-01S	4/4	-	7.52E+02			NV	NV	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	0.002 J	0.002 J	mg/L	HB-HB-01S	1/4	0.01-0.01	2.00E-03		1.00 E-01	1.10E-02	N	1.09E-02	nc	TOX	
	7440-50-8	COPPER	0.0136 J	0.0136 J	mg/L	HB-HB-01S	1/4	0.01-0.02	1.36E-02		1.30 E+00	1.46E-01	N	1.46E-01	nc	BSL	
	57-12-5	CYANIDE	0.026	0.026	mg/L	HB-HB-01S	1/4	0.01-0.01	2.60E-02		2.00 E-01	7.30E-02	N	7.30E-02	nc	BSL	
	7439-89-6	IRON	0.057	0.796	mg/L	HB-HB-01S	4/4	-	7.96E-01		3.00 E-01	2.56E+00	N	1.09E+00	nc	BSL	
	7439-92-1	LEAD	0.0068 J	0.0097	mg/L	HB-HB-01S	2/4	0.005-0.01	9.70E-03		1.50 E-02	NV	NV	NV	N	BSL	
	7439-95-4	MAGNESIUM	2.13 J	24	mg/L	HB-HB-01S	4/4	-	2.40E+01			NV	NV	NV	N	NUT	
	7439-96-5	MANGANESE	0.0024 J	0.041 J	mg/L	HB-HB-01S	4/4	-	4.10E-02		5.00 E-02	7.30E-02	N	8.76E-02	nc	BSL	
	7439-97-6	MERCURY ^b	0.00022 J	0.0042	mg/L	HB-HB-01S	4/4	-	4.20E-03		2.00 E-03	3.65E-04	N	3.65E-04	nc	ASL	
	7440-02-0	NICKEL	0.0069 J	0.0404	mg/L	HB-HB-01S	2/4	0.04-0.04	4.04E-02			7.30E-02	N	7.30E-02	nc	BSL	
	7440-09-7	POTASSIUM	42 J	98.3	mg/L	HB-HB-01S	4/4	-	9.83E+01			NV	NV	NV	N	NUT	
	7440-23-5	SODIUM	435	887	mg/L	HB-HB-01S	4/4	-	8.87E+02			NV	NV	NV	N	NUT	
	7440-62-2	VANADIUM	0.0011 J	0.0044 J	mg/L	HB-HB-01S	2/4	0.05-0.05	4.40E-03			3.65E-03	N	3.65E-03	nc	ASL	
	7440-66-6	ZINC	0.0122 J	0.018 J	mg/L	HB-HB-01S	2/4	0.02-0.02	1.80E-02		5.00 E+00	1.10E+00	N	1.09E+00	nc	BSL	
	PESTICIDES																
	33213-65-9	ENDOSULFAN II ^c	0.2	0.2	ug/l	HB-HB-01S	1/4	0.096-0.1	2.00E-01				2.19E+01	N	2.19E+01	nc	BSL
	SVOCs																
	92-52-4	1,1'-BIPHENYL	4.1 J	4.1 J	ug/l	HB-HB-01S	1/1	-	4.10E+00				3.04E+01	N	3.04E+01	nc	BSL
95-95-4	2,4,5-TRICHLOROPHENOL	7 J	7 J	ug/l	HB-HB-01S	1/4	100-1300	7.00E+00				3.65E+02	N	3.65E+02	nc	BSL	
120-83-2	2,4-DICHLOROPHENOL	7.4 J	7.4 J	ug/l	HB-HB-01S	1/4	100-260	7.40E+00				1.10E+01	N	1.09E+01	nc	BSL	
105-67-9	2,4-DIMETHYLPHENOL	3 J	3 J	ug/l	HB-HB-01S	1/4	100-260	3.00E+00				7.30E+01	N	7.30E+01	nc	BSL	
91-57-6	2-METHYLNAPHTHALENE	17	40 J	ug/l	HB-HB-01S	4/4	-	4.00E+01				2.43E+00	N	NV	NV	Y	ASL
106-44-5	4-METHYLPHENOL	1.8 J	1.8 J	ug/l	HB-HB-01S	1/1	-	1.80E+00				1.83E+01	N	1.82E+01	nc	BSL	
83-32-9	ACENAPHTHENE	1.4 J	1.4 J	ug/l	HB-HB-01S	1/4	100-260	1.40E+00				3.65E+01	N	3.65E+01	nc	BSL	
208-96-8	ACENAPHTHYLENE	1.5 J	1.5 J	ug/l	HB-HB-01S	1/4	100-260	1.50E+00				NV	NV	NV	Y	NTX	
86-74-8	CARBAZOLE	2 J	2 J	ug/l	HB-HB-01S	1/4	100-260	2.00E+00				3.35E+00	C	3.36E+00	ca	BSL	
132-64-9	DIBENZOFURAN	1.5 J	1.5 J	ug/l	HB-HB-01S	1/4	100-260	1.50E+00				3.65E+00	N	1.22E+00	nc	ASL	
91-20-3	NAPHTHALENE	510	1300	ug/l	HB-HB-01S	4/4	-	1.30E+03				6.51E-01	N	6.20E-01	nc	ASL	
85-01-8	PHENANTHRENE	1.2 J	1.2 J	ug/l	HB-HB-01S	1/4	100-260	1.20E+00				NV	NV	NV	Y	NTX	
108-95-2	PHENOL	15	160 J	ug/l	HB-HB-01S	4/4	-	1.60E+02				1.10E+03	N	1.09E+03	nc	BSL	
VOCs																	
87-61-6	1,2,3-TRICHLOROBENZENE	19	19	ug/l	HB-HB-01S	1/1	-	1.90E+01				NV	NV	NV	Y	NTX	
120-82-1	1,2,4-TRICHLOROBENZENE	46 J	170	ug/l	HB-HB-01S	4/5	250-250	1.70E+02		7.00 E+01	6.08E+00	N	7.16E-01	nc	ASL		
95-63-6	1,2,4-TRIMETHYLBENZENE	400	400	ug/l	HB-HB-01S	1/1	-	4.00E+02				1.46E+00	N	1.23E+00	nc	ASL	
95-50-1	1,2-DICHLOROBENZENE	1800	6480	ug/l	HB-HB-01S	4/4	-	6.48E+03		6.00 E+02	2.68E+01	N	3.70E+01	nc	ASL		
108-67-8	1,3,5-TRIMETHYLBENZENE	250	250	ug/l	HB-HB-01S	1/1	-	2.50E+02				NV	NV	NV	Y	ASL	
541-73-1	1,3-DICHLOROBENZENE	14 J	14 J	ug/l	HB-HB-01S	1/5	5-260	1.40E+01				1.83E+00	N	1.83E+01	nc	ASL	

TABLE 2.29a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- DREDGE SPOIL AREA #2 SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	106-46-7	1,4-DICHLOROBENZENE	990	2920	ug/l	HB-HB-01S	4/4	-	2.92E+03		7.50 E+01	2.81E-01	C	5.02E-01	ca	2.81E-01	Y	ASL
	78-93-3	2-BUTANONE	18 J	23 J	ug/l	HB-HB-01S	2/4	100-2500	2.30E+01			6.97E+02	N	6.97E+02	nc	6.97E+02	N	BSL
	67-64-1	ACETONE	51 J	57 J	ug/l	HB-HB-01S	2/4	100-2500	5.70E+01			5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	98-86-2	ACETOPHENONE	17	17	ug/l	HB-HB-01S	1/1	-	1.70E+01			6.08E+01	N	NV		6.08E+01	N	BSL
	71-43-2	BENZENE	162	330	ug/l	HB-HB-01S	4/4	-	3.30E+02		5.00 E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	104-51-8	BUTYLBENZENE	5 J	5 J	ug/l	HB-HB-01S	1/1	-	5.00E+00			NV		2.43E+01	nc	2.43E+01	N	BSL
	108-90-7	CHLOROBENZENE	580	3080	ug/l	HB-HB-01S	3/3	-	3.08E+03		1.00 E+02	8.96E+00	N	1.06E+01	nc	8.96E+00	Y	ASL
	100-41-4	ETHYLBENZENE	38 J	60	ug/l	HB-HB-01S	3/4	125-125	6.00E+01		7.00 E+02	1.34E+02	N	1.34E+02	nc	1.34E+02	N	BSL
	98-82-8	ISOPROPYLBENZENE	37.5 J	68	ug/l	HB-HB-01S	2/2	-	6.80E+01			6.58E+01	N	6.58E+01	nc	6.58E+01	Y	ASL
	75-09-2	METHYLENE CHLORIDE	25 J	25 J	ug/l	HB-HB-01S	1/4	20-50	2.50E+01		5.00 E+00	4.10E+00	C	4.28E+00	ca	4.10E+00	Y	ASL
	103-65-1	N-PROPYLBENZENE	9	9	ug/l	HB-HB-01S	1/1	-	9.00E+00			NV		2.43E+01	nc	2.43E+01	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	20	20	ug/l	HB-HB-01S	1/1	-	2.00E+01			NV		NV		NV	Y	NTX
	135-98-8	SEC-BUTYLBENZENE	120	120	ug/l	HB-HB-01S	1/1	-	1.20E+02			NV		2.43E+01	nc	2.43E+01	Y	ASL
	100-42-5	STYRENE	35 J	37	ug/l	HB-HB-01S	2/4	50-125	3.70E+01		1.00 E+02	1.62E+02	N	1.64E+02	nc	1.62E+02	N	BSL
	98-06-6	TERT-BUTYLBENZENE	11	11	ug/l	HB-HB-01S	1/1	-	1.10E+01			NV		2.43E+01	nc	2.43E+01	N	BSL
	108-88-3	TOLUENE	87.5 J	610	ug/l	HB-HB-01S	3/3	-	6.10E+02		1.00 E+03	2.27E+02	N	7.23E+01	nc	7.23E+01	Y	ASL
	1330-20-7	XYLENES, TOTAL	475	1230	ug/l	HB-HB-01S	3/3	-	1.23E+03		1.00 E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	Y	ASL

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.

(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by

(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG

(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.

(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = RBC and PRG values for chromium VI utilized.

b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

c = RBC and PRG values for Endosulfan (CAS# 115297) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NUT: Compound is an essential nutrient and not screened in

NV: No Value

PRG: Preliminary Remediation Goals, USEPA, 2004

RBC: Risk Based Concentration; USEPA, October, 2007

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.29b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #2 SHALLOW GROUNDWATER (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-01S	5/14/2003	4.95	9.95	95-47-6	O-XYLENE	Y		ug/l	300	
HB-HB-01S	5/14/2003	4.95	9.95	XYLENES1314	XYLENES, M & P	Y		ug/l	650	
HB-HB-01S	5/14/2003	4.95	9.95	CALCULATED	TOTAL	Y		ug/l		950
HB-HB-01S	8/19/2003	4.95	9.95	95-47-6	O-XYLENE	Y		ug/l	400	
HB-HB-01S	8/19/2003	4.95	9.95	XYLENES1314	XYLENES, M & P	Y		ug/l	830	
HB-HB-01S	8/19/2003	4.95	9.95	CALCULATED	TOTAL	Y		ug/l		1230
HB-HB-01S	3/12/2007	4.95	9.95	1330-20-7	XYLENES, TOTAL	Y		ug/l	475	475

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.30a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE: DREDGE SPOIL AREA #2 SHALLOW GROUND WATER: VAPOR INTRUSION
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	Target Groundwater Concentration Corresponding to Target Indoor Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor = 0.001 and Partitioning Across the Water Table Obeys Henry's Law (5)	Screening Toxicity Value	COPC Flag (Y/N)	Rationale for Selection or Deletion (6)
DSA #2 - Shallow Ground Water	SVOCs														
	95-95-4	2,4,5-TRICHLOROPHENOL	7 J	7 J	ug/l	HB-HB-01S	1/4	100-1300	7.00E+00				NV	Y	NTX
	120-83-2	2,4-DICHLOROPHENOL	7.4 J	7.4 J	ug/l	HB-HB-01S	1/4	100-260	7.40E+00				NV	Y	NTX
	105-67-9	2,4-DIMETHYLPHENOL	3 J	3 J	ug/l	HB-HB-01S	1/4	100-260	3.00E+00				NV	Y	NTX
	91-57-6	2-METHYLNAPHTHALENE	17	40 J	ug/l	HB-HB-01S	4/4	-	4.00E+01			3.30E+02	nc	N	BSL
	92-52-4	1,1'-BIPHENYL	4.1 J	4.1 J	ug/l	HB-HB-01S	1/1	-	4.10E+00			**	nc	N	INC
	106-44-5	4-METHYLPHENOL	1.8 J	1.8 J	ug/l	HB-HB-01S	1/1	-	1.80E+00				NV	Y	NTX
	83-32-9	ACENAPHTHENE	1.4 J	1.4 J	ug/l	HB-HB-01S	1/4	100-260	1.40E+00			**	nc	N	INC
	208-96-8	ACENAPHTHYLENE	1.5 J	1.5 J	ug/l	HB-HB-01S	1/4	100-260	1.50E+00				NV	Y	NTX
	86-74-8	CARBAZOLE	2 J	2 J	ug/l	HB-HB-01S	1/4	100-260	2.00E+00				NV	Y	NTX
	132-64-9	DIBENZOFURAN	1.5 J	1.5 J	ug/l	HB-HB-01S	1/4	100-260	1.50E+00			**	nc	N	INC
	91-20-3	NAPHTHALENE	510	1300	ug/l	HB-HB-01S	4/4	-	1.30E+03			1.50E+01	nc	Y	ASL
	85-01-8	PHENANTHRENE	1.2 J	1.2 J	ug/l	HB-HB-01S	1/4	100-260	1.20E+00				NV	Y	NTX
	108-95-2	PHENOL	15	160 J	ug/l	HB-HB-01S	4/4	-	1.60E+02				NV	Y	NTX
	VOCs														
	120-82-1	1,2,4-TRICHLOROBENZENE	46 J	170	ug/l	HB-HB-01S	4/5	250-250	1.70E+02		7.00 E+01	**	nc	N	INC
	95-50-1	1,2-DICHLOROBENZENE	1800	6480	ug/l	HB-HB-01S	4/4	-	6.48E+03		6.00 E+02	2.60E+02	nc	Y	ASL
	541-73-1	1,3-DICHLOROBENZENE	14 J	14 J	ug/l	HB-HB-01S	1/5	5-260	1.40E+01			8.30E+01	nc	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	990	2920	ug/l	HB-HB-01S	4/4	-	2.92E+03		7.50 E+01	8.20E+02	nc	Y	ASL
	78-93-3	2-BUTANONE	18 J	23 J	ug/l	HB-HB-01S	2/4	100-2500	2.30E+01			4.40E+04	nc	N	BSL
	67-64-1	ACETONE	51 J	57 J	ug/l	HB-HB-01S	2/4	100-2500	5.70E+01			2.20E+04	nc	N	BSL
	98-86-2	ACETOPHENONE	17	17	ug/l	HB-HB-01S	1/1	-	1.70E+01			8.00E+04	nc	N	BSL
	71-43-2	BENZENE	162	330	ug/l	HB-HB-01S	4/4	-	3.30E+02	5.00 E+00		1.37E+01	c	Y	TOX
	108-90-7	CHLOROBENZENE	580	3080	ug/l	HB-HB-01S	3/3	-	3.08E+03	1.00 E+02		3.90E+01	nc	Y	ASL
	100-41-4	ETHYLBENZENE	38 J	60	ug/l	HB-HB-01S	3/4	125-125	6.00E+01	7.00 E+02		3.01E+01	c	Y	ASL
	87-61-6	1,2,3-TRICHLOROBENZENE	19	19	ug/l	HB-HB-01S	1/1	-	1.90E+01			NV	Y	NTX	
	95-63-6	1,2,4-TRIMETHYLBENZENE	400	400	ug/l	HB-HB-01S	1/1	-	4.00E+02			2.40E+00	nc	Y	ASL
	108-67-8	1,3,5-TRIMETHYLBENZENE	250	250	ug/l	HB-HB-01S	1/1	-	2.50E+02			2.50E+00	nc	Y	ASL
	98-82-8	ISOPROPYLBENZENE	37.5 J	68	ug/l	HB-HB-01S	2/2	-	6.80E+01			NV	Y	NTX	
	75-09-2	METHYLENE CHLORIDE	25 J	25 J	ug/l	HB-HB-01S	1/4	20-50	2.50E+01	5.00 E+00		5.80E+01	c	N	BSL
	104-51-8	BUTYLBENZENE	5 J	5 J	ug/l	HB-HB-01S	1/1	-	5.00E+00		1.00 E+02	2.60E+01	nc	N	BSL
	100-42-5	STYRENE	35 J	37	ug/l	HB-HB-01S	2/4	50-125	3.70E+01		1.00 E+02	8.90E+02	nc	N	BSL
	103-65-1	N-PROPYLBENZENE	9	9	ug/l	HB-HB-01S	1/1	-	9.00E+00			3.20E+01	nc	N	BSL
	99-87-6	P-ISOPROPYLTOLUENE	20	20	ug/l	HB-HB-01S	1/1	-	2.00E+01			NV	Y	NTX	
	135-98-8	SEC-BUTYLBENZENE	120	120	ug/l	HB-HB-01S	1/1	-	1.20E+02			2.50E+01	nc	Y	ASL
	108-88-3	TOLUENE	87.5 J	610	ug/l	HB-HB-01S	3/3	-	6.10E+02	1.00 E+03		1.50E+02	nc	Y	ASL
	98-06-6	TERT-BUTYLBENZENE	11	11	ug/l	HB-HB-01S	1/1	-	1.10E+01			2.90E+01	nc	N	BSL
	1330-20-7	XYLENES, TOTAL ^a	475	1230	ug/l	HB-HB-01S	3/3	-	1.23E+03	1.00 E+04		2.20E+03	nc	N	BSL

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

**Target soil gas concentration exceeds maximum possible vapor concentration (pathway incomplete)

(1) J - estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) Primary and Secondary Drinking Water Regulations

(5) USEPA - OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils (Subsurface Vapor Intrusion Guidance) Tables. November 2002. ca = Cancer; nc = Noncancer. Screening criteria correspond to a cancer risk of 10⁻⁶ and a noncancer hazard of 0.1. For USEPA (2002) criteria that defaulted to MCLs, criteria were derived (in italics) from USEPA (2009) RSL residential air concentration based on an attenuation factor of 10 and the Henry's Law constant for each compound at 25 deg C.

(6) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level; INC - Pathway Incomplete

- = Compound detected in 100% of samples.

a = Target groundwater concentration for p-xylene (CAS #106-42-3) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NV: No Value

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.30b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - DREDGE SPOIL AREA #2 SHALLOW GROUND WATER: VAPOR INTRUSION

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-01S	5/14/2003	4.95	9.95	95-47-6	O-XYLENE	Y		ug/l	300	
HB-HB-01S	5/14/2003	4.95	9.95	XYLENES1314	XYLENES, M & P	Y		ug/l	650	
HB-HB-01S	5/14/2003	4.95	9.95	CALCULATED	TOTAL	Y		ug/l		950
HB-HB-01S	8/19/2003	4.95	9.95	95-47-6	O-XYLENE	Y		ug/l	400	
HB-HB-01S	8/19/2003	4.95	9.95	XYLENES1314	XYLENES, M & P	Y		ug/l	830	
HB-HB-01S	8/19/2003	4.95	9.95	CALCULATED	TOTAL	Y		ug/l		1230
HB-HB-01S	3/12/2007	4.95	9.95	1330-20-7	XYLENES, TOTAL	Y		ug/l	475	475

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.31a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL HARBOR BROOK SITE- AOS #1 SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
AOS #1 Surface Soil	DIOXIN/FURAN (9)																		
	1746-01-6	2,3,7,8-TCDD Equivalent	0.000003	0.00002	mg/kg	HB-HB-20D	6/6		1.83E-05			4.26E-06	C	3.90E-06	ca	3.899E-06	Y	ASL	
	METALS																		
	7429-90-5	ALUMINUM	2820	14300 J	mg/kg	HB-HB-20D	20/20	-	1.43E+04			7.82E+03	N	7.61E+03	nc	7.61E+03	Y	ASL	
	7440-38-2	ARSENIC	2.1	14 J	mg/kg	HB-RISB-04	18/19	7.1-7.1	1.40E+01		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX	
	7440-39-3	BARIUM	46.2	389 J	mg/kg	HB-HB-20D	20/20	-	3.89E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL	
	7440-43-9	CADMIUM	0.97	9.9 J	mg/kg	HB-RISB-07	14/19	0.53-3.5	9.90E+00		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	Y	ASL	
	7440-70-2	CALCIUM	23000 J	201000	mg/kg	HB-HB-18S	20/20	-	2.01E+05			NV	NV	NV	N	NUT			
	7440-47-3	CHROMIUM ^a	18.4	191 J	mg/kg	HB-HB-20D	20/20	-	1.91E+02			2.35E+01	N	3.01E+01	ca	2.35E+01	Y	TOX	
	7440-48-4	COBALT	8	8.9	mg/kg	HB-RISB-11	2/19	6.28-35.4	8.90E+00			NV	NV	9.03E+02	ca	9.03E+02	N	BSL	
	7440-50-8	COPPER	21.8	302 J	mg/kg	HB-HB-19S	20/20	-	3.02E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL	
	7439-89-6	IRON	5810 J	22300 J	mg/kg	HB-HB-19S	20/20	-	2.23E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	58.8 J	2320 J	mg/kg	HB-HB-20D	20/20	-	2.32E+03			NV	NV	4.00E+02	nc	4.00E+02	Y	ASL	
	7439-95-4	MAGNESIUM	2880 J	24200 J	mg/kg	HB-HB-20D	20/20	-	2.42E+04			NV	NV	NV	N	NUT			
	7439-96-5	MANGANESE	60.6 J	330 J	mg/kg	HB-RISB-17	20/20	-	3.30E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL	
	7439-97-6	MERCURY ^b	0.72	11.3 J	mg/kg	HB-RISB-04	20/20	-	1.13E+01			2.35E+00	N	2.35E+01		2.35E+00	Y	ASL	
	22967-92-6	METHYL MERCURY	1.12	37.3 J	mg/kg	HB-RISB-07	12/12	-	3.73E+01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL	
	7440-02-0	NICKEL	9.1	104 J	mg/kg	HB-RISB-06	20/20	-	1.04E+02		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL	
	9/77440	POTASSIUM	535	3670 J	mg/kg	HB-HB-20D	19/19	-	3.67E+03			NV	NV	NV	N	NUT			
	7782-49-2	SELENIUM	1.4 J	4.1 J	mg/kg	HB-RISB-07	3/15	0.53-3.5	4.10E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-22-4	SILVER	1.7	4.3 J	mg/kg	HB-RISB-16	4/19	1.1-7.1	4.30E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-23-5	SODIUM	154	12800 J	mg/kg	HB-RISB-05	20/20	-	1.28E+04			NV	NV	NV	N	NUT			
	7440-62-2	VANADIUM	7.5	40.9 J	mg/kg	HB-HB-20D	17/19	20-35.4	4.09E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	64.3	823 J	mg/kg	HB-RISB-06	19/19	-	8.23E+02		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL	
	PCBs																		
		HIGHLY CHLORINATED PCBs ^c		0.24	4	mg/kg	HB-RISB-07	12/19	0.036-5.3	4.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
		TOTAL PCBs ^d		0.24	4	mg/kg	HB-RISB-07	12/19	0.036-5.3	4.00E+00			3.19E-01	C	2.22E-01	ca	2.22E-01	Y	ASL
	PESTICIDES																		
	72-54-8	4,4'-DDD	0.043 J	0.73 J	mg/kg	HB-HB-19S	4/20	0.0091-0.53	7.30E-01		2.60E+00	2.66E+00	C	2.44E+00	ca	2.44E+00	N	BSL	
	72-55-9	4,4'-DDE	0.076	0.11 J	mg/kg	HB-HB-19S	3/19	0.0097-0.53	1.10E-01		1.80E+00	1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL	
	50-29-3	4,4'-DDT	0.04 J	0.39 J	mg/kg	HB-HB-19S	3/19	0.0091-0.53	3.90E-01		1.70E+00	1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL	
	57-74-9	TOTAL CHLORDANE ^e	0.12 J	0.12 J	mg/kg	HB-HB-19S	1/19	0.0047-0.27	1.20E-01			1.82E+00	C	1.62E+00	ca	1.62E+00	N	BSL	
	60-57-1	DIELDRIN	0.11	0.11	mg/kg	HB-HB-18S	1/19	0.0091-0.53	1.10E-01		3.90E-02	3.99E-02	C	3.04E-02	ca	3.04E-02	Y	ASL	
	1024-57-3	HEPTACHLOR EPOXIDE	0.052 J	0.052 J	mg/kg	HB-HB-19S	1/19	0.0047-0.27	5.20E-02			7.02E-02	C	5.34E-02	ca	5.34E-02	N	BSL	
	SVOCs																		
	91-57-6	2-METHYLNAPHTHALENE	0.17 J	3.4 J	mg/kg	HB-RISB-16	10/19	5.5-33	3.40E+00			3.13E+01	N	NV		3.13E+01	N	BSL	
	83-32-9	ACENAPHTHENE	0.35 J	5.4 J	mg/kg	HB-RISB-16	13/19	5.5-32	5.40E+00		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL	
	208-96-8	ACENAPHTHYLENE	0.8 J	7.7 J	mg/kg	HB-HB-19S	17/19	12-32	7.70E+00		1.00E+02	NV	NV	NV	Y	NTX			
	120-12-7	ANTHRACENE	1.3 J	14	mg/kg	HB-RISB-16	18/20	12-32	1.40E+01		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL	
	56-55-3	BENZ(A)ANTHRACENE	1.2 J	32 J	mg/kg	HB-RISB-06	20/20	-	3.20E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	50-32-8	BENZO(A)PYRENE	2 J	32 J	mg/kg	HB-RISB-06	20/20	-	3.20E+01		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL	
	205-99-2	BENZO(B)FLUORANTHENE	1.9 J	27 J	mg/kg	HB-RISB-06	20/20	-	2.70E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	191-24-2	BENZO(G,H,I)PERYLENE	1.6 J	24 J	mg/kg	HB-RISB-06	20/20	-	2.40E+01		1.00E+02	NV	NV	NV	Y	NTX			
	207-08-9	BENZO(K)FLUORANTHENE	1.3 J	25 J	mg/kg	HB-RISB-06	20/20	-	2.50E+01		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00	Y	ASL	
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.72 J	3.7 J	mg/kg	HB-RISB-07	5/19	1.1-33	3.70E+00			4.56E+01	C	3.47E+01	ca	3.47E+01	N	BSL	
	86-74-8	CARBAZOLE	0.62 J	5.8 J	mg/kg	HB-RISB-06	10/19	1.1-32	5.80E+00			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL	
	218-01-9	CHRYSENE	1.8 J	34 J	mg/kg	HB-HB-19S	20/20	-	3.40E+01		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL	
	53-70-3	DIBENZ(A,H)ANTHRACENE	1.1	6.2 J	mg/kg	HB-HB-19S	15/19	12-33	6.20E+00		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL	
	132-64-9	DIBENZOFURAN	0.2 J	4.3 J	mg/kg	HB-RISB-16	9/19	3.6-33	4.30E+00		1.40E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	N	BSL	
	206-44-0	FLUORANTHENE	2.4 J	77 J	mg/kg	HB-HB-19S	20/20	-	7.70E+01		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL	
	86-73-7	FLUORENE	0.33 J	6.8 J	mg/kg	HB-HB-19S	14/20	5.5-33	6.80E+00		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL	
	118-74-1	HEXACHLORO BENZENE	0.93 J	2.4 J	mg/kg	HB-RISB-07	2/19	1.1-33	2.40E+00		3.30E-01	3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL	
	193-39-5	INDENO(1,2,3-CD)PYRENE	1.4 J	20 J	mg/kg	HB-RISB-06	19/20	32-32	2.00E+01		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	91-20-3	NAPHTHALENE	0.3 J	21 J	mg/kg	HB-RISB-04	18/20	5.5-32	2.10E+01		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL	
	85-01-8	PHENANTHRENE	1.3 J	61 J	mg/kg	HB-HB-19S	20/20	-	6.10E+01		1.00E+02	NV	NV	NV	Y	NTX			
	108-95-2	PHENOL	1.9 J	1.9 J	mg/kg	HB-RISB-16	1/19	1.1-33	1.90E+00		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL	
	129-00-0	PYRENE	2.3 J	59 J	mg/kg	HB-HB-19S	20/20	-	5.90E+01		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL	

TABLE 2.31a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL HARBOR BROOK SITE- AOS #1 SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	VOCs																	
	120-82-1	1,2,4-TRICHLOROBENZENE	1 J	8.7 J	mg/kg	HB-RISB-04	8/19	1.1-33	8.70E+00			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	2.3 J	14 J	mg/kg	HB-RISB-04	8/19	1.1-33	1.40E+01		1.00E+02	7.04E+02	N	6.00E+01	nc	6.00E+01	N	BSL
	541-73-1	1,3-DICHLOROBENZENE	0.61 J	2.2 J	mg/kg	HB-RISB-04	2/19	1.1-33	2.20E+00		1.70E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.98 J	38 J	mg/kg	HB-RISB-04	13/19	1.1-33	3.80E+01		9.80E+00	2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL
	78-93-3	2-BUTANONE	0.0075 J	0.047 J	mg/kg	HB-HB-20D	4/19	0.011-0.073	4.70E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.0077 J	0.18 J	mg/kg	HB-HB-20D	9/20	0.022-0.08	1.80E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.0048 J	0.017	mg/kg	HB-RISB-07	5/19	0.0054-0.036	1.70E-02		2.90E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	108-90-7	CHLOROBENZENE	0.0024 J	0.1	mg/kg	HB-RISB-07	12/20	0.0054-0.02	1.00E-01		1.00E+02	1.56E+02	N	1.51E+01	nc	1.51E+01	N	BSL
	156-59-2	CIS-1,2-DICHLOROETHENE	0.0026 J	0.0026 J	mg/kg	HB-HB-20D	1/19	0.0054-0.036	2.60E-03		5.90E+01	7.82E+01	N	4.29E+00	nc	4.29E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.0022 J	0.11 J	mg/kg	HB-RISB-05	5/20	0.0054-0.02	1.10E-01		3.00E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.0021 J	0.0052 J	mg/kg	HB-HB-20D	5/19	0.0069-0.036	5.20E-03		5.10E+01	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL
	100-42-5	STYRENE	0.0052 J	0.0052 J	mg/kg	HB-HB-20D	1/19	0.0054-0.036	5.20E-03			1.56E+03	N	1.70E+02	nc	1.70E+02	N	BSL
	127-18-4	TETRACHLOROETHENE	0.004 J	0.0085 J	mg/kg	HB-RISB-07	2/19	0.0054-0.036	8.50E-03		5.50E+00	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL
	108-88-3	TOLUENE	0.0046 J	0.028 J	mg/kg	HB-HB-20D	5/20	0.0054-0.036	2.80E-02		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL
	79-01-6	TRICHLOROETHENE	0.0071 J	0.0071 J	mg/kg	HB-RISB-07	1/19	0.0054-0.036	7.10E-03		1.00E+01	1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL
	1330-20-7	XYLENES, TOTAL	0.005 J	0.33 J	mg/Kg	HB-RISB-05	8/20	0.0054-0.02	3.30E-01		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL
		OTHER																
		112-40-3	DODECANE	590 J	1100 J	mg/kg	HB-RISB-05	2/4	450-640	1.10E+03			NV		NV		NV	Y

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
(5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.31b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for mercury compounds utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor 1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor-1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency
AOS #1: Additional Area of Study #1

TABLE 2.31b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HB-18S	8/29/2003	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	74.975	74.975	ng/kg		0.01	0.750
HB-HB-18S	8/29/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.765	8.765	ng/kg		0.01	0.088
HB-HB-18S	8/29/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.202	1.202	ng/kg	EMPC	0.01	0.012
HB-HB-18S	8/29/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.635	0.635	ng/kg	EMPC	0.1	0.064
HB-HB-18S	8/29/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.906	3.906	ng/kg	J	0.1	0.391
HB-HB-18S	8/29/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.43	3.43	ng/kg	J	0.1	0.343
HB-HB-18S	8/29/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.065	1.065	ng/kg	J	0.1	0.107
HB-HB-18S	8/29/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.306	3.306	ng/kg	J	0.1	0.331
HB-HB-18S	8/29/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-HB-18S	8/29/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.388	1.388	ng/kg	J	1	1.388
HB-HB-18S	8/29/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.863	1.863	ng/kg	EMPC	0.03	0.056
HB-HB-18S	8/29/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-HB-18S	8/29/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	5.016	5.016	ng/kg		0.1	0.502
HB-HB-18S	8/29/2003	0	0.5	39001-02-0	OCDF	Y	24.165	24.165	ng/kg		0.0003	0.007
Sample Location TEQ =											4.7	
HB-HB-18S	8/29/2003	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	229.416	229.416	ng/kg		0.01	2.294
HB-HB-18S	8/29/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	28.993	28.993	ng/kg		0.01	0.290
HB-HB-18S	8/29/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.052	4.052	ng/kg	J	0.01	0.041
HB-HB-18S	8/29/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.923	2.923	ng/kg	J	0.1	0.292
HB-HB-18S	8/29/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.597	12.597	ng/kg		0.1	1.260
HB-HB-18S	8/29/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	14.043	14.043	ng/kg		0.1	1.404
HB-HB-18S	8/29/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	4.201	4.201	ng/kg	J	0.1	0.420
HB-HB-18S	8/29/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	14.045	14.045	ng/kg		0.1	1.405
HB-HB-18S	8/29/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-HB-18S	8/29/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	5.849	5.849	ng/kg		1	5.849
HB-HB-18S	8/29/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	9.707	9.707	ng/kg	J	0.03	0.291
HB-HB-18S	8/29/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.49	1.49	ng/kg	J	1	1.490
HB-HB-18S	8/29/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	19.27	19.27	ng/kg		0.1	1.927
HB-HB-18S	8/29/2003	0.5	1	3268-87-9	OCDD	Y	1373.023	1373.023	ng/kg		0.0003	0.412
HB-HB-18S	8/29/2003	0.5	1	39001-02-0	OCDF	Y	49.114	49.114	ng/kg		0.0003	0.015
Sample Location TEQ =											17.5	
HB-HB-20D	8/29/2003	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	40.629	40.629	ng/kg	J	0.01	0.406
HB-HB-20D	8/29/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	5.857	5.857	ng/kg	J	0.01	0.059
HB-HB-20D	8/29/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.717	0.717	ng/kg	J	0.01	0.007
HB-HB-20D	8/29/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-HB-20D	8/29/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.939	1.939	ng/kg	J	0.1	0.194
HB-HB-20D	8/29/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.904	1.904	ng/kg	J	0.1	0.190
HB-HB-20D	8/29/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.648	0.648	ng/kg	J	0.1	0.065
HB-HB-20D	8/29/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.276	1.276	ng/kg	J	0.1	0.128
HB-HB-20D	8/29/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-HB-20D	8/29/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-HB-20D	8/29/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.077	1.077	ng/kg	J	0.03	0.032
HB-HB-20D	8/29/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-HB-20D	8/29/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.641	2.641	ng/kg	J	0.1	0.264
HB-HB-20D	8/29/2003	0	0.5	3268-87-9	OCDD	Y	293.433	293.433	ng/kg	J	0.0003	0.088
HB-HB-20D	8/29/2003	0	0.5	39001-02-0	OCDF	Y	14.539	14.539	ng/kg	J	0.0003	0.004
Sample Location TEQ =											3.4	

TABLE 2.31b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HB-20D	8/29/2003	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	282.196	282.196	ng/kg	J	0.01	2.822
HB-HB-20D	8/29/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	35.287	35.287	ng/kg	J	0.01	0.353
HB-HB-20D	8/29/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.493	4.493	ng/kg	EMPC	0.01	0.045
HB-HB-20D	8/29/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.131	3.131	ng/kg	J	0.1	0.313
HB-HB-20D	8/29/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.268	12.268	ng/kg	J	0.1	1.227
HB-HB-20D	8/29/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	16.879	16.879	ng/kg	J	0.1	1.688
HB-HB-20D	8/29/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.902	3.902	ng/kg	EMPC	0.1	0.390
HB-HB-20D	8/29/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	15.445	15.445	ng/kg	J	0.1	1.545
HB-HB-20D	8/29/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-HB-20D	8/29/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	5.975	5.975	ng/kg	J	1	5.975
HB-HB-20D	8/29/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	8.695	8.695	ng/kg	J	0.03	0.261
HB-HB-20D	8/29/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.54	1.54	ng/kg	EMPC	1	1.540
HB-HB-20D	8/29/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	14.863	14.863	ng/kg	J	0.1	1.486
HB-HB-20D	8/29/2003	0.5	1	3268-87-9	OCDD	Y	1686.851	1686.851	ng/kg	J	0.0003	0.506
HB-HB-20D	8/29/2003	0.5	1	39001-02-0	OCDF	Y	84.01	84.01	ng/kg	J	0.0003	0.025
Sample Location TEQ =												18.3
HB-RISB-11	8/29/2003	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	34.672	34.672	ng/kg		0.01	0.347
HB-RISB-11	8/29/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	16.787	16.787	ng/kg		0.01	0.168
HB-RISB-11	8/29/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.618	1.618	ng/kg	J	0.01	0.016
HB-RISB-11	8/29/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-11	8/29/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	8.172	8.172	ng/kg		0.1	0.817
HB-RISB-11	8/29/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.436	5.436	ng/kg		0.1	0.544
HB-RISB-11	8/29/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.426	2.426	ng/kg	EMPC	0.1	0.243
HB-RISB-11	8/29/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.426	3.426	ng/kg	EMPC	0.1	0.343
HB-RISB-11	8/29/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.195	1.195	ng/kg	J	0.1	0.120
HB-RISB-11	8/29/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-RISB-11	8/29/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.018	4.018	ng/kg	J	0.03	0.121
HB-RISB-11	8/29/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-RISB-11	8/29/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	12.767	12.767	ng/kg		0.1	1.277
HB-RISB-11	8/29/2003	0	0.5	3268-87-9	OCDD	Y	196.43	196.43	ng/kg	J	0.0003	0.059
HB-RISB-11	8/29/2003	0	0.5	39001-02-0	OCDF	Y	22.011	22.011	ng/kg		0.0003	0.007
Sample Location TEQ =												5.9

TABLE 2.31b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)	
HB-RISB-11	8/29/2003	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	24.416	24.416	ng/kg	EMPC	0.01	0.244	
HB-RISB-11	8/29/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	11.65	11.65	ng/kg		0.01	0.117	
HB-RISB-11	8/29/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.033	1.033	ng/kg		0.01	0.010	
HB-RISB-11	8/29/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.488	0.488	ng/kg		J	0.1	0.049
HB-RISB-11	8/29/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	5.198	5.198	ng/kg			0.1	0.520
HB-RISB-11	8/29/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.881	3.881	ng/kg	J	0.1	0.388	
HB-RISB-11	8/29/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.82	1.82	ng/kg			0.1	0.182
HB-RISB-11	8/29/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.16	2.16	ng/kg	J	0.1	0.216	
HB-RISB-11	8/29/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125	
HB-RISB-11	8/29/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	0.927	0.927	ng/kg	J	1	0.927	
HB-RISB-11	8/29/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	2.402	2.402	ng/kg	J	0.03	0.072	
HB-RISB-11	8/29/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500	
HB-RISB-11	8/29/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	4.103	4.103	ng/kg		0.1	0.410	
HB-RISB-11	8/29/2003	0.5	1	39001-02-0	OCDF	Y	14.329	14.329	ng/kg			0.0003	0.004
Sample Location TEQ =												3.8	

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

EMPC = Estimated Maximum Possible Concentration

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223-241.

TABLE 2.31c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL HARBOR BROOK SITE- AOS #1 SURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-HB-18S	0	0.5	8/29/2003	0.74	mg/kg
Highly Chlorinated PCBs	HB-HB-18S	0.5	1	8/29/2003	3.9	mg/kg
Highly Chlorinated PCBs	HB-HB-19S	0	0.5	12/30/2002	0.24	mg/kg
Highly Chlorinated PCBs	HB-HB-19S	0.5	1	12/30/2002	1	mg/kg
Highly Chlorinated PCBs	HB-HB-20D	0	0.5	8/29/2003	1.7	mg/kg
Highly Chlorinated PCBs	HB-HB-20D	0.5	1	8/29/2003	2.6	mg/kg
Highly Chlorinated PCBs	HB-RISB-05	0.5	1	1/2/2003	1.39	mg/kg
Highly Chlorinated PCBs	HB-RISB-07	0	0.5	12/17/2002	4	mg/kg
Highly Chlorinated PCBs	HB-RISB-16	0	0.5	5/25/2004	0.49	mg/kg
Highly Chlorinated PCBs	HB-RISB-16	0.5	1	5/25/2004	0.39	mg/kg
Highly Chlorinated PCBs	HB-RISB-17	0	0.5	5/25/2004	0.44	mg/kg
Highly Chlorinated PCBs	HB-RISB-17	0.5	1	5/25/2004	0.75	mg/kg
Total PCBs	HB-HB-18S	0	0.5	8/29/2003	0.74	mg/kg
Total PCBs	HB-HB-18S	0.5	1	8/29/2003	3.9	mg/kg
Total PCBs	HB-HB-19S	0	0.5	12/30/2002	0.24	mg/kg
Total PCBs	HB-HB-19S	0.5	1	12/30/2002	1	mg/kg
Total PCBs	HB-HB-20D	0	0.5	8/29/2003	1.7	mg/kg
Total PCBs	HB-HB-20D	0.5	1	8/29/2003	2.6	mg/kg
Total PCBs	HB-RISB-05	0.5	1	1/2/2003	1.39	mg/kg
Total PCBs	HB-RISB-07	0	0.5	12/17/2002	4	mg/kg
Total PCBs	HB-RISB-16	0	0.5	5/25/2004	0.49	mg/kg
Total PCBs	HB-RISB-16	0.5	1	5/25/2004	0.39	mg/kg
Total PCBs	HB-RISB-17	0	0.5	5/25/2004	0.44	mg/kg
Total PCBs	HB-RISB-17	0.5	1	5/25/2004	0.75	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.31d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-18S	8/29/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-HB-18S	8/29/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-HB-18S	8/29/2003	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.031
HB-HB-18S	8/29/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.031
Total Chlordane =									ND
HB-HB-19S	12/30/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.011
HB-HB-19S	12/30/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.011
Total Chlordane =									ND
HB-HB-19S	12/30/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.015
HB-HB-19S	12/30/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.12
Total Chlordane =									0.12
HB-HB-20D	8/29/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.052
HB-HB-20D	8/29/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.052
Total Chlordane =									ND
HB-HB-20D	8/29/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.04
HB-HB-20D	8/29/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.04
Total Chlordane =									ND
HB-RISB-04	12/18/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.16
HB-RISB-04	12/18/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.16
Total Chlordane =									ND
HB-RISB-04	12/18/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.13
HB-RISB-04	12/18/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.13
Total Chlordane =									ND
HB-RISB-05	1/2/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.025
HB-RISB-05	1/2/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.025
Total Chlordane =									ND
HB-RISB-06	12/18/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.27
HB-RISB-06	12/18/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.27
Total Chlordane =									ND
HB-RISB-06	12/18/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.27
HB-RISB-06	12/18/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.27
Total Chlordane =									ND
HB-RISB-07	12/17/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.19
HB-RISB-07	12/17/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.19
Total Chlordane =									ND
HB-RISB-07	12/17/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.089
HB-RISB-07	12/17/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.089
Total Chlordane =									ND
HB-RISB-11	8/29/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.019
HB-RISB-11	8/29/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.019
Total Chlordane =									ND
HB-RISB-11	8/29/2003	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.018
HB-RISB-11	8/29/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.018
Total Chlordane =									ND
HB-RISB-16	5/25/2004	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.005
HB-RISB-16	5/25/2004	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.005
Total Chlordane =									ND

TABLE 2.31d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-RISB-16	5/25/2004	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0054
HB-RISB-16	5/25/2004	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0054
Total Chlordane =									ND
HB-RISB-17	5/25/2004	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0047
HB-RISB-17	5/25/2004	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0047
Total Chlordane =									ND
HB-RISB-17	5/25/2004	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0054
HB-RISB-17	5/25/2004	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0054
Total Chlordane =									ND

TABLE 2.31e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-18S	8/29/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0088	
HB-HB-18S	8/29/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0088	
HB-HB-18S	8/29/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0088
HB-HB-18S	8/29/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0033	
HB-HB-18S	8/29/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.0018	
HB-HB-18S	8/29/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0051
HB-HB-19S	12/30/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.017	
HB-HB-19S	12/30/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.017	
HB-HB-19S	12/30/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.017
HB-HB-19S	12/30/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.011	
HB-HB-19S	12/30/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.011	
HB-HB-19S	12/30/2002	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.011
HB-HB-20D	8/29/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.015	
HB-HB-20D	8/29/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.015	
HB-HB-20D	8/29/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.015
HB-HB-20D	8/29/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.014	
HB-HB-20D	8/29/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.0045	
HB-HB-20D	8/29/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0185
HB-RISB-04	12/18/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0084	
HB-RISB-04	12/18/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.012	
HB-RISB-04	12/18/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.0084
HB-RISB-04	12/18/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.005	
HB-RISB-04	12/18/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0096	
HB-RISB-04	12/18/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.005
HB-RISB-05	1/2/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.22	
HB-RISB-05	1/2/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.11	
HB-RISB-05	1/2/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.33
HB-RISB-05	1/2/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.036	
HB-RISB-05	1/2/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.008	
HB-RISB-05	1/2/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.008
HB-RISB-06	12/18/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.02
HB-RISB-06	12/18/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.02
HB-RISB-07	12/17/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.018	
HB-RISB-07	12/17/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.03	
HB-RISB-07	12/17/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.018
HB-RISB-07	12/17/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.012	
HB-RISB-07	12/17/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.011	
HB-RISB-07	12/17/2002	0.5	1	CALCULATED	TOTAL	Y		mg/kg		0.012
HB-RISB-11	8/29/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0055	
HB-RISB-11	8/29/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0055	
HB-RISB-11	8/29/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0055
HB-RISB-11	8/29/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0054	
HB-RISB-11	8/29/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0054	
HB-RISB-11	8/29/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0054
HB-RISB-16	5/25/2004	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0074	
HB-RISB-16	5/25/2004	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0074	
HB-RISB-16	5/25/2004	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0074
HB-RISB-16	5/25/2004	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0079	
HB-RISB-16	5/25/2004	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0079	
HB-RISB-16	5/25/2004	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0079

TABLE 2.31e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-RISB-17	5/25/2004	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0069	
HB-RISB-17	5/25/2004	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0069	
HB-RISB-17	5/25/2004	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0069
HB-RISB-17	5/25/2004	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0079	
HB-RISB-17	5/25/2004	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0079	
HB-RISB-17	5/25/2004	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0079

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.32a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
AOS #1 Subsurface Soil	DIOXON/FURAN (9)																		
	1746-01-6	2,3,7,8-TCDD Equivalent	0.000003	0.00002	mg/Kg	HB-HB-20D	6/6		1.83E-05			4.26E-06	C	3.90E-06	ca	3.90E-06	Y	ASL	
	METALS																		
	7429-90-5	ALUMINUM	910	14300 J	mg/Kg	HB-HB-20D	25/25	-	1.43E+04			7.82E+03	N	7.61E+03	nc	7.61E+03	Y	ASL	
	7440-36-0	ANTIMONY	0.26 J	0.26 J	mg/Kg	HB-SB-68	1/24	6.4-42.5	2.60E-01			3.13E+00	N	3.13E+00	nc	3.13E+00	N	BSL	
	7440-38-2	ARSENIC	2.1	14 J	mg/Kg	HB-RISB-04	22/24	1.9-7.1	1.40E+01		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX	
	7440-39-3	BARIIUM	46.2	389 J	mg/Kg	HB-HB-20D	25/25	-	3.89E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL	
	7440-41-7	BERYLLIUM	0.09 J	0.44 J	mg/Kg	HB-SB-67	3/24	0.53-3.5	4.40E-01		1.40E+01	1.56E+01	N	1.54E+01	nc	1.54E+01	N	BSL	
	7440-43-9	CADMIUM	0.27 J	9.9 J	mg/Kg	HB-RISB-07	17/24	0.53-3.5	9.90E+00		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00	Y	ASL	
	7440-70-2	CALCIUM	23000 J	340000	mg/Kg	HB-SB-66	25/25	-	3.40E+05			NV	NV	NV	NV	N	NUT		
	7440-47-3	CHROMIUM ^a	4	191 J	mg/Kg	HB-HB-20D	25/25	-	1.91E+02			2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX	
	7440-48-4	COBALT	0.55 J	8.9	mg/Kg	HB-RISB-11	5/24	6.28-35.4	8.90E+00			NV	NV	9.03E+01	nc	9.03E+01	N	BSL	
	7440-50-8	COPPER	5.4	302 J	mg/Kg	HB-HB-19S	25/25	-	3.02E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL	
	57-12-5	CYANIDE	1.8 J	1.8 J	mg/Kg	HB-SB-67	1/24	0.56-7.3	1.80E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL	
	7439-89-6	IRON	3000	22300 J	mg/Kg	HB-HB-19S	25/25	-	2.23E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	8.4	2320 J	mg/Kg	HB-HB-20D	25/25	-	2.32E+03			NV	NV	4.00E+02	nc	4.00E+02	Y	ASL	
	7439-95-4	MAGNESIUM	2880 J	25000	mg/Kg	HB-SB-68	25/25	-	2.50E+04			NV	NV	NV	NV	N	NUT		
	7439-96-5	MANGANESE	60.6 J	460	mg/Kg	HB-SB-66	25/25	-	4.60E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL	
	7439-97-6	MERCURY ^b	0.19 J	11.3 J	mg/kg	HB-RISB-04	25/25	-	1.13E+01			2.35E+00	N	2.35E+00	nc	2.35E+00	Y	ASL	
	22967-92-6	METHYL MERCURY	0.00112	0.0373 J	ug/kg	HB-RISB-07	12/12	-	3.73E-02			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL	
	7440-02-0	NICKEL	3.4 J	104 J	mg/Kg	HB-RISB-06	25/25	-	1.04E+02		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL	
	7440-09-7	POTASSIUM	280 J	3670 J	mg/Kg	HB-HB-20D	24/24	-	3.67E+03			NV	NV	NV	NV	N	NUT		
	7782-49-2	SELENIUM	0.87 J	4.1 J	mg/Kg	HB-RISB-07	4/20	0.53-3.5	4.10E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-22-4	SILVER	0.47 J	4.3 J	mg/Kg	HB-RISB-16	5/24	1.1-7.1	4.30E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01	N	BSL	
	7440-23-5	SODIUM	154	12800 J	mg/Kg	HB-RISB-05	25/25	-	1.28E+04			NV	NV	NV	NV	N	NUT		
	7440-62-2	VANADIUM	2.3 J	40.9 J	mg/Kg	HB-HB-20D	22/24	20-35.4	4.09E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	37	823 J	mg/Kg	HB-RISB-06	23/24	14-14	8.23E+02		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL	
	PCBs																		
			HIGHLY CHLORINATED PCBs ^c	0.24	4	mg/kg	HB-RISB-07	13/24	0.019-5.3	4.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
			TOTAL PCBs ^d	0.24	4	mg/kg	HB-RISB-07	13/24	0.019-5.3	4.00E+00			3.19E-01	C	2.22E-02	nc	2.22E-02	Y	ASL
	PESTICIDES																		
	72-54-8	4,4'-DDD	0.043 J	0.73 J	mg/kg	HB-HB-19S	4/25	0.0037-0.53	7.30E-01			2.60E+00	2.66E+00	C	2.44E+00	ca	2.44E+00	N	BSL
	72-55-9	4,4'-DDE	0.076	0.11 J	mg/kg	HB-HB-19S	3/24	0.0037-0.53	1.10E-01			1.80E+00	1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL
	50-29-3	4,4'-DDT	0.04 J	0.39 J	mg/kg	HB-HB-19S	3/24	0.0037-0.53	3.90E-01			1.70E+00	1.88E+00	C	1.72E+00	ca	1.72E+00	N	BSL
	57-74-9	TOTAL CHLORDANE ^e	0.12 J	0.12 J	mg/kg	HB-HB-19S	1/19	0.0047-0.27	1.20E-01			1.82E+00	C	1.62E+00	ca	1.62E+00	N	BSL	
	60-57-1	DIELDRIN	0.11	0.11	mg/kg	HB-HB-18S	1/24	0.0037-0.53	1.10E-01		3.90E-02	3.99E-02	C	3.04E-02	ca	3.04E-02	Y	ASL	
	1024-57-3	HEPTACHLOR EPOXIDE	0.052 J	0.052 J	mg/kg	HB-HB-19S	1/24	0.0019-0.27	5.20E-02			7.02E-02	C	5.34E-02	ca	5.34E-02	N	BSL	
	SVOCs																		
	92-52-4	1,1'-BIPHENYL	0.038 J	28 J	mg/kg	HB-SB-66	3/3	-	2.80E+01			3.91E+02	N	3.01E+02	nc	3.01E+02	N	BSL	
	91-57-6	2-METHYLNAPHTHALENE	0.16 J	140	mg/kg	HB-SB-66	15/24	5.5-33	1.40E+02			3.13E+01	N	NV	nc	3.13E+01	Y	ASL	
	106-44-5	4-METHYLPHENOL	0.39 J	0.39 J	mg/kg	HB-SB-67	1/3	0.37-62	3.90E-01		3.40E+01	3.91E+01	N	3.06E+01	nc	3.06E+01	N	BSL	
	83-32-9	ACENAPHTHENE	0.35 J	33 J	mg/kg	HB-SB-66	17/24	0.37-32	3.30E+01		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL	
	208-96-8	ACENAPHTHYLENE	0.12 J	31 J	mg/kg	HB-SB-66	21/24	2.1-32	3.10E+01		1.00E+02	NV	NV	NV	NV	Y	NTX		
	120-12-7	ANTHRACENE	0.13 J	510	mg/kg	HB-RISB-16	23/25	12-32	5.10E+02		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL	
	56-55-3	BENZ(A)ANTHRACENE	0.6	63	mg/kg	HB-RISB-16	25/25	-	6.30E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	50-32-8	BENZO(A)PYRENE	0.61	56	mg/kg	HB-RISB-16	24/25	62-62	5.60E+01		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL	

TABLE 2.32a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	205-99-2	BENZO(B)FLUORANTHENE	1	35 J	mg/kg	HB-RISB-16	24/25	62-62	3.50E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.28 J	27 J	mg/kg	HB-RISB-16	24/25	62-62	2.70E+01		1.00E+02	NV		NV		NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.37 J	37	mg/kg	HB-RISB-16	24/25	62-62	3.70E+01		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00	Y	ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.72 J	3.7 J	mg/kg	HB-RISB-07	6/24	1.1-62	3.70E+00			4.56E+01	C	3.47E+01	ca	3.47E+01	N	BSL
	105-60-2	CAPROLACTAM	0.75	0.75	mg/kg	HB-SB-68	1/3	2.1-62	7.50E-01			3.91E+03	N	3.06E+03	nc	3.06E+03	N	BSL
	86-74-8	CARBAZOLE	0.059 J	10 J	mg/kg	HB-SB-66	15/24	1.1-32	1.00E+01			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL
	218-01-9	CHRYSENE	0.73	74	mg/kg	HB-RISB-16	25/25	-	7.40E+01		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.1 J	9.5 J	mg/kg	HB-RISB-16	18/24	2.1-62	9.50E+00		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	132-64-9	DIBENZOFURAN	0.08 J	51 J	mg/kg	HB-SB-66	14/24	3.6-33	5.10E+01		1.40E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL
	206-44-0	FLUORANTHENE	0.88	110	mg/kg	HB-RISB-16	25/25	-	1.10E+02		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	86-73-7	FLUORENE	0.064 J	59 J	mg/kg	HB-SB-66	19/25	5.5-33	5.90E+01		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	118-74-1	HEXACHLOROBENZENE	0.93 J	2.4 J	mg/kg	HB-RISB-07	2/24	0.37-62	2.40E+00		3.30E-01	3.99E-01	C	3.04E-01	ca	3.04E-01	Y	ASL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.22 J	24 J	mg/kg	HB-RISB-16	23/25	32-62	2.40E+01		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	91-20-3	NAPHTHALENE	0.3 J	570	mg/kg	HB-SB-66	23/25	5.5-32	5.70E+02		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL
	85-01-8	PHENANTHRENE	0.47	120	mg/kg	HB-SB-66	25/25	-	1.20E+02		1.00E+02	NV		NV		NV	Y	NTX
	108-95-2	PHENOL	0.25 J	1.9 J	mg/kg	HB-RISB-16	2/24	0.37-62	1.90E+00		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL
	129-00-0	PYRENE	0.84	93	mg/kg	HB-RISB-16	25/25	-	9.30E+01		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																	
	120-82-1	1,2,4-TRICHLOROBENZENE	1 J	8.7 J	mg/kg	HB-RISB-04	8/24	0.0056-36	8.70E+00			7.82E+01	N	6.22E+00	nc	6.22E+00	Y	ASL
	95-50-1	1,2-DICHLOROBENZENE	2.3 J	14 J	mg/kg	HB-RISB-04	8/24	0.0028-36	1.40E+01		1.00E+02	7.04E+02	N	6.00E+01	nc	6.00E+01	N	BSL
541-73-1	1,3-DICHLOROBENZENE	0.61 J	2.2 J	mg/kg	HB-RISB-04	2/24	0.0028-36	2.20E+00		1.70E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL	
106-46-7	1,4-DICHLOROBENZENE	0.98 J	38 J	mg/kg	HB-RISB-04	13/24	0.0028-36	3.80E+01		9.80E+00	2.66E+01	C	3.45E+00	ca	3.45E+00	Y	ASL	
78-93-3	2-BUTANONE	0.0042 J	0.073 J	mg/kg	HB-SB-67	8/24	0.011-3.7	7.30E-02		1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL	
67-64-1	ACETONE	0.0077 J	0.25	mg/kg	HB-SB-67	12/25	0.022-3.7	2.50E-01		1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL	
71-43-2	BENZENE	0.0013 J	1.6 J	mg/kg	HB-RISB-17	10/24	0.0054-0.036	1.60E+00		2.90E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX	
75-15-0	CARBON DISULFIDE	0.0017 J	0.0067 J	mg/kg	HB-RISB-16	4/24	0.011-0.94	6.70E-03			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL	
108-90-7	CHLOROBENZENE	0.0023 J	0.1	mg/kg	HB-RISB-07	14/25	0.0028-0.94	1.00E-01		1.00E+02	1.56E+02	N	1.51E+01	nc	1.51E+01	N	BSL	
156-59-2	CIS-1,2-DICHLOROETHENE	0.0026 J	0.0026 J	mg/kg	HB-HB-20D	1/24	0.0028-0.94	2.60E-03		5.90E+01	7.82E+01	N	4.29E+00	nc	4.29E+00	N	BSL	
110-82-7	CYCLOHEXANE	0.0023 J	0.0023 J	mg/kg	HB-SB-67	1/3	0.0028-0.94	2.30E-03			NV		1.40E+01	nc	1.40E+01	N	BSL	
100-41-4	ETHYLBENZENE	0.001 J	5.7	mg/kg	HB-SB-66	10/25	0.0054-0.02	5.70E+00		3.00E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL	
98-82-8	ISOPROPYLBENZENE	0.018	3.4	mg/kg	HB-SB-66	2/3	0.0028-0.0028	3.40E+00			7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL	
108-87-2	METHYLCYCLOHEXANE	0.0055	0.54 J	mg/kg	HB-SB-66	2/3	0.0028-0.0028	5.40E-01			NV		2.59E+02	nc	2.59E+02	N	BSL	
75-09-2	METHYLENE CHLORIDE	0.0021 J	0.0052 J	mg/kg	HB-HB-20D	5/24	0.0056-1.9	5.20E-03		5.10E+01	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL	
100-42-5	STYRENE	0.0052 J	1.7 J	mg/kg	HB-RISB-17	2/24	0.0028-0.94	1.70E+00			1.56E+03	N	1.70E+02	nc	1.70E+02	N	BSL	
127-18-4	TETRACHLOROETHENE	0.004 J	0.0085 J	mg/kg	HB-RISB-07	2/24	0.0028-0.94	8.50E-03		5.50E+00	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL	
108-88-3	TOLUENE	0.0023 J	5.3 J	mg/kg	HB-RISB-17	10/25	0.0054-0.036	5.30E+00		1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL	
79-01-6	TRICHLOROETHENE	0.0071 J	0.0071 J	mg/kg	HB-RISB-07	1/24	0.0028-0.94	7.10E-03		1.00E+01	1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL	
1330-20-7	XYLENES, TOTAL	0.005	29	mg/kg	HB-SB-66	13/25	0.0054-0.02	2.90E+01		1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	Y	ASL	
OTHER																		
112-40-3	DODECANE	590 J	1100 J	mg/kg	HB-RISB-04	2/4	450-640	1.10E+03			NV		NV		NV	Y	NTX	

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) Values are from New York Subpart 375-6 Soil Cleanup Objectives. Values reflect residential restricted use for the protection of human health.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
 - (9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.32b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for mercury compounds utilized.
c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
USEPA: United States Environmental Protection Agency
AOS #1: Additional Area of Study #1

TABLE 2.32b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HB-18S	8/29/2003	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	74.975	74.975	ng/kg		0.01	0.750
HB-HB-18S	8/29/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.765	8.765	ng/kg		0.01	0.088
HB-HB-18S	8/29/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.202	1.202	ng/kg	EMPC	0.01	0.012
HB-HB-18S	8/29/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.635	0.635	ng/kg	EMPC	0.1	0.064
HB-HB-18S	8/29/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.906	3.906	ng/kg	J	0.1	0.391
HB-HB-18S	8/29/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.43	3.43	ng/kg	J	0.1	0.343
HB-HB-18S	8/29/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.065	1.065	ng/kg	J	0.1	0.107
HB-HB-18S	8/29/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.306	3.306	ng/kg	J	0.1	0.331
HB-HB-18S	8/29/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-HB-18S	8/29/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.388	1.388	ng/kg	J	1	1.388
HB-HB-18S	8/29/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.863	1.863	ng/kg	EMPC	0.03	0.056
HB-HB-18S	8/29/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-HB-18S	8/29/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	5.016	5.016	ng/kg		0.1	0.502
HB-HB-18S	8/29/2003	0	0.5	39001-02-0	OCDF	Y	24.165	24.165	ng/kg		0.0003	0.007
Sample Location TEQ =												4.7
HB-HB-18S	8/29/2003	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	229.416	229.416	ng/kg		0.01	2.294
HB-HB-18S	8/29/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	28.993	28.993	ng/kg		0.01	0.290
HB-HB-18S	8/29/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.052	4.052	ng/kg	J	0.01	0.041
HB-HB-18S	8/29/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.923	2.923	ng/kg	J	0.1	0.292
HB-HB-18S	8/29/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.597	12.597	ng/kg		0.1	1.260
HB-HB-18S	8/29/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	14.043	14.043	ng/kg		0.1	1.404
HB-HB-18S	8/29/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	4.201	4.201	ng/kg	J	0.1	0.420
HB-HB-18S	8/29/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	14.045	14.045	ng/kg		0.1	1.405
HB-HB-18S	8/29/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-HB-18S	8/29/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	5.849	5.849	ng/kg		1	5.849
HB-HB-18S	8/29/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	9.707	9.707	ng/kg	J	0.03	0.291
HB-HB-18S	8/29/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.49	1.49	ng/kg	J	1	1.490
HB-HB-18S	8/29/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	19.27	19.27	ng/kg		0.1	1.927
HB-HB-18S	8/29/2003	0.5	1	3268-87-9	OCDD	Y	1373.023	1373.023	ng/kg		0.0003	0.412
HB-HB-18S	8/29/2003	0.5	1	39001-02-0	OCDF	Y	49.114	49.114	ng/kg		0.0003	0.015
Sample Location TEQ =												17.5
HB-HB-20D	8/29/2003	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	40.629	40.629	ng/kg	J	0.01	0.406
HB-HB-20D	8/29/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	5.857	5.857	ng/kg	J	0.01	0.059
HB-HB-20D	8/29/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.717	0.717	ng/kg	J	0.01	0.007
HB-HB-20D	8/29/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-HB-20D	8/29/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.939	1.939	ng/kg	J	0.1	0.194
HB-HB-20D	8/29/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.904	1.904	ng/kg	J	0.1	0.190
HB-HB-20D	8/29/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.648	0.648	ng/kg	J	0.1	0.065
HB-HB-20D	8/29/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.276	1.276	ng/kg	J	0.1	0.128
HB-HB-20D	8/29/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-HB-20D	8/29/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-HB-20D	8/29/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.077	1.077	ng/kg	J	0.03	0.032
HB-HB-20D	8/29/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	UJ	1	0.500
HB-HB-20D	8/29/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.641	2.641	ng/kg	J	0.1	0.264
HB-HB-20D	8/29/2003	0	0.5	3268-87-9	OCDD	Y	293.433	293.433	ng/kg	J	0.0003	0.088
HB-HB-20D	8/29/2003	0	0.5	39001-02-0	OCDF	Y	14.539	14.539	ng/kg	J	0.0003	0.004
Sample Location TEQ =												3.4

TABLE 2.32b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HB-20D	8/29/2003	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	282.196	282.196	ng/kg	J	0.01	2.822
HB-HB-20D	8/29/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	35.287	35.287	ng/kg	J	0.01	0.353
HB-HB-20D	8/29/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.493	4.493	ng/kg	EMPC	0.01	0.045
HB-HB-20D	8/29/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.131	3.131	ng/kg	J	0.1	0.313
HB-HB-20D	8/29/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.268	12.268	ng/kg	J	0.1	1.227
HB-HB-20D	8/29/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	16.879	16.879	ng/kg	J	0.1	1.688
HB-HB-20D	8/29/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.902	3.902	ng/kg	EMPC	0.1	0.390
HB-HB-20D	8/29/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	15.445	15.445	ng/kg	J	0.1	1.545
HB-HB-20D	8/29/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	UJ	0.1	0.125
HB-HB-20D	8/29/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	5.975	5.975	ng/kg	J	1	5.975
HB-HB-20D	8/29/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	8.695	8.695	ng/kg	J	0.03	0.261
HB-HB-20D	8/29/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.54	1.54	ng/kg	EMPC	1	1.540
HB-HB-20D	8/29/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	14.863	14.863	ng/kg	J	0.1	1.486
HB-HB-20D	8/29/2003	0.5	1	3268-87-9	OCDD	Y	1686.851	1686.851	ng/kg	J	0.0003	0.506
HB-HB-20D	8/29/2003	0.5	1	39001-02-0	OCDF	Y	84.01	84.01	ng/kg	J	0.0003	0.025
Sample Location TEQ =												18.3
HB-RISB-11	8/29/2003	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	34.672	34.672	ng/kg		0.01	0.347
HB-RISB-11	8/29/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	16.787	16.787	ng/kg		0.01	0.168
HB-RISB-11	8/29/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.618	1.618	ng/kg	J	0.01	0.016
HB-RISB-11	8/29/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-11	8/29/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	8.172	8.172	ng/kg		0.1	0.817
HB-RISB-11	8/29/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	5.436	5.436	ng/kg		0.1	0.544
HB-RISB-11	8/29/2003	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.426	2.426	ng/kg	EMPC	0.1	0.243
HB-RISB-11	8/29/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	3.426	3.426	ng/kg	EMPC	0.1	0.343
HB-RISB-11	8/29/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	1.195	1.195	ng/kg	J	0.1	0.120
HB-RISB-11	8/29/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	2.5	1.25	ng/kg	UJ	1	1.250
HB-RISB-11	8/29/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.018	4.018	ng/kg	J	0.03	0.121
HB-RISB-11	8/29/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-RISB-11	8/29/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	12.767	12.767	ng/kg		0.1	1.277
HB-RISB-11	8/29/2003	0	0.5	3268-87-9	OCDD	Y	196.43	196.43	ng/kg	J	0.0003	0.059
HB-RISB-11	8/29/2003	0	0.5	39001-02-0	OCDF	Y	22.011	22.011	ng/kg		0.0003	0.007
Sample Location TEQ =												5.9

TABLE 2.32b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-RISB-11	8/29/2003	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	24.416	24.416	ng/kg	EMPC J	0.01	0.244
HB-RISB-11	8/29/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	11.65	11.65	ng/kg		0.01	0.117
HB-RISB-11	8/29/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.033	1.033	ng/kg		0.01	0.010
HB-RISB-11	8/29/2003	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.488	0.488	ng/kg		0.1	0.049
HB-RISB-11	8/29/2003	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	5.198	5.198	ng/kg	J J U U	0.1	0.520
HB-RISB-11	8/29/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.881	3.881	ng/kg		0.1	0.388
HB-RISB-11	8/29/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.82	1.82	ng/kg		0.1	0.182
HB-RISB-11	8/29/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.16	2.16	ng/kg		0.1	0.216
HB-RISB-11	8/29/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	2.5	1.25	ng/kg	U	0.1	0.125
HB-RISB-11	8/29/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	0.927	0.927	ng/kg	J	1	0.927
HB-RISB-11	8/29/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	2.402	2.402	ng/kg	J	0.03	0.072
HB-RISB-11	8/29/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	1	0.5	ng/kg	U	1	0.500
HB-RISB-11	8/29/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	4.103	4.103	ng/kg		0.1	0.410
HB-RISB-11	8/29/2003	0.5	1	39001-02-0	OCDF	Y	14.329	14.329	ng/kg		0.0003	0.004
Sample Location TEQ =												3.8

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

EMPC = Estimated Maximum Possible Concentration

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.32c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Unit
Highly Chlorinated PCBs	HB-HB-18S	0	0.5	8/29/2003	0.74	mg/kg
Highly Chlorinated PCBs	HB-HB-18S	0.5	1	8/29/2003	3.9	mg/kg
Highly Chlorinated PCBs	HB-HB-19S	0	0.5	12/30/2002	0.24	mg/kg
Highly Chlorinated PCBs	HB-HB-19S	0.5	1	12/30/2002	1	mg/kg
Highly Chlorinated PCBs	HB-HB-20D	0	0.5	8/29/2003	1.7	mg/kg
Highly Chlorinated PCBs	HB-HB-20D	0.5	1	8/29/2003	2.6	mg/kg
Highly Chlorinated PCBs	HB-RISB-05	0.5	1	1/2/2003	1.39	mg/kg
Highly Chlorinated PCBs	HB-RISB-07	0	0.5	12/17/2002	4	mg/kg
Highly Chlorinated PCBs	HB-RISB-16	0	0.5	5/25/2004	0.49	mg/kg
Highly Chlorinated PCBs	HB-RISB-16	0.5	1	5/25/2004	0.39	mg/kg
Highly Chlorinated PCBs	HB-RISB-16	4	6	5/24/2004	1.25	mg/kg
Highly Chlorinated PCBs	HB-RISB-17	0	0.5	5/25/2004	0.44	mg/kg
Highly Chlorinated PCBs	HB-RISB-17	0.5	1	5/25/2004	0.75	mg/kg
Total PCBs	HB-HB-18S	0	0.5	8/29/2003	0.74	mg/kg
Total PCBs	HB-HB-18S	0.5	1	8/29/2003	3.9	mg/kg
Total PCBs	HB-HB-19S	0	0.5	12/30/2002	0.24	mg/kg
Total PCBs	HB-HB-19S	0.5	1	12/30/2002	1	mg/kg
Total PCBs	HB-HB-20D	0	0.5	8/29/2003	1.7	mg/kg
Total PCBs	HB-HB-20D	0.5	1	8/29/2003	2.6	mg/kg
Total PCBs	HB-RISB-05	0.5	1	1/2/2003	1.39	mg/kg
Total PCBs	HB-RISB-07	0	0.5	12/17/2002	4	mg/kg
Total PCBs	HB-RISB-16	0	0.5	5/25/2004	0.49	mg/kg
Total PCBs	HB-RISB-16	0.5	1	5/25/2004	0.39	mg/kg
Total PCBs	HB-RISB-16	4	6	5/24/2004	1.25	mg/kg
Total PCBs	HB-RISB-17	0	0.5	5/25/2004	0.44	mg/kg
Total PCBs	HB-RISB-17	0.5	1	5/25/2004	0.75	mg/kg

Notes:

* Highly Chlorinated PCB's were defined as Aroclores 1248, 1254, 1260, and higher if reported. Total PCBs' are the sum of all detected Aroclores.

TABLE 2.32d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-HB-18S	8/29/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.03
HB-HB-18S	8/29/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.03
Total Chlordane =									ND
HB-HB-18S	8/29/2003	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.031
HB-HB-18S	8/29/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.031
Total Chlordane =									ND
HB-HB-19S	12/30/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.011
HB-HB-19S	12/30/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.011
Total Chlordane =									ND
HB-HB-19S	12/30/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.015
HB-HB-19S	12/30/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.12
Total Chlordane =									0.12
HB-HB-20D	8/29/2003	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.052
HB-HB-20D	8/29/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.052
Total Chlordane =									ND
HB-HB-20D	8/29/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.04
HB-HB-20D	8/29/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.04
Total Chlordane =									ND
HB-RISB-04	12/18/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.16
HB-RISB-04	12/18/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.16
Total Chlordane =									ND
HB-RISB-04	12/18/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.13
HB-RISB-04	12/18/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.13
Total Chlordane =									ND
HB-RISB-05	1/2/2003	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.025
HB-RISB-05	1/2/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.025
Total Chlordane =									ND
HB-RISB-06	12/18/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.27
HB-RISB-06	12/18/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.27
Total Chlordane =									ND
HB-RISB-06	12/18/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.27
HB-RISB-06	12/18/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.27
Total Chlordane =									ND
HB-RISB-07	12/17/2002	0	0.5	57-74-9	CHLORDANE	N	UJ	mg/kg	0.19
HB-RISB-07	12/17/2002	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.19
Total Chlordane =									ND
HB-RISB-07	12/17/2002	0.5	1	57-74-9	CHLORDANE	N	UJ	mg/kg	0.089
HB-RISB-07	12/17/2002	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.089
Total Chlordane =									ND
HB-RISB-11	8/29/2003	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.019
HB-RISB-11	8/29/2003	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.019
Total Chlordane =									ND
HB-RISB-11	8/29/2003	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.018
HB-RISB-11	8/29/2003	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.018
Total Chlordane =									ND
HB-RISB-16	5/25/2004	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.005
HB-RISB-16	5/25/2004	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.005
Total Chlordane =									ND

TABLE 2.32d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-RISB-16	5/25/2004	0.5	1	57-74-9	CHLORDANE	N	U	mg/kg	0.0054
HB-RISB-16	5/25/2004	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0054
Total Chlordane =									ND
HB-RISB-16	5/24/2004	4	6	57-74-9	CHLORDANE	N	U	mg/kg	0.005
HB-RISB-16	5/24/2004	4	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.005
Total Chlordane =									ND
HB-RISB-17	5/25/2004	0	0.5	57-74-9	CHLORDANE	N	U	mg/kg	0.0047
HB-RISB-17	5/25/2004	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0047
Total Chlordane =									ND

TABLE 2.32e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-SB-66	10/31/2006	7	8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	29	29
HB-SB-67	10/31/2006	5	6	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.27	0.27
HB-SB-68	11/14/2006	6	8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.0067	0.0067
HB-HB-18S	8/29/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0088	
HB-HB-18S	8/29/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0088	
HB-HB-18S	8/29/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0088
HB-HB-18S	8/29/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.0018	
HB-HB-18S	8/29/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0033	
HB-HB-18S	8/29/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0051
HB-HB-19S	12/30/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.017	
HB-HB-19S	12/30/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.017	
HB-HB-19S	12/30/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.017
HB-HB-19S	12/30/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.011	
HB-HB-19S	12/30/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.011	
HB-HB-19S	12/30/2002	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.011
HB-HB-20D	8/29/2003	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.015	
HB-HB-20D	8/29/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.015	
HB-HB-20D	8/29/2003	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.015
HB-HB-20D	8/29/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.0045	
HB-HB-20D	8/29/2003	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.014	
HB-HB-20D	8/29/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.0185
HB-RISB-04	12/18/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.012	
HB-RISB-04	12/18/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.0084	
HB-RISB-04	12/18/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.0084
HB-RISB-04	12/18/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0096	
HB-RISB-04	12/18/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.005	
HB-RISB-04	12/18/2002	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.005
HB-RISB-05	1/2/2003	0	0.5	95-47-6	O-XYLENE	Y	J	mg/kg	0.11	
HB-RISB-05	1/2/2003	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.22	
HB-RISB-05	1/2/2003	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.33
HB-RISB-05	1/2/2003	0.5	1	95-47-6	O-XYLENE	Y	J	mg/kg	0.008	
HB-RISB-05	1/2/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.036	
HB-RISB-05	1/2/2003	0.5	1	CALCULATED	TOTAL	Y	J	mg/kg		0.008
HB-RISB-06	12/18/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0	0.5	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0	0.5	CALCULATED	TOTAL	N	UJ	mg/kg		0.02
HB-RISB-06	12/18/2002	0.5	1	95-47-6	O-XYLENE	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0.5	1	XYLENES1314	XYLENES, M & P	N	UJ	mg/kg	0.02	
HB-RISB-06	12/18/2002	0.5	1	CALCULATED	TOTAL	N	UJ	mg/kg		0.02
HB-RISB-07	12/17/2002	0	0.5	95-47-6	O-XYLENE	N	UJ	mg/kg	0.03	
HB-RISB-07	12/17/2002	0	0.5	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	0.018	
HB-RISB-07	12/17/2002	0	0.5	CALCULATED	TOTAL	Y	J	mg/kg		0.018
HB-RISB-07	12/17/2002	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.011	
HB-RISB-07	12/17/2002	0.5	1	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.012	
HB-RISB-07	12/17/2002	0.5	1	CALCULATED	TOTAL	Y		mg/kg		0.012
HB-RISB-11	8/29/2003	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0055	
HB-RISB-11	8/29/2003	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0055	
HB-RISB-11	8/29/2003	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0055
HB-RISB-11	8/29/2003	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0054	
HB-RISB-11	8/29/2003	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0054	
HB-RISB-11	8/29/2003	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0054
HB-RISB-16	5/25/2004	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0074	
HB-RISB-16	5/25/2004	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0074	
HB-RISB-16	5/25/2004	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0074
HB-RISB-16	5/25/2004	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0079	
HB-RISB-16	5/25/2004	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0079	
HB-RISB-16	5/25/2004	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0079

TABLE 2.32e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-RISB-16	5/24/2004	4	6	95-47-6	O-XYLENE	Y		mg/kg	0.011	
HB-RISB-16	5/24/2004	4	6	XYLENES1314	XYLENES, M & P	Y		mg/kg	0.018	
HB-RISB-16	5/24/2004	4	6	CALCULATED	TOTAL	Y		mg/kg		0.029
HB-RISB-17	5/25/2004	0	0.5	95-47-6	O-XYLENE	N	U	mg/kg	0.0069	
HB-RISB-17	5/25/2004	0	0.5	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0069	
HB-RISB-17	5/25/2004	0	0.5	CALCULATED	TOTAL	N	U	mg/kg		0.0069
HB-RISB-17	5/25/2004	0.5	1	95-47-6	O-XYLENE	N	U	mg/kg	0.0079	
HB-RISB-17	5/25/2004	0.5	1	XYLENES1314	XYLENES, M & P	N	U	mg/kg	0.0079	
HB-RISB-17	5/25/2004	0.5	1	CALCULATED	TOTAL	N	U	mg/kg		0.0079
HB-RISB-17	5/25/2004	6	8	95-47-6	O-XYLENE	Y	J	mg/kg	3	
HB-RISB-17	5/25/2004	6	8	XYLENES1314	XYLENES, M & P	Y	J	mg/kg	5.8	
HB-RISB-17	5/25/2004	6	8	CALCULATED	TOTAL	Y	J	mg/kg		8.8

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.33a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- ADDITIONAL AREA OF STUDY #1 SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
AOS #1 Shallow Ground Water	METALS																		
	7429-90-5	ALUMINUM	0.12	2.23 J	mg/L	HB-HB-18S	6/6	-	2.23E+00		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	N	BSL	
	7440-39-3	BARIUM	0.194	2.12 J	mg/L	HB-HB-20S	9/9	-	2.12E+00		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	Y	ASL	
	7440-70-2	CALCIUM	400	1940	mg/L	HB-HB-20S	9/9	-	1.94E+03			NV	NV	NV	N	NV	N	NUT	
	7440-47-3	CHROMIUM ^a	0.0024 J	0.012 J	mg/L	HB-HB-18S	3/7	0.01-0.03	1.20E-02		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX	
	7440-50-8	COPPER	0.006 J	0.0202 J	mg/L	HB-HB-18S	2/9	0.01-0.06	2.02E-02		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	N	BSL	
	57-12-5	CYANIDE	0.015	0.0226	mg/L	HB-HB-18S	4/9	0.01-0.01	2.26E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL	
	7439-89-6	IRON	0.166 J	43 J	mg/L	HB-HB-20S	9/9	-	4.30E+01		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL	
	7439-92-1	LEAD	0.013	0.0263 J	mg/L	HB-HB-20S	3/9	0.008-0.0229	2.63E-02		1.50E-02	NV	NV	NV	Y	1.50E-02	Y	ASL	
	7439-95-4	MAGNESIUM	0.697	33.6	mg/L	HB-HB-20S	8/9	1.31-1.31	3.36E+01			NV	NV	NV	N	NV	N	NUT	
	7439-96-5	MANGANESE	0.11	5.11	mg/L	HB-HB-20S	7/9	0.01-0.03	5.11E+00		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	Y	ASL	
	7439-97-6	MERCURY ^b	0.000037 J	0.0004	mg/L	HB-HB-20S	3/9	0.0002-0.0002	4.00E-04		2.00E-03	3.65E-04	N	3.65E-04	nc	3.65E-04	Y	ASL	
	7440-02-0	NICKEL	0.0018 J	0.0062 J	mg/L	HB-HB-19S	3/9	0.04-0.12	6.20E-03			7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL	
	7440-09-7	POTASSIUM	19 J	101 J	mg/L	HB-HB-20S	9/9	-	1.01E+02			NV	NV	NV	N	NV	N	NUT	
	7440-23-5	SODIUM	910	4620	mg/L	HB-HB-20S	9/9	-	4.62E+03			NV	NV	NV	N	NV	N	NUT	
	7440-62-2	VANADIUM	0.003 J	0.003 J	mg/L	HB-HB-19S	1/9	0.05-0.15	3.00E-03			3.65E-03	N	3.65E-03	nc	3.65E-03	N	BSL	
	7440-66-6	ZINC	0.0835	0.0835	mg/L	HB-HB-20S	1/9	0.02-0.06	8.35E-02		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	N	BSL	
	PESTICIDES																		
	72-54-8	4,4'-DDD		0.025 J	0.025 J	ug/l	HB-HB-18S	1/9	0.093-0.11	2.50E-02			2.79E-01	C	2.80E-01	ca	2.79E-01	N	BSL
	SVOCs																		
	91-57-6	2-METHYLNAPHTHALENE		1.5 J	4.7 J	ug/l	HB-HB-20S	7/9	10-23	4.70E+00			2.43E+00	N	NV		2.43E+00	Y	ASL
	95-48-7	2-METHYLPHENOL		1.8 J	4.2 J	ug/l	HB-HB-18S	6/9	9.5-10	4.20E+00			1.83E+02	N	1.82E+02	nc	1.82E+02	N	BSL
	106-44-5	4-METHYLPHENOL		22	44	ug/l	HB-HB-19S	2/3	10-10	4.40E+01			1.83E+01	N	1.82E+01	nc	1.82E+01	Y	ASL
	83-32-9	ACENAPHTHENE		1.6 J	7.7 J	ug/l	HB-HB-20S	7/9	10-23	7.70E+00			3.65E+01	N	3.65E+01	nc	3.65E+01	N	BSL
	120-12-7	ANTHRACENE		1.3 J	1.8 J	ug/l	HB-HB-20S	3/9	9.5-23	1.80E+00			1.83E+02	N	1.83E+02	nc	1.83E+02	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE		1 J	6.7 J	ug/l	HB-HB-19S	7/9	10-21	6.70E+00		6.00E+00	4.78E+00	C	4.80E+00	ca	4.78E+00	Y	ASL
	86-74-8	CARBAZOLE		1 J	2.3 J	ug/l	HB-HB-19S	4/9	9.4-23	2.30E+00			3.35E+00	C	3.36E+00	ca	3.35E+00	N	BSL
	132-64-9	DIBENZOFURAN		1.2 J	1.8 J	ug/l	HB-HB-20S	5/9	9.5-23	1.80E+00			3.65E+00	N	1.22E+00	nc	1.22E+00	Y	ASL
	206-44-0	FLUORANTHENE		0.97 J	2 J	ug/l	HB-HB-20S	5/9	9.5-23	2.00E+00			1.46E+02	N	1.46E+02	nc	1.46E+02	N	BSL
	86-73-7	FLUORENE		1.2 J	4.8 J	ug/l	HB-HB-20S	7/9	10-23	4.80E+00			2.43E+01	N	2.43E+01	nc	2.43E+01	N	BSL
	34METPH	3&4-METHYLPHENOL ^c		1.1 J	17	ug/l	HB-HB-19S	6/6	-	1.70E+01			1.83E+01	N	1.82E+01	nc	1.82E+01	N	BSL
	91-20-3	NAPHTHALENE		1.9 J	38	ug/l	HB-HB-19S	7/9	10-11	3.80E+01			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL
	85-01-8	PHENANTHRENE		2 J	9.5 J	ug/l	HB-HB-20S	7/9	10-23	9.50E+00			NV	NV	NV	Y	NV	Y	NTX
	108-95-2	PHENOL		1.4 J	230	ug/l	HB-HB-19S	8/9	10-10	2.30E+02			1.10E+03	N	1.09E+03	nc	1.09E+03	N	BSL
	129-00-0	PYRENE		1.1 J	1.7 J	ug/l	HB-HB-20S	3/9	9.5-23	1.70E+00			1.83E+01	N	1.83E+01	nc	1.83E+01	N	BSL

TABLE 2.33a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- ADDITIONAL AREA OF STUDY #1 SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)		
	VOCs																	
	106-46-7	1,4-DICHLOROBENZENE	0.2 J	0.96 J	ug/l	HB-HB-20S	3/9	0.5-10	9.60E-01		7.50E+01	2.81E-01	C	5.02E-01	ca	2.81E-01	Y	ASL
	78-93-3	2-BUTANONE	2.03 J	2.12 J	ug/l	HB-HB-19S	2/9	10-20	2.12E+00			6.97E+02	N	6.97E+02	nc	6.97E+02	N	BSL
	108-10-1	4-METHYL-2-PENTANONE	1.37 J	1.37 J	ug/l	HB-HB-18S	1/9	5-20	1.37E+00			6.28E+02	N	1.99E+02	nc	1.99E+02	N	BSL
	67-64-1	ACETONE	4 J	24 J	ug/l	HB-HB-18S	7/9	40-40	2.40E+01			5.48E+02	N	5.48E+02	nc	5.48E+02	N	BSL
	71-43-2	BENZENE	0.51	2.1 J	ug/l	HB-HB-20S	4/9	10-10	2.10E+00		5.00E+00	3.36E-01	C	3.54E-01	ca	3.36E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.23 J	2.9 J	ug/l	HB-HB-18S	3/9	0.5-20	2.90E+00			1.04E+02	N	1.04E+02	nc	1.04E+02	N	BSL
	108-90-7	CHLOROBENZENE	0.16 J	0.16 J	ug/l	HB-HB-18S	1/9	0.5-10	1.60E-01		1.00E+02	8.96E+00	N	1.06E+01	nc	8.96E+00	N	BSL
	67-66-3	CHLOROFORM	0.13 J	0.13 J	ug/l	HB-HB-20S	1/9	0.5-10	1.30E-01			1.55E-01	C	1.65E-01	ca	1.55E-01	N	BSL
	100-41-4	ETHYLBENZENE	0.13 J	0.24 J	ug/l	HB-HB-20S	3/9	10-10	2.40E-01		7.00E+02	1.34E+02	N	1.34E+02	nc	1.34E+02	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.11 J	0.13 J	ug/l	HB-HB-18S	2/3	0.5-0.5	1.30E-01			6.58E+01	N	6.58E+01	nc	6.58E+01	N	BSL
	1634-04-4	METHYL TERT-BUTYL ETHER	0.22 J	0.5	ug/l	HB-HB-18S	3/3	-	5.00E-01			2.64E+00	C	1.10E+01	ca	2.64E+00	N	BSL
	108-88-3	TOLUENE	0.25 J	17.6	ug/l	HB-HB-18S	4/9	10-10	1.76E+01		1.00E+03	2.27E+02	N	7.23E+01	nc	7.23E+01	N	BSL
	1330-20-7	XYLENES, TOTAL	0.52 J	0.73 J	ug/l	HB-HB-18S	3/9	10-10	7.30E-01		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01	N	BSL

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.

(4) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.

(5) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.

(6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.

(7) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = RBC and PRG values for chromium VI utilized.

b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

c = RBC and PRG values for 4-methylphenol utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NUT: Compound is an essential nutrient and not screened in

NV: No Value

PRG: Preliminary Remediation Goals, USEPA, 2004

RBC: Risk Based Concentration; USEPA, October, 2007

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

AOS #1: Additional Area of Study #1

TABLE 2.33b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - AOS #1 SHALLOW GROUND WATER (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-18S	5/21/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-18S	5/21/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-18S	5/21/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-18S	8/27/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-18S	8/27/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-18S	8/27/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-18S	3/20/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.73	0.73
HB-HB-19S	5/21/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-19S	5/21/2003	4.01	14.01	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-19S	5/21/2003	4.01	14.01	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-19S	8/27/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-19S	8/27/2003	4.01	14.01	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-19S	8/27/2003	4.01	14.01	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-19S	3/20/2007	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.57	0.57
HB-HB-20S	5/22/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20S	5/22/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20S	5/22/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20S	8/25/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20S	8/25/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20S	8/25/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20S	3/22/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.52	0.52

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.34a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- AOS #1 SHALLOW GROUND WATER: VAPOR INTRUSION
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	Target Groundwater Concentration Corresponding to Target Indoor Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor = 0.001 and Partitioning Across the Water Table Obeys Henry's Law (10-6) (5)	Screening Toxicity Value	COPC Flag (Y/N)	Rationale for Selection or Deletion (6)	
AOS #1 - Shallow Ground Water	SVOCs															
	91-57-6	2-METHYLNAPHTHALENE	1.5 J	4.7 J	ug/l	HB-HB-20S	7/9	10-23	4.70E+00			3.30E+02	nc	3.30E+02	N	BSL
	95-48-7	2-METHYLPHENOL	1.8 J	4.2 J	ug/l	HB-HB-18S	6/9	9.5-10	4.20E+00					NV	Y	NTX
	106-44-5	4-METHYLPHENOL	22	44	ug/l	HB-HB-19S	2/3	10-10	4.40E+01					NV	Y	NTX
	83-32-9	ACENAPHTHENE	1.6 J	7.7 J	ug/l	HB-HB-20S	7/9	10-23	7.70E+00			**	nc	**	N	BSL
	120-12-7	ANTHRACENE	1.3 J	1.8 J	ug/l	HB-HB-20S	3/9	9.5-23	1.80E+00					NV	Y	NTX
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1 J	6.7 J	ug/l	HB-HB-19S	7/9	10-21	6.70E+00		6.00E+00			NV	Y	NTX
	86-74-8	CARBAZOLE	1 J	2.3 J	ug/l	HB-HB-19S	4/9	9.4-23	2.30E+00					NV	Y	NTX
	132-64-9	DIBENZOFURAN	1.2 J	1.8 J	ug/l	HB-HB-20S	5/9	9.5-23	1.80E+00			**	nc	NV	Y	NTX
	206-44-0	FLUORANTHENE	0.97 J	2 J	ug/l	HB-HB-20S	5/9	9.5-23	2.00E+00					NV	Y	NTX
	86-73-7	FLUORENE	1.2 J	4.8 J	ug/l	HB-HB-20S	7/9	10-23	4.80E+00			**	nc	**	N	BSL
	34METPH	3&4-METHYLPHENOL	1.1 J	17	ug/l	HB-HB-19S	6/6	-	1.70E+01					NV	Y	NTX
	91-20-3	NAPHTHALENE	1.9 J	38	ug/l	HB-HB-19S	7/9	10-11	3.80E+01			1.50E+01	nc	1.50E+01	Y	ASL
	85-01-8	PHENANTHRENE	2 J	9.5 J	ug/l	HB-HB-20S	7/9	10-23	9.50E+00					NV	Y	NTX
	108-95-2	PHENOL	1.4 J	230	ug/l	HB-HB-19S	8/9	10-10	2.30E+02					NV	Y	NTX
	129-00-0	PYRENE	1.1 J	1.7 J	ug/l	HB-HB-20S	3/9	9.5-23	1.70E+00			**	nc	**	N	BSL
	VOCs															
	106-46-7	1,4-DICHLOROBENZENE	0.2 J	0.96 J	ug/l	HB-HB-20S	3/9	0.5-10	9.60E-01		7.50E+01	8.20E+02	nc	8.20E+02	N	BSL
	78-93-3	2-BUTANONE	2.03 J	2.12 J	ug/l	HB-HB-19S	2/9	10-20	2.12E+00			4.40E+04	nc	4.40E+04	N	BSL
	108-10-1	4-METHYL-2-PENTANONE	1.37 J	1.37 J	ug/l	HB-HB-18S	1/9	5-20	1.37E+00					NV	Y	NTX
	67-64-1	ACETONE	4 J	24 J	ug/l	HB-HB-18S	7/9	40-40	2.40E+01			2.20E+04	nc	2.20E+04	N	BSL
	71-43-2	BENZENE	0.51	2.1 J	ug/l	HB-HB-20S	4/9	10-10	2.10E+00		5.00E+00	1.72E+01	c	1.72E+01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.23 J	2.9 J	ug/l	HB-HB-18S	3/9	0.5-20	2.90E+00			5.60E+01	nc	5.60E+01	N	BSL
	108-90-7	CHLOROBENZENE	0.16 J	0.16 J	ug/l	HB-HB-18S	1/9	0.5-10	1.60E-01		1.00E+02	3.90E+01	nc	3.90E+01	N	BSL
	67-66-3	CHLOROFORM	0.13 J	0.13 J	ug/l	HB-HB-20S	1/9	0.5-10	1.30E-01			4.40E+00	c	4.40E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.13 J	0.24 J	ug/l	HB-HB-20S	3/9	10-10	2.40E-01		7.00E+02	8.08E+01	c	8.08E+01	N	BSL
	98-82-8	ISOPROPYLBENZENE	0.11 J	0.13 J	ug/L	HB-HB-18S	2/3	0.5-0.5	1.30E-01					NV	Y	NTX
	1634-04-4	METHYL TERT-BUTYL ETHER	0.22 J	0.5	ug/l	HB-HB-18S	3/3	-	5.00E-01					NV	Y	NTX
	108-88-3	TOLUENE	0.25 J	17.6	ug/l	HB-HB-18S	4/9	10-10	1.76E+01		1.00E+03	1.50E+02	nc	1.50E+02	N	BSL
	1330-20-7	XYLENES, TOTAL ^a	0.52 J	0.73 J	ug/l	HB-HB-18S	3/9	10-10	7.30E-01		1.00E+04	2.20E+03	nc	2.20E+03	N	BSL

Footnotes:

* Sample start depth less than or equal to 10 ft bgs.

** Target soil gas concentration exceeds maximum possible vapor concentration (pathway incomplete)

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) Primary and Secondary Drinking Water Regulations

(5) USEPA - OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils (Subsurface Vapor Intrusion Guidance) Tables. November 2002. ca = Cancer; nc = Noncancer.

Screening criteria correspond to a cancer risk of 10-6 and a noncancer hazard of 0.1. For USEPA (2002) criteria that defaulted to MCLs, criteria were derived (in italics) from USEPA (2009) RSL residential air concentration based on an attenuation factor of 10 and the Henry's Law constant for each compound at 25 deg C.

(6) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = Target groundwater concentration for p-xylene (CAS #106-42-3) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NV: No Value

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

AOS #1: Additional Area of Study #1

TABLE 2.34b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - AOS #1 SHALLOW GROUND WATER: VAPOR INTRUSION

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-HB-18S	5/21/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-18S	5/21/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-18S	5/21/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-18S	8/27/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-18S	8/27/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-18S	8/27/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-18S	3/20/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.73	0.73
HB-HB-19S	5/21/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-19S	5/21/2003	4.01	14.01	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-19S	5/21/2003	4.01	14.01	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-19S	8/27/2003	4.01	14.01	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-19S	8/27/2003	4.01	14.01	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-19S	8/27/2003	4.01	14.01	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-19S	3/20/2007	4.01	14.01	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.57	0.57
HB-HB-20S	5/22/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20S	5/22/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20S	5/22/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20S	8/25/2003	3.98	13.98	XYLENES1314	XYLENES, M & P	N	U	ug/l	10	
HB-HB-20S	8/25/2003	3.98	13.98	95-47-6	O-XYLENE	N	U	ug/l	10	
HB-HB-20S	8/25/2003	3.98	13.98	CALCULATED	TOTAL	N	U	ug/l		10
HB-HB-20S	3/22/2007	3.98	13.98	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.52	0.52

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.35
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- AOS #2 SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
AOS #2 Surface Soil	METALS																		
	7429-90-5	ALUMINUM	3860 J	3920 J	mg/kg	HB-RISB-10	2/2	-	3.92E+03			7.82E+03	N	7.61E+03	nc	7.61E+03	N	BSL	
	7440-38-2	ARSENIC	2.6	3.4 J	mg/kg	HB-RISB-10	2/2	-	3.40E+00		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX	
	7440-39-3	BARIUM	174 J	250	mg/kg	HB-RISB-10	2/2	-	2.50E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02	N	BSL	
	7440-70-2	CALCIUM	192000	277000 J	mg/kg	HB-RISB-10	2/2	-	2.77E+05			NV		NV		NV	N	NUT	
	7440-47-3	CHROMIUM ^a	12.3 J	17.7 J	mg/kg	HB-RISB-10	2/2	-	1.77E+01			2.35E+01	N	3.01E+00	nc	3.01E+00	Y	TOX	
	7440-50-8	COPPER	16.6	22.3 J	mg/kg	HB-RISB-10	2/2	-	2.23E+01		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL	
	7439-89-6	IRON	8810	9320 J	mg/kg	HB-RISB-10	2/2	-	9.32E+03			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL	
	7439-92-1	LEAD	42.9 J	71.9 J	mg/kg	HB-RISB-10	2/2	-	7.19E+01			NV		4.00E+02	nc	4.00E+02	N	BSL	
	7439-95-4	MAGNESIUM	41300 J	60500	mg/kg	HB-RISB-10	2/2	-	6.05E+04			NV		NV		NV	N	NUT	
	7439-96-5	MANGANESE	239	297 J	mg/kg	HB-RISB-10	2/2	-	2.97E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL	
	7439-97-6	MERCURY ^b	0.15	0.27 J	mg/kg	HB-RISB-10	2/2	-	2.70E-01			7.82E-01	N	6.11E-01	nc	6.11E-01	N	BSL	
	7440-02-0	NICKEL	13.5	16.2 J	mg/kg	HB-RISB-10	2/2	-	1.62E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL	
	7440-09-7	POTASSIUM	1180	2170 J	mg/kg	HB-RISB-10	2/2	-	2.17E+03			NV		NV		NV	N	NUT	
	7440-23-5	SODIUM	240	294 J	mg/kg	HB-RISB-10	2/2	-	2.94E+02			NV		NV		NV	N	NUT	
	7440-62-2	VANADIUM	12.6	19.9 J	mg/kg	HB-RISB-10	2/2	-	1.99E+01			7.82E+00	N	7.82E+00	nc	7.82E+00	Y	ASL	
	7440-66-6	ZINC	50.4	88.6 J	mg/kg	HB-RISB-10	2/2	-	8.86E+01		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL	
	SVOCS																		
	83-32-9	ACENAPHTHENE	1 J	1 J	mg/kg	HB-RISB-10	1/2	5.6-5.6	1.00E+00		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02	N	BSL	
	120-12-7	ANTHRACENE	1.2 J	1.7 J	mg/kg	HB-RISB-10	2/2	-	1.70E+00			2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL	
	56-55-3	BENZ(A)ANTHRACENE	3.3 J	5.8 J	mg/kg	HB-RISB-10	2/2	-	5.80E+00			1.00E+00	C	6.21E-01	ca	2.20E-01	Y	ASL	
	50-32-8	BENZO(A)PYRENE	3.2 J	6.6 J	mg/kg	HB-RISB-10	2/2	-	6.60E+00			1.00E+00	C	6.21E-02	ca	2.20E-02	Y	ASL	
	205-99-2	BENZO(B)FLUORANTHENE	2.3 J	5 J	mg/kg	HB-RISB-10	2/2	-	5.00E+00			1.00E+00	C	6.21E-01	ca	2.20E-01	Y	ASL	
	191-24-2	BENZO(G,H,I)PERYLENE	1.8 J	3.9 J	mg/kg	HB-RISB-10	2/2	-	3.90E+00			1.00E+02	NV	NV		NV	Y	NTX	
	207-08-9	BENZO(K)FLUORANTHENE	2.3 J	4.7 J	mg/kg	HB-RISB-10	2/2	-	4.70E+00			1.00E+00	C	6.21E+00	ca	2.20E+00	Y	ASL	
	86-74-8	CARBAZOLE	1.1 J	1.1 J	mg/kg	HB-RISB-10	1/2	5.6-5.6	1.10E+00			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL	
	218-01-9	CHRYSENE	3 J	5.5 J	mg/kg	HB-RISB-10	2/2	-	5.50E+00			2.20E+01	C	6.21E+01	ca	2.20E+01	N	BSL	
	53-70-3	DIBENZ(A,H)ANTHRACENE	1.2 J	1.2 J	mg/kg	HB-RISB-10	1/2	5.6-5.6	1.20E+00			3.30E-01	C	6.21E-02	ca	2.20E-02	Y	ASL	
	206-44-0	FLUORANTHENE	6.4	9.4 J	mg/kg	HB-RISB-10	2/2	-	9.40E+00			1.00E+02	N	2.29E+02	nc	2.29E+02	N	BSL	
	193-39-5	INDENO(1,2,3-CD)PYRENE	1.7 J	3.8 J	mg/kg	HB-RISB-10	2/2	-	3.80E+00			5.00E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	85-01-8	PHENANTHRENE	3.9 J	5.9 J	mg/kg	HB-RISB-10	2/2	-	5.90E+00			1.00E+02	NV	NV		NV	Y	NTX	
	129-00-0	PYRENE	5 J	7.5 J	mg/kg	HB-RISB-10	2/2	-	7.50E+00			1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																		
	67-64-1	ACETONE	0.11 J	0.19 J	mg/kg	HB-RISB-10	2/2	-	1.90E-01			1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
 - (5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
 - (6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
 - (7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency
AOS #2: Additional Area of Study #2

TABLE 2.36a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE- AOS #2 SEDIMENT
GEDDES AND SYRACUSE, NY

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment (0-1 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value	USEPA RBC for Residential Soil (4)	USEPA PRG for Residential Soil (5)	Screening Toxicity Value (6)	COPC Flag (Y/N)	Rationale for Selection or Deletion (7)		
AOS #2 Sediment	DIOXIN/FURAN (8)																	
	1746-01-6	2,3,7,8 TCDD Equivalent	0.0000005	0.0000006	mg/kg	HB-HBSED-18	2/2		6.09E-07			4.26E-06	C	3.90E-06	ca	3.90E-06	N	BSL
	METALS																	
	7429-90-5	ALUMINUM	1700 J	3120	mg/kg	HB-HBSED-18	2/2	-	3.12E+03			7.82E+03	N	7.61E+03	nc	7.61E+03	N	BSL
	7440-38-2	ARSENIC	2.2 J	2.2 J	mg/kg	HB-HBSED-18	1/2	1.9-1.9	2.20E+00			4.26E-01	C	3.90E-01	ca	3.90E-01	Y	TOX
	7440-39-3	BARIUM	576	659 J	mg/kg	HB-HBSED-18	2/2	-	6.59E+02			1.56E+03	N	5.37E+02	nc	5.37E+02	Y	ASL
	7440-70-2	CALCIUM	311000	344000 J	mg/kg	HB-HBSED-18	2/2	-	3.44E+05			NV	NV	NV	N	NV	N	NUT
	7440-47-3	CHROMIUM ^a	7.4 J	9.3	mg/kg	HB-HBSED-18	2/2	-	9.30E+00			2.35E+01	N	3.01E+01	ca	2.35E+01	Y	TOX
	7440-50-8	COPPER	20.6 J	24.6 J	mg/kg	HB-HBSED-18	2/2	-	2.46E+01			3.13E+02	N	3.13E+02	nc	3.13E+02	N	BSL
	57-12-5	CYANIDE	3.08 J	3.08 J	mg/kg	HB-HBSED-18	1/2	1.89-1.89	3.08E+00			1.56E+02	N	1.22E+02	nc	1.22E+02	N	BSL
	7439-89-6	IRON	15700	16700 J	mg/kg	HB-HBSED-18	2/2	-	1.67E+04			5.48E+03	N	2.35E+03	nc	2.35E+03	Y	ASL
	7439-92-1	LEAD	65.3 J	82 J	mg/kg	HB-HBSED-18	2/2	-	8.20E+01			NV	NV	4.00E+02	nc	4.00E+02	N	BSL
	7439-95-4	MAGNESIUM	5120 J	7400	mg/kg	HB-HBSED-18	2/2	-	7.40E+03			NV	NV	NV	N	NV	N	NUT
	7439-96-5	MANGANESE	499	532 J	mg/kg	HB-HBSED-18	2/2	-	5.32E+02			1.56E+02	N	1.76E+02	nc	1.56E+02	Y	ASL
	7439-97-6	MERCURY ^a	0.16 J	0.17	mg/kg	HB-HBSED-18	2/2	-	1.70E-01			7.82E-01	N	6.11E-01	nc	6.11E-01	N	BSL
	7440-02-0	NICKEL	8.1	8.1	mg/kg	HB-HBSED-18	1/2	7.5-7.5	8.10E+00			1.56E+02	N	1.56E+02	nc	1.56E+02	N	BSL
	7440-09-7	POTASSIUM	444 J	704	mg/kg	HB-HBSED-18	2/2	-	7.04E+02			NV	NV	NV	N	NV	N	NUT
	7440-23-5	SODIUM	2060	2330 J	mg/kg	HB-HBSED-18	2/2	-	2.33E+03			NV	NV	NV	N	NV	N	NUT
	7440-66-6	ZINC	65.8 J	71.4	mg/kg	HB-HBSED-18	2/2	-	7.14E+01			2.35E+03	N	2.35E+03	nc	2.35E+03	N	BSL
	SVOCs																	
	120-12-7	ANTHRACENE	0.23 J	0.72	mg/kg	HB-HBSED-18	2/2	-	7.20E-01			2.35E+03	N	2.19E+03	nc	2.19E+03	N	BSL
	56-55-3	BENZ(A)ANTHRACENE	0.91 J	1.2	mg/kg	HB-HBSED-18	2/2	-	1.20E+00			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	50-32-8	BENZO(A)PYRENE	0.96 J	1.3	mg/kg	HB-HBSED-18	2/2	-	1.30E+00			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.83 J	1.2	mg/kg	HB-HBSED-18	2/2	-	1.20E+00			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.85 J	1	mg/kg	HB-HBSED-18	2/2	-	1.00E+00			NV	NV	NV	Y	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	1 J	1.2	mg/kg	HB-HBSED-18	2/2	-	1.20E+00			2.20E+00	C	6.21E+00	ca	2.20E+00	N	BSL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.43	0.46 J	mg/kg	HB-HBSED-18	2/2	-	4.60E-01			4.56E+01	C	3.47E+01	ca	3.47E+01	N	BSL
	218-01-9	CHRYSENE	1.1 J	1.5	mg/kg	HB-HBSED-18	2/2	-	1.50E+00			2.20E+01	C	6.21E+01	ca	2.20E+01	N	BSL
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.22 J	0.27	mg/kg	HB-HBSED-18	2/2	-	2.70E-01			2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL
	84-74-2	DI-N-BUTYL PHTHALATE	0.22 J	0.22 J	mg/kg	HB-HBSED-18	1/2	1.9-1.9	2.20E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL
	206-44-0	FLUORANTHENE	2.3 J	4.3	mg/kg	HB-HBSED-18	2/2	-	4.30E+00			3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL
	86-73-7	FLUORENE	0.21	0.21	mg/kg	HB-HBSED-18	1/2	2-2	2.10E-01			3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.74 J	0.9	mg/kg	HB-HBSED-18	2/2	-	9.00E-01			2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL
	85-01-8	PHENANTHRENE	1.5 J	4.7	mg/kg	HB-HBSED-18	2/2	-	4.70E+00			NV	NV	NV	Y	NV	Y	NTX
	129-00-0	PYRENE	1.8 J	3.1	mg/kg	HB-HBSED-18	2/2	-	3.10E+00			2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL
	VOCs																	
	78-93-3	2-BUTANONE	0.0049 J	0.0049 J	mg/kg	HB-HBSED-18	1/3	0.019-0.02	4.90E-03			4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.0096 J	0.035 J	mg/kg	HB-HBSED-18	3/3	-	3.50E-02			7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	71-43-2	BENZENE	0.0026 J	0.0039 J	mg/kg	HB-HBSED-18	3/3	-	3.90E-03			1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.0042 J	0.01 J	mg/kg	HB-HBSED-18	3/3	-	1.00E-02			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL

Footnotes:

- (1) J = estimated value; N = tentatively identified at an estimated value
 - (2) Concentration used for screening is the maximum detected concentration.
 - (3) No background screening performed.
 - (4) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted
 - (5) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs
 - (6) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
 - (7) Selection Rationale: ASL = Above Screening Level; TOX = Class A Carcinogen; NTX = No Toxicity Information. Deletion Rationale: BSL = Below Screening Level
 - (8) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.36b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized
b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
USEPA: United States Environmental Protection Agency
AOS #2: Additional Area of Study #2

TABLE 2.36b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - AOS #2 SURFACE SEDIMENT (0-1 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-HBSED-18	6/4/2003	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	6.803	6.803	ng/kg		0.01	0.068
HB-HBSED-18	6/4/2003	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.698	0.349	ng/kg	U	0.01	0.003
HB-HBSED-18	6/4/2003	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.258	0.129	ng/kg	U	0.1	0.013
HB-HBSED-18	6/4/2003	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.78	0.78	ng/kg	J	0.1	0.078
HB-HBSED-18	6/4/2003	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.092	1.092	ng/kg	J	0.1	0.109
HB-HBSED-18	6/4/2003	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.02	1.02	ng/kg	J	0.1	0.102
HB-HBSED-18	6/4/2003	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.218	0.109	ng/kg	U	0.1	0.011
HB-HBSED-18	6/4/2003	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.155	0.0775	ng/kg	U	1	0.078
HB-HBSED-18	6/4/2003	0	0.5	57117-41-6	1,2,3,7,8-PECDF	N	0.145	0.0725	ng/kg	U	0.03	0.002
HB-HBSED-18	6/4/2003	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.117	0.0585	ng/kg	U	1	0.059
HB-HBSED-18	6/4/2003	0	0.5	51207-31-9	2,3,7,8-TCDF	N	0.133	0.0665	ng/kg	U	0.1	0.007
HB-HBSED-18	6/4/2003	0	0.5	3268-87-9	OCDD	Y	248.352	248.352	ng/kg	J	0.0003	0.075
HB-HBSED-18	6/4/2003	0	0.5	39001-02-0	OCDF	Y	16.398	16.398	ng/kg	J	0.0003	0.005
Sample Location TEQ =												0.6
HB-HBSED-18	6/4/2003	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	4.782	4.782	ng/kg		0.01	0.048
HB-HBSED-18	6/4/2003	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.467	0.2335	ng/kg	U	0.01	0.002
HB-HBSED-18	6/4/2003	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1	1	ng/kg	J	0.1	0.100
HB-HBSED-18	6/4/2003	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.386	0.386	ng/kg	J	0.1	0.039
HB-HBSED-18	6/4/2003	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	0.87	0.87	ng/kg	J	0.1	0.087
HB-HBSED-18	6/4/2003	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.187	0.0935	ng/kg	U	0.1	0.009
HB-HBSED-18	6/4/2003	0.5	1	40321-76-4	1,2,3,7,8-PECDD	N	0.136	0.068	ng/kg	U	1	0.068
HB-HBSED-18	6/4/2003	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	0.126	0.063	ng/kg	U	0.03	0.002
HB-HBSED-18	6/4/2003	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.1	0.05	ng/kg	U	1	0.050
HB-HBSED-18	6/4/2003	0.5	1	51207-31-9	2,3,7,8-TCDF	N	0.096	0.048	ng/kg	U	0.1	0.005
HB-HBSED-18	6/4/2003	0.5	1	3268-87-9	OCDD	Y	191.287	191.287	ng/kg	J	0.0003	0.057
Sample Location TEQ =												0.5

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.37a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- SYW-12 SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
SYW-12 Surface Soil*	DIOXIN/FURAN (9)															
	1746-01-6	2,3,7,8-TCDD Equivalent	0.000001	0.0002	mg/kg	HB-WSD-15	30/30		1.62E-04			4.26E-06	C	3.90E-06	ca	3.90E-06 Y ASL
	METALS															
	7429-90-5	ALUMINUM	620	14000	mg/kg	HB-WSD-24	88/88	-	1.40E+04			7.82E+03	N	7.61E+03	nc	7.61E+03 Y ASL
	7440-36-0	ANTIMONY	0.19 J	2.1 J	mg/kg	HB-WSD-27	34/88	6.5-13	2.10E+00			3.13E+00	N	3.13E+00	nc	3.13E+00 N BSL
	7440-38-2	ARSENIC	0.77 J	20	mg/Kg	HB-WSD-27	85/88	1.3-1.8	2.00E+01		1.60E+01	4.26E-01	C	3.90E-02	nc	3.90E-02 Y TOX
	7440-39-3	BARIUM	11 J	320	mg/Kg	HB-WSD-16	88/88	-	3.20E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02 N BSL
	7440-41-7	BERYLLIUM	0.077 J	0.77 J	mg/Kg	HB-WSD-24	88/88	-	7.70E-01		1.40E+01	1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL
	7440-43-9	CADMIUM	0.38 J	52	mg/Kg	HB-WSD-16	81/88	1.1-1.8	5.20E+01		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00 Y ASL
	7440-70-2	CALCIUM	22000	370000	mg/Kg	HB-WSD-03	88/88	-	3.70E+05			NV	NV	NV	NV	N NUT
	7440-47-3	CHROMIUM ⁶	4.2 J	410	mg/Kg	HB-WSD-02	88/88	-	4.10E+02			2.35E+01	N	3.01E+01	ca	2.35E+01 Y TOX
	7440-48-4	COBALT	0.4 J	13	mg/Kg	HB-WSD-24	88/88	-	1.30E+01			NV	NV	9.03E+01	nc	9.03E+01 N BSL
	7440-50-8	COPPER	3.7	370	mg/Kg	HB-WSD-28	88/88	-	3.70E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02 Y ASL
	57-12-5	CYANIDE	0.83	2.3	mg/Kg	HB-WSD-16	14/88	0.54-1.7	2.30E+00			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	7439-89-6	IRON	2200 J	31000	mg/Kg	HB-WSD-24	88/88	-	3.10E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL
	7439-92-1	LEAD	2.1 J	390	mg/Kg	HB-WSD-16	88/88	-	3.90E+02			NV	NV	4.00E+02	nc	4.00E+02 N BSL
	7439-95-4	MAGNESIUM	2600 J	27000	mg/Kg	HB-WSD-28	88/88	-	2.70E+04			NV	NV	NV	NV	N NUT
	7439-96-5	MANGANESE	170	630 J	mg/Kg	HB-WSD-29	88/88	-	6.30E+02		2.00E+03	1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL
	7439-97-6	MERCURY ²	0.0047 J	8.6	mg/kg	HB-WSD-18	88/88	-	8.60E+00			2.35E+00	N	2.35E+00	nc	2.35E+00 Y ASL
	22967-92-6	METHYL MERCURY	0.00035	0.0135	mg/kg	HB-WSD-18	58/88	0.000021-0.00415	1.35E-02			7.82E-01	N	6.11E-01	nc	6.11E-01 N BSL
	7440-02-0	NICKEL	2.6 J	87	mg/Kg	HB-WSD-16	88/88	-	8.70E+01		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL
	7440-09-7	POTASSIUM	170 J	2300	mg/Kg	HB-WSD-24	86/88	1400-1400	2.30E+03			NV	NV	NV	NV	N NUT
	7782-49-2	SELENIUM	0.27 J	2.6	mg/Kg	HB-WSD-28	81/88	1.1-2.2	2.60E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-22-4	SILVER	0.13 J	13	mg/Kg	HB-WSD-16	66/88	1.1-2.2	1.30E+01		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-23-5	SODIUM	120 J	2000 J	mg/Kg	HB-WSD-30	62/88	110-340	2.00E+03			NV	NV	NV	NV	N NUT
	7440-62-2	VANADIUM	1.6 J	53	mg/Kg	HB-WSD-27	88/88	-	5.30E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL
	7440-66-6	ZINC	15	780	mg/Kg	HB-WSD-16	88/88	-	7.80E+02		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL
	PCBs															
		HIGHLY CHLORINATED PCBs ^c	0.0227	3.47	mg/kg	HB-WSD-16	73/86	0.0183-0.323	3.47E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL
		TOTAL PCBs ^d	0.0227	3.47	mg/kg	HB-WSD-16	73/86	0.0183-0.323	3.47E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL
	PESTICIDES															
	72-54-8	4,4'-DDD	0.00043 J	0.073 J	mg/kg	HB-WSD-02	11/87	0.0036-0.55	7.30E-02		2.60E+00	2.66E+00	C	2.44E+00	ca	2.44E+00 N BSL
	72-55-9	4,4'-DDE	0.0005 J	0.014 J	mg/kg	HB-WSD-22	5/88	0.0036-0.55	1.40E-02		1.80E+00	1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	50-29-3	4,4'-DDT	0.0025 J	0.1 J	mg/kg	HB-WSD-24	16/75	0.0036-0.55	1.00E-01		1.70E+00	1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	319-84-6	ALPHA-BHC	0.00045 J	0.00049 J	mg/kg	HB-WSD-03	2/88	0.0018-0.28	4.90E-04		9.70E-02	1.01E-01	C	9.02E-02	ca	9.02E-02 N BSL
	57-74-9	TOTAL CHLORDANE ^e	0.0012 J	0.063 J	mg/kg	HB-WSD-16	35/88	0.0018-0.28	6.30E-02		9.10E-01	1.82E+00	C	1.62E+00	ca	1.62E+00 N BSL
	60-57-1	DIELDRIN	0.0066 J	0.03	mg/kg	HB-WSD-07	9/87	0.0036-0.55	3.00E-02		3.90E-02	3.99E-02	C	3.04E-02	ca	3.04E-02 N BSL
	72-20-8	ENDRIN	0.026 J	0.026 J	mg/kg	HB-WSD-24	1/87	0.0036-0.55	2.60E-02		2.20E+00	2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL
	53494-70-5	ENDRIN KETONE ^f	0.005 J	0.0057 J	mg/kg	HB-WSD-13	2/86	0.0036-0.55	5.70E-03			2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL
	SVOCs															
	92-52-4	1,1'-BIPHENYL	0.047 J	4.9 J	mg/kg	HB-WSD-27	40/89	0.36-11	4.90E+00			3.91E+02	N	3.01E+02	nc	3.01E+02 N BSL
	91-57-6	2-METHYLNAPHTHALENE	0.046 J	16 J	mg/kg	HB-WSD-27	70/89	0.36-9.8	1.60E+01			3.13E+01	N	NV	NV	N BSL
	106-47-8	4-CHLOROANILINE	0.059 J	0.2 J	mg/kg	HB-WSD-14	3/89	0.36-25	2.00E-01			3.13E+01	N	2.44E+01	nc	2.44E+01 N BSL
	106-44-5	4-METHYLPHENOL	0.04 J	1.3 J	mg/kg	HB-WSD-26	20/89	0.36-25	1.30E+00		3.40E+01	3.91E+01	N	3.06E+01	nc	3.06E+01 N BSL
	83-32-9	ACENAPHTHENE	0.048 J	31	mg/kg	HB-WSD-27	61/89	0.36-4.1	3.10E+01		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02 N BSL
	208-96-8	ACENAPHTHYLENE	0.046 J	15 J	mg/kg	HB-WSD-27	77/89	0.36-0.63	1.50E+01		1.00E+02	NV	NV	NV	NV	Y NTX
	120-12-7	ANTHRACENE	0.047 J	88	mg/kg	HB-WSD-27	80/89	0.36-0.63	8.80E+01		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03 N BSL
	100-52-7	BENZALDEHYDE	0.047 J	1.7 J	mg/kg	HB-WSD-01	40/89	0.36-25	1.70E+00			7.82E+02	N	6.11E+02	nc	6.11E+02 N BSL
	56-55-3	BENZ(A)ANTHRACENE	0.053 J	91	mg/kg	HB-WSD-27	84/89	0.36-0.63	9.10E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	50-32-8	BENZO(A)PYRENE	0.052 J	49	mg/kg	HB-WSD-27	84/89	0.36-0.63	4.90E+01		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.098 J	67	mg/kg	HB-WSD-27	84/89	0.36-0.63	6.70E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.077 J	15 J	mg/kg	HB-WSD-27	81/89	0.36-0.63	1.50E+01		1.00E+02	NV	NV	NV	NV	Y NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.063 J	24 J	mg/kg	HB-WSD-27	82/89	0.36-0.63	2.40E+01		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00 Y ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.041 J	2.2	mg/kg	HB-WSD-04	32/89	0.087-25	2.20E+00			4.56E+01	C	3.47E+01	ca	3.47E+01 N BSL
	85-68-7	BUTYLBENZYL PHTHALATE	0.051 J	1.2 J	mg/kg	HB-WSD-27	8/89	0.36-25	1.20E+00			1.56E+03	N	1.22E+03	nc	1.22E+03 N BSL

TABLE 2.37a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE- SYW-12 SURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0-2 ft)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
	105-60-2	CAPROLACTAM	0.057 J	0.093 J	mg/kg	HB-WSD-23	3/89	0.36-25	9.30E-02			3.91E+03	N	3.06E+03	nc	3.06E+03	N	BSL	
	86-74-8	CARBAZOLE	0.047 J	6.2 J	mg/kg	HB-WSD-27	62/89	0.36-9.8	6.20E+00			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL	
	218-01-9	CHRYSENE	0.094 J	89	mg/kg	HB-WSD-27	84/89	0.36-0.63	8.90E+01	1.00E+00		2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL	
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.05 J	5.3 J	mg/kg	HB-WSD-27	74/89	0.36-0.63	5.30E+00	3.30E-01		2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL	
	132-64-9	DIBENZOFURAN	0.061 J	20 J	mg/kg	HB-WSD-27	57/89	0.36-9.8	2.00E+01	1.40E+01		7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL	
	84-74-2	DI-N-BUTYL PHTHALATE	0.15 J	0.15 J	mg/kg	HB-WSD-10	1/89	0.36-25	1.50E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL	
	206-44-0	FLUORANTHENE	0.069 J	220	mg/kg	HB-WSD-27	86/89	0.36-0.44	2.20E+02	1.00E+02		3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL	
	86-73-7	FLUORENE	0.051 J	37	mg/kg	HB-WSD-27	69/89	0.36-0.85	3.70E+01	1.00E+02		3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL	
	118-74-1	HEXACHLOROBENZENE	0.048 J	0.24 J	mg/kg	HB-WSD-18	17/89	0.36-25	2.40E-01	3.30E-01		3.99E-01	C	3.04E-01	ca	3.04E-01	N	BSL	
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.052 J	13 J	mg/kg	HB-WSD-27	81/89	0.36-0.63	1.30E+01	5.00E-01		2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	91-20-3	NAPHTHALENE	0.041 J	4.2 J	mg/kg	HB-WSD-01	72/89	0.36-9.8	4.20E+00	1.00E+02		1.56E+02	N	5.59E+00	nc	5.59E+00	N	BSL	
	85-01-8	PHENANTHRENE	0.073 J	200	mg/kg	HB-WSD-27	84/89	0.36-0.63	2.00E+02	1.00E+02		NV	NV	NV	Y	NTX			
	108-95-2	PHENOL	0.048 J	0.071 J	mg/kg	HB-WSD-16	4/89	0.36-25	7.10E-02	1.00E+02		2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL	
	129-00-0	PYRENE	0.07 J	190	mg/kg	HB-WSD-27	85/89	0.36-0.6	1.90E+02	1.00E+02		2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL	
	VOCs																		
	95-50-1	1,2-DICHLOROBENZENE	0.0009 J	0.0023 J	mg/kg	HB-WSD-06	7/87	0.0027-0.0084	2.30E-03			1.00E+02	7.04E+02	N	6.00E+01	nc	6.00E+01	N	BSL
	541-73-1	1,3-DICHLOROBENZENE	0.0011 J	0.0011 J	mg/kg	HB-WSD-24	1/87	0.0027-0.0084	1.10E-03			1.70E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL
	106-46-7	1,4-DICHLOROBENZENE	0.00069 J	0.0087	mg/kg	HB-WSD-06	31/87	0.0027-0.0084	8.70E-03			9.80E+00	2.66E+01	C	3.45E+00	ca	3.45E+00	N	BSL
	78-93-3	2-BUTANONE	0.0021 J	0.0025 J	mg/kg	HB-WSD-30	4/88	0.011-0.034	2.50E-03			1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL
	67-64-1	ACETONE	0.019 J	0.019 J	mg/kg	HB-WSD-30	1/88	0.011-0.034	1.90E-02			1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL
	98-86-2	ACETOPHENONE	0.048 J	0.76 J	mg/kg	HB-WSD-01	44/89	0.36-25	7.60E-01			7.82E+02	NV	NV	7.82E+02	N	7.82E+02	N	BSL
	71-43-2	BENZENE	0.00073 J	0.0043	mg/kg	HB-WSD-13	3/88	0.0027-0.0084	4.30E-03			2.90E+00	1.16E+01	C	6.43E-01	ca	6.43E-01	Y	TOX
	75-15-0	CARBON DISULFIDE	0.00072 J	0.0032	mg/kg	HB-WSD-19	12/88	0.0027-0.0084	3.20E-03			7.82E+02	N	3.55E+01	nc	3.55E+01	N	BSL	
	108-90-7	CHLOROBENZENE	0.00064 J	0.0058	mg/kg	HB-WSD-06	23/88	0.0027-0.0084	5.80E-03			1.00E+02	1.56E+02	N	1.51E+01	nc	1.51E+01	N	BSL
	156-59-2	CIS-1,2-DICHLOROETHENE	0.0014 J	0.0014 J	mg/kg	HB-WSD-13	1/88	0.0027-0.0084	1.40E-03			5.90E+01	7.82E+01	N	4.29E+00	nc	4.29E+00	N	BSL
	100-41-4	ETHYLBENZENE	0.00074 J	0.001 J	mg/kg	HB-WSD-04	6/88	0.0027-0.0084	1.00E-03			3.00E+01	7.82E+02	N	3.95E+01	nc	3.95E+01	N	BSL
	75-09-2	METHYLENE CHLORIDE	0.0093 J	0.0093 J	mg/kg	HB-WSD-30	1/88	0.0054-0.018	9.30E-03			5.10E+01	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL
	127-18-4	TETRACHLOROETHENE	0.0006 J	0.0041	mg/kg	HB-WSD-13	2/88	0.0027-0.0084	4.10E-03			5.50E+00	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL
	108-88-3	TOLUENE	0.00058 J	0.0038	mg/kg	HB-WSD-13	25/88	0.0027-0.0084	3.80E-03			1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL
	79-01-6	TRICHLOROETHENE	0.0043	0.0043	mg/kg	HB-WSD-13	1/88	0.0027-0.0084	4.30E-03			1.00E+01	1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL
	1330-20-7	XYLENES, TOTAL	0.00077 J	0.0073	mg/kg	HB-WSD-10	27/88	0.00285-0.0085	7.30E-03			1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL

Footnotes:

*SYW-12 wetland sediment is considered hydric soil and was evaluated as surface soil.

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) No background screening performed.

(4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.

(5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.

(6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.

(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.

(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

(9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.37b.

- = Compound detected in 100% of samples.

NA = Not applicable, minimum and maximum values are calculated.

a = RBC and PRG values for chromium VI utilized.

b = RBC and PRG values for mercury compounds utilized.

c = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

d = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.

e = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.

f = RBC and PRG values for Endrin (CAS # 72208) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NUT: Compound is an essential nutrient and is not screened in

NV: No Value

PRG: Preliminary Remediation Goals, USEPA, 2004

RBC: Risk Based Concentration; USEPA, October, 2007

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-02	12/14/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	202.332	202.332	ng/kg		0.01	2.023
HB-WSD-02	12/14/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	37.189	37.189	ng/kg		0.01	0.372
HB-WSD-02	12/14/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.021	2.021	ng/kg	J	0.01	0.020
HB-WSD-02	12/14/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.575	2.575	ng/kg	J	0.1	0.258
HB-WSD-02	12/14/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.218	3.218	ng/kg	J	0.1	0.322
HB-WSD-02	12/14/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	13.061	13.061	ng/kg	J	0.1	1.306
HB-WSD-02	12/14/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.394	1.394	ng/kg	J	0.1	0.139
HB-WSD-02	12/14/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.94	6.94	ng/kg	J	0.1	0.694
HB-WSD-02	12/14/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.247	0.1235	ng/kg	UU	0.1	0.012
HB-WSD-02	12/14/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	4.717	4.717	ng/kg		1	4.717
HB-WSD-02	12/14/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.109	1.109	ng/kg	J	0.03	0.033
HB-WSD-02	12/14/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.206	0.103	ng/kg	U	1	0.103
HB-WSD-02	12/14/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.318	2.318	ng/kg		0.1	0.232
HB-WSD-02	12/14/2006	0	0.5	3268-87-9	OCDD	Y	1226.67	1226.67	ng/kg		0.0003	0.368
HB-WSD-02	12/14/2006	0	0.5	39001-02-0	OCDF	Y	79.575	79.575	ng/kg		0.0003	0.024
Sample Location TEQ =												10.6
HB-WSD-02	12/14/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	435.541	435.541	ng/kg		0.01	4.355
HB-WSD-02	12/14/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	253.166	253.166	ng/kg		0.01	2.532
HB-WSD-02	12/14/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.815	8.815	ng/kg		0.01	0.088
HB-WSD-02	12/14/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.653	3.653	ng/kg	J	0.1	0.365
HB-WSD-02	12/14/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	17.476	17.476	ng/kg		0.1	1.748
HB-WSD-02	12/14/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	58.048	58.048	ng/kg	J	0.1	5.805
HB-WSD-02	12/14/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.795	7.795	ng/kg		0.1	0.780
HB-WSD-02	12/14/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	16.455	16.455	ng/kg	J	0.1	1.646
HB-WSD-02	12/14/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.233	0.233	ng/kg	J	0.1	0.023
HB-WSD-02	12/14/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	8.221	8.221	ng/kg		1	8.221
HB-WSD-02	12/14/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	6.786	6.786	ng/kg		0.03	0.204
HB-WSD-02	12/14/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.856	1.856	ng/kg		1	1.856
HB-WSD-02	12/14/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	12.507	12.507	ng/kg		0.1	1.251
HB-WSD-02	12/14/2006	0.5	1	3268-87-9	OCDD	Y	2633.947	2633.947	ng/kg	J	0.0003	0.790
HB-WSD-02	12/14/2006	0.5	1	39001-02-0	OCDF	Y	395.443	395.443	ng/kg		0.0003	0.119
Sample Location TEQ =												29.8
HB-WSD-02	12/14/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	310.144	310.144	ng/kg		0.01	3.101
HB-WSD-02	12/14/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	194.746	194.746	ng/kg		0.01	1.947
HB-WSD-02	12/14/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	6.088	6.088	ng/kg		0.01	0.061
HB-WSD-02	12/14/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.167	3.167	ng/kg	J	0.1	0.317
HB-WSD-02	12/14/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	13.04	13.04	ng/kg		0.1	1.304
HB-WSD-02	12/14/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	48.451	48.451	ng/kg	J	0.1	4.845
HB-WSD-02	12/14/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	5.425	5.425	ng/kg		0.1	0.543
HB-WSD-02	12/14/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	15.308	15.308	ng/kg	J	0.1	1.531
HB-WSD-02	12/14/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.194	0.097	ng/kg	U	0.1	0.010
HB-WSD-02	12/14/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	6.904	6.904	ng/kg		1	6.904
HB-WSD-02	12/14/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	3.894	3.894	ng/kg		0.03	0.117
HB-WSD-02	12/14/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.289	0.1445	ng/kg	U	1	0.145
HB-WSD-02	12/14/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	8.535	8.535	ng/kg		0.1	0.854
HB-WSD-02	12/14/2006	1	2	3268-87-9	OCDD	Y	1592.141	1592.141	ng/kg	J	0.0003	0.478
HB-WSD-02	12/14/2006	1	2	39001-02-0	OCDF	Y	250.845	250.845	ng/kg		0.0003	0.075
Sample Location TEQ =												22.2

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-05	12/14/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	30.893	30.893	ng/kg		0.01	0.309
HB-WSD-05	12/14/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.542	8.542	ng/kg		0.01	0.085
HB-WSD-05	12/14/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.509	0.2545	ng/kg	U	0.1	0.025
HB-WSD-05	12/14/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.864	2.864	ng/kg	J	0.1	0.286
HB-WSD-05	12/14/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.796	0.398	ng/kg	U	0.1	0.040
HB-WSD-05	12/14/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.951	0.4755	ng/kg	U	0.1	0.048
HB-WSD-05	12/14/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.363	0.1815	ng/kg	U	1	0.182
HB-WSD-05	12/14/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.471	0.471	ng/kg	EMPC	0.03	0.014
HB-WSD-05	12/14/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.261	0.1305	ng/kg	U	1	0.131
HB-WSD-05	12/14/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.006	1.006	ng/kg		0.1	0.101
HB-WSD-05	12/14/2006	0	0.5	3268-87-9	OCDD	Y	200.178	200.178	ng/kg	J	0.0003	0.060
HB-WSD-05	12/14/2006	0	0.5	39001-02-0	OCDF	Y	10.497	10.497	ng/kg		0.0003	0.003
Sample Location TEQ =												1.3
HB-WSD-05	12/14/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	44.966	44.966	ng/kg		0.01	0.450
HB-WSD-05	12/14/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	20.131	20.131	ng/kg		0.01	0.201
HB-WSD-05	12/14/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.287	1.287	ng/kg	J	0.01	0.013
HB-WSD-05	12/14/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.719	0.719	ng/kg	J	0.1	0.072
HB-WSD-05	12/14/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.299	3.299	ng/kg	J	0.1	0.330
HB-WSD-05	12/14/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	4.337	4.337	ng/kg	J	0.1	0.434
HB-WSD-05	12/14/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.343	1.343	ng/kg	J	0.1	0.134
HB-WSD-05	12/14/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.422	2.422	ng/kg	EMPC	0.1	0.242
HB-WSD-05	12/14/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.512	0.512	ng/kg	EMPC	0.1	0.051
HB-WSD-05	12/14/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	1.455	1.455	ng/kg	J	1	1.455
HB-WSD-05	12/14/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	1.718	1.718	ng/kg	J	0.03	0.052
HB-WSD-05	12/14/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.258	0.129	ng/kg	U	1	0.129
HB-WSD-05	12/14/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	3.994	3.994	ng/kg		0.1	0.399
HB-WSD-05	12/14/2006	0.5	1	3268-87-9	OCDD	Y	313.989	313.989	ng/kg		0.0003	0.094
HB-WSD-05	12/14/2006	0.5	1	39001-02-0	OCDF	Y	35.598	35.598	ng/kg		0.0003	0.011
Sample Location TEQ =												4.1
HB-WSD-05	12/14/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	6.861	6.861	ng/kg		0.01	0.069
HB-WSD-05	12/14/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	2.378	2.378	ng/kg	J	0.01	0.024
HB-WSD-05	12/14/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.308	0.154	ng/kg	U	0.01	0.002
HB-WSD-05	12/14/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.242	0.121	ng/kg	U	0.1	0.012
HB-WSD-05	12/14/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.652	0.652	ng/kg	J	0.1	0.065
HB-WSD-05	12/14/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.667	0.667	ng/kg	J	0.1	0.067
HB-WSD-05	12/14/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.197	0.0985	ng/kg	U	0.1	0.010
HB-WSD-05	12/14/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.225	0.1125	ng/kg	U	0.1	0.011
HB-WSD-05	12/14/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.235	0.1175	ng/kg	U	0.1	0.012
HB-WSD-05	12/14/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	N	0.212	0.106	ng/kg	U	1	0.106
HB-WSD-05	12/14/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	N	0.19	0.095	ng/kg	U	0.03	0.003
HB-WSD-05	12/14/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.252	0.126	ng/kg	U	1	0.126
HB-WSD-05	12/14/2006	1	2	51207-31-9	2,3,7,8-TCDF	N	1.217	0.6085	ng/kg	U	0.1	0.061
HB-WSD-05	12/14/2006	1	2	3268-87-9	OCDD	Y	48.681	48.681	ng/kg		0.0003	0.015
HB-WSD-05	12/14/2006	1	2	39001-02-0	OCDF	Y	3.13	3.13	ng/kg	J	0.0003	0.001
Sample Location TEQ =												0.6

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)	
HB-WSD-07	12/14/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	163.883	163.883	ng/kg	EMPC	0.01	1.639	
HB-WSD-07	12/14/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.73	30.73	ng/kg		0.01	0.307	
HB-WSD-07	12/14/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.919	2.919	ng/kg		0.01	0.029	
HB-WSD-07	12/14/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.346	2.346	ng/kg		0.1	0.235	
HB-WSD-07	12/14/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.502	7.502	ng/kg		0.1	0.750	
HB-WSD-07	12/14/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	11.073	11.073	ng/kg		0.1	1.107	
HB-WSD-07	12/14/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.545	2.545	ng/kg		0.1	0.255	
HB-WSD-07	12/14/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	10.24	10.24	ng/kg		0.1	1.024	
HB-WSD-07	12/14/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.983	0.983	ng/kg		0.1	0.098	
HB-WSD-07	12/14/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	4.888	4.888	ng/kg		1	4.888	
HB-WSD-07	12/14/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.661	4.661	ng/kg		0.03	0.140	
HB-WSD-07	12/14/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	0.99	0.99	ng/kg		1	0.990	
HB-WSD-07	12/14/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	7.925	7.925	ng/kg		0.1	0.793	
HB-WSD-07	12/14/2006	0	0.5	3268-87-9	OCDD	Y	1424.109	1424.109	ng/kg		0.0003	0.427	
HB-WSD-07	12/14/2006	0	0.5	39001-02-0	OCDF	Y	71.604	71.604	ng/kg		0.0003	0.021	
Sample Location TEQ =												12.7	
HB-WSD-07	12/14/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	555.33	555.33	ng/kg	J	0.01	5.553	
HB-WSD-07	12/14/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	96.129	96.129	ng/kg		0.01	0.961	
HB-WSD-07	12/14/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.122	8.122	ng/kg		0.01	0.081	
HB-WSD-07	12/14/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	5.663	5.663	ng/kg		0.1	0.566	
HB-WSD-07	12/14/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	17.377	17.377	ng/kg		0.1	1.738	
HB-WSD-07	12/14/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	33.299	33.299	ng/kg		0.1	3.330	
HB-WSD-07	12/14/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	5.41	5.41	ng/kg		0.1	0.541	
HB-WSD-07	12/14/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	27.191	27.191	ng/kg		0.1	2.719	
HB-WSD-07	12/14/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	2.973	2.973	ng/kg		0.1	0.297	
HB-WSD-07	12/14/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	11.881	11.881	ng/kg		1	11.881	
HB-WSD-07	12/14/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	11.1	11.1	ng/kg		0.03	0.333	
HB-WSD-07	12/14/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	3.764	3.764	ng/kg		1	3.764	
HB-WSD-07	12/14/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	19.742	19.742	ng/kg		0.1	1.974	
HB-WSD-07	12/14/2006	0.5	1	3268-87-9	OCDD	Y	4959.239	4959.239	ng/kg		0.0003	1.488	
HB-WSD-07	12/14/2006	0.5	1	39001-02-0	OCDF	Y	249.411	249.411	ng/kg		0.0003	0.075	
Sample Location TEQ =												35.3	
HB-WSD-07	12/14/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	131.057	131.057	ng/kg	EMPC	0.01	1.311	
HB-WSD-07	12/14/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	17.02	17.02	ng/kg		0.01	0.170	
HB-WSD-07	12/14/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.514	1.514	ng/kg		0.01	0.015	
HB-WSD-07	12/14/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.844	0.844	ng/kg		0.1	0.084	
HB-WSD-07	12/14/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.56	2.56	ng/kg		0.1	0.256	
HB-WSD-07	12/14/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.084	7.084	ng/kg		0.1	0.708	
HB-WSD-07	12/14/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.904	0.904	ng/kg		0.1	0.090	
HB-WSD-07	12/14/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.567	5.567	ng/kg		0.1	0.557	
HB-WSD-07	12/14/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.575	0.575	ng/kg		0.1	0.058	
HB-WSD-07	12/14/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	1.992	1.992	ng/kg		1	1.992	
HB-WSD-07	12/14/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	1.836	1.836	ng/kg		0.03	0.055	
HB-WSD-07	12/14/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	0.606	0.606	ng/kg		1	0.606	
HB-WSD-07	12/14/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	2.88	2.88	ng/kg		0.1	0.288	
HB-WSD-07	12/14/2006	1	2	39001-02-0	OCDF	Y	40.477	40.477	ng/kg		0.0003	0.012	
Sample Location TEQ =												6.2	

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-09	12/13/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	330.224	330.224	ng/kg		0.01	3.302
HB-WSD-09	12/13/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	65.094	65.094	ng/kg		0.01	0.651
HB-WSD-09	12/13/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	1.455	0.7275	ng/kg	U	0.01	0.007
HB-WSD-09	12/13/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	1.2	0.6	ng/kg	U	0.1	0.060
HB-WSD-09	12/13/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.601	12.601	ng/kg		0.1	1.260
HB-WSD-09	12/13/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	23.755	23.755	ng/kg	J	0.1	2.376
HB-WSD-09	12/13/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	5.164	5.164	ng/kg		0.1	0.516
HB-WSD-09	12/13/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	1.115	0.5575	ng/kg	U	0.1	0.056
HB-WSD-09	12/13/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.907	0.4535	ng/kg	U	0.1	0.045
HB-WSD-09	12/13/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	7.195	7.195	ng/kg	EMPC	1	7.195
HB-WSD-09	12/13/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.121	4.121	ng/kg		0.03	0.124
HB-WSD-09	12/13/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.791	1.791	ng/kg		1	1.791
HB-WSD-09	12/13/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	7.393	7.393	ng/kg		0.1	0.739
HB-WSD-09	12/13/2006	0	0.5	3268-87-9	OCDD	Y	2405.134	2405.134	ng/kg	J	0.0003	0.722
HB-WSD-09	12/13/2006	0	0.5	39001-02-0	OCDF	Y	125.131	125.131	ng/kg		0.0003	0.038
Sample Location TEQ =												18.9
HB-WSD-09	12/13/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	231.681	231.681	ng/kg		0.01	2.317
HB-WSD-09	12/13/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	43.357	43.357	ng/kg		0.01	0.434
HB-WSD-09	12/13/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	3.361	1.6805	ng/kg	U	0.01	0.017
HB-WSD-09	12/13/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	1.449	0.7245	ng/kg	U	0.1	0.072
HB-WSD-09	12/13/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	9.724	9.724	ng/kg		0.1	0.972
HB-WSD-09	12/13/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	12.58	12.58	ng/kg	J	0.1	1.258
HB-WSD-09	12/13/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.884	2.884	ng/kg		0.1	0.288
HB-WSD-09	12/13/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	10.476	10.476	ng/kg	J	0.1	1.048
HB-WSD-09	12/13/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.086	0.543	ng/kg	U	0.1	0.054
HB-WSD-09	12/13/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	5.35	5.35	ng/kg		1	5.350
HB-WSD-09	12/13/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	5.93	5.93	ng/kg		0.03	0.178
HB-WSD-09	12/13/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.643	1.643	ng/kg		1	1.643
HB-WSD-09	12/13/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	8.196	8.196	ng/kg		0.1	0.820
HB-WSD-09	12/13/2006	0.5	1	3268-87-9	OCDD	Y	1953.408	1953.408	ng/kg	J	0.0003	0.586
HB-WSD-09	12/13/2006	0.5	1	39001-02-0	OCDF	Y	107.509	107.509	ng/kg		0.0003	0.032
Sample Location TEQ =												15.1
HB-WSD-09	12/13/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	214.007	214.007	ng/kg		0.01	2.140
HB-WSD-09	12/13/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	44.856	44.856	ng/kg		0.01	0.449
HB-WSD-09	12/13/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	3.103	3.103	ng/kg		0.01	0.031
HB-WSD-09	12/13/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.253	7.253	ng/kg		0.1	0.725
HB-WSD-09	12/13/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	14.375	14.375	ng/kg	J	0.1	1.438
HB-WSD-09	12/13/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.277	2.277	ng/kg	J	0.1	0.228
HB-WSD-09	12/13/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	10.671	10.671	ng/kg	J	0.1	1.067
HB-WSD-09	12/13/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.966	0.966	ng/kg	J	0.1	0.097
HB-WSD-09	12/13/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	5.282	5.282	ng/kg		1	5.282
HB-WSD-09	12/13/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	5.545	5.545	ng/kg		0.03	0.166
HB-WSD-09	12/13/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	1.522	1.522	ng/kg	EMPC	1	1.522
HB-WSD-09	12/13/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	9.356	9.356	ng/kg		0.1	0.936
HB-WSD-09	12/13/2006	1	2	3268-87-9	OCDD	Y	1798.069	1798.069	ng/kg	J	0.0003	0.539
HB-WSD-09	12/13/2006	1	2	39001-02-0	OCDF	Y	114.266	114.266	ng/kg		0.0003	0.034
Sample Location TEQ =												14.7

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-14	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	431.849	431.849	ng/kg		0.01	4.318
HB-WSD-14	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	78.327	78.327	ng/kg		0.01	0.783
HB-WSD-14	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	6.357	6.357	ng/kg		0.01	0.064
HB-WSD-14	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.979	3.979	ng/kg		0.1	0.398
HB-WSD-14	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	13.335	13.335	ng/kg		0.1	1.334
HB-WSD-14	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	22.887	22.887	ng/kg		0.1	2.289
HB-WSD-14	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	4.002	4.002	ng/kg		0.1	0.400
HB-WSD-14	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	21.317	21.317	ng/kg		0.1	2.132
HB-WSD-14	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.601	0.601	ng/kg	J	0.1	0.060
HB-WSD-14	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	8.879	8.879	ng/kg		1	8.879
HB-WSD-14	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	11.183	11.183	ng/kg		0.03	0.335
HB-WSD-14	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.88	1.88	ng/kg		1	1.880
HB-WSD-14	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	10.536	10.536	ng/kg		0.1	1.054
HB-WSD-14	12/12/2006	0	0.5	3268-87-9	OCDD	Y	3643.782	3643.782	ng/kg	J	0.0003	1.093
HB-WSD-14	12/12/2006	0	0.5	39001-02-0	OCDF	Y	231.906	231.906	ng/kg		0.0003	0.070
Sample Location TEQ =											25.1	
HB-WSD-14	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	558.12	558.12	ng/kg	J	0.01	5.581
HB-WSD-14	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	118.858	118.858	ng/kg	J	0.01	1.189
HB-WSD-14	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	10.928	10.928	ng/kg	J	0.01	0.109
HB-WSD-14	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	4.824	4.824	ng/kg	J	0.1	0.482
HB-WSD-14	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	28.77	28.77	ng/kg	J	0.1	2.877
HB-WSD-14	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	34.174	34.174	ng/kg	J	0.1	3.417
HB-WSD-14	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.15	7.15	ng/kg	J	0.1	0.715
HB-WSD-14	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	12.308	12.308	ng/kg	J	0.1	1.231
HB-WSD-14	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.547	0.547	ng/kg	J	0.1	0.055
HB-WSD-14	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	12.195	12.195	ng/kg		1	12.195
HB-WSD-14	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	17.302	17.302	ng/kg		0.03	0.519
HB-WSD-14	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	3.208	3.208	ng/kg		1	3.208
HB-WSD-14	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	31.621	31.621	ng/kg		0.1	3.162
HB-WSD-14	12/12/2006	0.5	1	3268-87-9	OCDD	Y	5082.505	5082.505	ng/kg	J	0.0003	1.525
HB-WSD-14	12/12/2006	0.5	1	39001-02-0	OCDF	Y	336.353	336.353	ng/kg		0.0003	0.101
Sample Location TEQ =											36.4	
HB-WSD-14	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	716.665	716.665	ng/kg	J	0.01	7.167
HB-WSD-14	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	137.031	137.031	ng/kg		0.01	1.370
HB-WSD-14	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	12.599	12.599	ng/kg		0.01	0.126
HB-WSD-14	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.4	6.4	ng/kg	J	0.1	0.640
HB-WSD-14	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	33.084	33.084	ng/kg	J	0.1	3.308
HB-WSD-14	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	44.232	44.232	ng/kg	J	0.1	4.423
HB-WSD-14	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	8.271	8.271	ng/kg	J	0.1	0.827
HB-WSD-14	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	24.564	24.564	ng/kg	J	0.1	2.456
HB-WSD-14	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.672	0.672	ng/kg	J	0.1	0.067
HB-WSD-14	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	17.131	17.131	ng/kg		1	17.131
HB-WSD-14	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	19.627	19.627	ng/kg		0.03	0.589
HB-WSD-14	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	5.202	5.202	ng/kg		1	5.202
HB-WSD-14	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	32.37	32.37	ng/kg		0.1	3.237
HB-WSD-14	12/12/2006	1	2	3268-87-9	OCDD	Y	6620.931	6620.931	ng/kg	J	0.0003	1.986
HB-WSD-14	12/12/2006	1	2	39001-02-0	OCDF	Y	386.447	386.447	ng/kg		0.0003	0.116
Sample Location TEQ =											48.6	

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-15	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	753.214	753.214	ng/kg	J	0.01	7.532
HB-WSD-15	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	87.219	87.219	ng/kg		0.01	0.872
HB-WSD-15	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	10.186	10.186	ng/kg		0.01	0.102
HB-WSD-15	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	7.487	7.487	ng/kg		0.1	0.749
HB-WSD-15	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	35.132	35.132	ng/kg		0.1	3.513
HB-WSD-15	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	44.812	44.812	ng/kg		0.1	4.481
HB-WSD-15	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.881	7.881	ng/kg		0.1	0.788
HB-WSD-15	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	32.584	32.584	ng/kg		0.1	3.258
HB-WSD-15	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.227	0.6135	ng/kg	U	0.1	0.061
HB-WSD-15	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	17.989	17.989	ng/kg		1	17.989
HB-WSD-15	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	28.706	28.706	ng/kg		0.03	0.861
HB-WSD-15	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	4.84	4.84	ng/kg		1	4.840
HB-WSD-15	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	40.053	40.053	ng/kg		0.1	4.005
HB-WSD-15	12/12/2006	0	0.5	3268-87-9	OCDD	Y	6052.757	6052.757	ng/kg	J	0.0003	1.816
HB-WSD-15	12/12/2006	0	0.5	39001-02-0	OCDF	Y	237.15	237.15	ng/kg		0.0003	0.071
Sample Location TEQ =												50.9
HB-WSD-15	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	779.206	779.206	ng/kg		0.01	7.792
HB-WSD-15	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	163.755	163.755	ng/kg		0.01	1.638
HB-WSD-15	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	16.513	16.513	ng/kg		0.01	0.165
HB-WSD-15	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	11.517	11.517	ng/kg		0.1	1.152
HB-WSD-15	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	49.737	49.737	ng/kg	J	0.1	4.974
HB-WSD-15	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	63.221	63.221	ng/kg		0.1	6.322
HB-WSD-15	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	10.67	10.67	ng/kg	J	0.1	1.067
HB-WSD-15	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	36.157	36.157	ng/kg		0.1	3.616
HB-WSD-15	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.903	0.903	ng/kg	J	0.1	0.090
HB-WSD-15	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	22.66	22.66	ng/kg		1	22.660
HB-WSD-15	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	32.845	32.845	ng/kg		0.03	0.985
HB-WSD-15	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	7.062	7.062	ng/kg		1	7.062
HB-WSD-15	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	47.633	47.633	ng/kg		0.1	4.763
HB-WSD-15	12/12/2006	0.5	1	3268-87-9	OCDD	Y	4105.959	4105.959	ng/kg		0.0003	1.232
HB-WSD-15	12/12/2006	0.5	1	39001-02-0	OCDF	Y	496.463	496.463	ng/kg		0.0003	0.149
Sample Location TEQ =												63.7
HB-WSD-15	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	2652.667	2652.667	ng/kg		0.01	26.527
HB-WSD-15	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	468.813	468.813	ng/kg		0.01	4.688
HB-WSD-15	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	37.932	37.932	ng/kg		0.01	0.379
HB-WSD-15	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	28.805	28.805	ng/kg	J	0.1	2.881
HB-WSD-15	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	78.979	78.979	ng/kg	EMPC	0.1	7.898
HB-WSD-15	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	195.928	195.928	ng/kg	J	0.1	19.593
HB-WSD-15	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	24.375	24.375	ng/kg		0.1	2.438
HB-WSD-15	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	83.138	83.138	ng/kg	J	0.1	8.314
HB-WSD-15	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	2.662	2.662	ng/kg	J	0.1	0.266
HB-WSD-15	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	58.19	58.19	ng/kg		1	58.190
HB-WSD-15	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	49.563	49.563	ng/kg		0.03	1.487
HB-WSD-15	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	18.091	18.091	ng/kg		1	18.091
HB-WSD-15	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	64.741	64.741	ng/kg		0.1	6.474
HB-WSD-15	12/12/2006	1	2	3268-87-9	OCDD	Y	15853.936	15853.936	ng/kg	J	0.0003	4.756
HB-WSD-15	12/12/2006	1	2	39001-02-0	OCDF	Y	1313.477	1313.477	ng/kg		0.0003	0.394
Sample Location TEQ =												162.4

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-20	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	83.532	83.532	ng/kg		0.01	0.835
HB-WSD-20	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	9.077	9.077	ng/kg		0.01	0.091
HB-WSD-20	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.539	0.539	ng/kg	EMPC	0.01	0.005
HB-WSD-20	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.403	0.403	ng/kg	EMPC	0.1	0.040
HB-WSD-20	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.589	1.589	ng/kg	J	0.1	0.159
HB-WSD-20	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.308	2.308	ng/kg	J	0.1	0.231
HB-WSD-20	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.635	0.635	ng/kg	J	0.1	0.064
HB-WSD-20	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.289	1.289	ng/kg	J	0.1	0.129
HB-WSD-20	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.08	0.04	ng/kg	U	0.1	0.004
HB-WSD-20	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.327	0.327	ng/kg	EMPC	1	0.327
HB-WSD-20	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.499	0.499	ng/kg	J	0.03	0.015
HB-WSD-20	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.158	0.079	ng/kg	U	1	0.079
HB-WSD-20	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.583	0.583	ng/kg	J	0.1	0.058
HB-WSD-20	12/12/2006	0	0.5	3268-87-9	OCDD	Y	594.68	594.68	ng/kg		0.0003	0.178
HB-WSD-20	12/12/2006	0	0.5	39001-02-0	OCDF	Y	29.72	29.72	ng/kg		0.0003	0.009
Sample Location TEQ =											2.2	
HB-WSD-20	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	226.496	226.496	ng/kg		0.01	2.265
HB-WSD-20	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.083	30.083	ng/kg		0.01	0.301
HB-WSD-20	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.537	2.537	ng/kg	J	0.01	0.025
HB-WSD-20	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.032	2.032	ng/kg	J	0.1	0.203
HB-WSD-20	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.203	7.203	ng/kg	EMPC	0.1	0.720
HB-WSD-20	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.428	8.428	ng/kg		0.1	0.843
HB-WSD-20	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.497	2.497	ng/kg	J	0.1	0.250
HB-WSD-20	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.385	5.385	ng/kg		0.1	0.539
HB-WSD-20	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.147	0.147	ng/kg	EMPC	0.1	0.015
HB-WSD-20	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	1.678	1.678	ng/kg	J	1	1.678
HB-WSD-20	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	2.195	2.195	ng/kg	EMPC	0.03	0.066
HB-WSD-20	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	0.322	0.322	ng/kg	EMPC	1	0.322
HB-WSD-20	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	1.894	1.894	ng/kg		0.1	0.189
HB-WSD-20	12/12/2006	0.5	1	3268-87-9	OCDD	Y	1552.764	1552.764	ng/kg	J	0.0003	0.466
HB-WSD-20	12/12/2006	0.5	1	39001-02-0	OCDF	Y	82.442	82.442	ng/kg	EMPC	0.0003	0.025
Sample Location TEQ =											7.9	
HB-WSD-20	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	50.081	50.081	ng/kg		0.01	0.501
HB-WSD-20	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.551	8.551	ng/kg		0.01	0.086
HB-WSD-20	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.796	0.796	ng/kg	J	0.01	0.008
HB-WSD-20	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.621	0.621	ng/kg	EMPC	0.1	0.062
HB-WSD-20	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.638	1.638	ng/kg	J	0.1	0.164
HB-WSD-20	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.739	1.739	ng/kg	J	0.1	0.174
HB-WSD-20	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.631	0.631	ng/kg	J	0.1	0.063
HB-WSD-20	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	0.883	0.883	ng/kg	J	0.1	0.088
HB-WSD-20	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.234	0.117	ng/kg	U	0.1	0.012
HB-WSD-20	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	0.404	0.404	ng/kg	J	1	0.404
HB-WSD-20	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	0.624	0.624	ng/kg	EMPC	0.03	0.019
HB-WSD-20	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.227	0.1135	ng/kg	U	1	0.114
HB-WSD-20	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	N	0.898	0.449	ng/kg	U	0.1	0.045
HB-WSD-20	12/12/2006	1	2	3268-87-9	OCDD	Y	327.841	327.841	ng/kg		0.0003	0.098
HB-WSD-20	12/12/2006	1	2	39001-02-0	OCDF	Y	19.945	19.945	ng/kg		0.0003	0.006
Sample Location TEQ =											1.8	

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-23	12/11/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	370.109	370.109	ng/kg		0.01	3.701
HB-WSD-23	12/11/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	63.501	63.501	ng/kg		0.01	0.635
HB-WSD-23	12/11/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.516	4.516	ng/kg	J	0.01	0.045
HB-WSD-23	12/11/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.367	3.367	ng/kg	J	0.1	0.337
HB-WSD-23	12/11/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.42	12.42	ng/kg		0.1	1.242
HB-WSD-23	12/11/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	21.372	21.372	ng/kg		0.1	2.137
HB-WSD-23	12/11/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.327	3.327	ng/kg	EMPC	0.1	0.333
HB-WSD-23	12/11/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	18.03	18.03	ng/kg		0.1	1.803
HB-WSD-23	12/11/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.562	0.281	ng/kg	U	0.1	0.028
HB-WSD-23	12/11/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	6.695	6.695	ng/kg		1	6.695
HB-WSD-23	12/11/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	9.872	9.872	ng/kg		0.03	0.296
HB-WSD-23	12/11/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.334	2.334	ng/kg	EMPC	1	2.334
HB-WSD-23	12/11/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	13.919	13.919	ng/kg		0.1	1.392
HB-WSD-23	12/11/2006	0	0.5	3268-87-9	OCDD	Y	3055.34	3055.34	ng/kg	J	0.0003	0.917
HB-WSD-23	12/11/2006	0	0.5	39001-02-0	OCDF	Y	154.389	154.389	ng/kg		0.0003	0.046
Sample Location TEQ =											21.9	
HB-WSD-23	12/11/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	169.883	169.883	ng/kg		0.01	1.699
HB-WSD-23	12/11/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.657	30.657	ng/kg		0.01	0.307
HB-WSD-23	12/11/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.856	1.856	ng/kg	J	0.01	0.019
HB-WSD-23	12/11/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.952	0.952	ng/kg	J	0.1	0.095
HB-WSD-23	12/11/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.619	3.619	ng/kg	J	0.1	0.362
HB-WSD-23	12/11/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	9.351	9.351	ng/kg		0.1	0.935
HB-WSD-23	12/11/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.183	1.183	ng/kg	J	0.1	0.118
HB-WSD-23	12/11/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.243	6.243	ng/kg		0.1	0.624
HB-WSD-23	12/11/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.351	0.1755	ng/kg	U	0.1	0.018
HB-WSD-23	12/11/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	1.74	1.74	ng/kg	J	1	1.740
HB-WSD-23	12/11/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	2.729	2.729	ng/kg	J	0.03	0.082
HB-WSD-23	12/11/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	0.638	0.638	ng/kg	J	1	0.638
HB-WSD-23	12/11/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	3.628	3.628	ng/kg		0.1	0.363
HB-WSD-23	12/11/2006	0.5	1	3268-87-9	OCDD	Y	1368.891	1368.891	ng/kg		0.0003	0.411
HB-WSD-23	12/11/2006	0.5	1	39001-02-0	OCDF	Y	68.66	68.66	ng/kg		0.0003	0.021
Sample Location TEQ =											7.4	
HB-WSD-23	12/11/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	175.094	175.094	ng/kg		0.01	1.751
HB-WSD-23	12/11/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.142	30.142	ng/kg		0.01	0.301
HB-WSD-23	12/11/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.127	2.127	ng/kg	J	0.01	0.021
HB-WSD-23	12/11/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.748	2.748	ng/kg	J	0.1	0.275
HB-WSD-23	12/11/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	4.462	4.462	ng/kg		0.1	0.446
HB-WSD-23	12/11/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	9.459	9.459	ng/kg		0.1	0.946
HB-WSD-23	12/11/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.549	1.549	ng/kg	EMPC	0.1	0.155
HB-WSD-23	12/11/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	9.045	9.045	ng/kg		0.1	0.905
HB-WSD-23	12/11/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.257	0.1285	ng/kg	U	0.1	0.013
HB-WSD-23	12/11/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	2.305	2.305	ng/kg	J	1	2.305
HB-WSD-23	12/11/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	2.919	2.919	ng/kg	J	0.03	0.088
HB-WSD-23	12/11/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	0.679	0.679	ng/kg	J	1	0.679
HB-WSD-23	12/11/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	3.992	3.992	ng/kg		0.1	0.399
HB-WSD-23	12/11/2006	1	2	3268-87-9	OCDD	Y	1130.739	1130.739	ng/kg		0.0003	0.339
HB-WSD-23	12/11/2006	1	2	39001-02-0	OCDF	Y	56.102	56.102	ng/kg		0.0003	0.017
Sample Location TEQ =											8.6	

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-25	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	40.851	40.851	ng/kg		0.01	0.409
HB-WSD-25	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	14.532	14.532	ng/kg		0.01	0.145
HB-WSD-25	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.748	0.748	ng/kg	EMPC	0.01	0.007
HB-WSD-25	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.3	0.3	ng/kg	EMPC	0.1	0.030
HB-WSD-25	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.763	1.763	ng/kg	J	0.1	0.176
HB-WSD-25	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.326	2.326	ng/kg	J	0.1	0.233
HB-WSD-25	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.766	0.766	ng/kg	J	0.1	0.077
HB-WSD-25	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.584	1.584	ng/kg	EMPC	0.1	0.158
HB-WSD-25	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.151	0.0755	ng/kg	U	0.1	0.008
HB-WSD-25	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.52	0.52	ng/kg	J	1	0.520
HB-WSD-25	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.911	0.911	ng/kg	J	0.03	0.027
HB-WSD-25	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.103	0.0515	ng/kg	U	1	0.052
HB-WSD-25	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.44	1.44	ng/kg		0.1	0.144
HB-WSD-25	12/12/2006	0	0.5	3268-87-9	OCDD	Y	343.457	343.457	ng/kg		0.0003	0.103
HB-WSD-25	12/12/2006	0	0.5	39001-02-0	OCDF	Y	29.272	29.272	ng/kg		0.0003	0.009
Sample Location TEQ =											2.1	
HB-WSD-25	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	45.622	45.622	ng/kg		0.01	0.456
HB-WSD-25	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	19.263	19.263	ng/kg		0.01	0.193
HB-WSD-25	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.956	0.956	ng/kg	J	0.01	0.010
HB-WSD-25	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.474	0.474	ng/kg	J	0.1	0.047
HB-WSD-25	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.705	2.705	ng/kg	J	0.1	0.271
HB-WSD-25	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.001	3.001	ng/kg	J	0.1	0.300
HB-WSD-25	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.065	1.065	ng/kg	J	0.1	0.107
HB-WSD-25	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.075	2.075	ng/kg	J	0.1	0.208
HB-WSD-25	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.061	0.061	ng/kg	EMPC	0.1	0.006
HB-WSD-25	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	0.756	0.756	ng/kg	J	1	0.756
HB-WSD-25	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	1.21	1.21	ng/kg	J	0.03	0.036
HB-WSD-25	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.167	0.0835	ng/kg	U	1	0.084
HB-WSD-25	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	2.579	2.579	ng/kg		0.1	0.258
HB-WSD-25	12/12/2006	0.5	1	3268-87-9	OCDD	Y	339.534	339.534	ng/kg		0.0003	0.102
HB-WSD-25	12/12/2006	0.5	1	39001-02-0	OCDF	Y	32.493	32.493	ng/kg		0.0003	0.010
Sample Location TEQ =											2.8	
HB-WSD-25	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	32.976	32.976	ng/kg		0.01	0.330
HB-WSD-25	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	15.156	15.156	ng/kg		0.01	0.152
HB-WSD-25	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.839	0.839	ng/kg	J	0.01	0.008
HB-WSD-25	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.403	0.403	ng/kg	J	0.1	0.040
HB-WSD-25	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.06	2.06	ng/kg	J	0.1	0.206
HB-WSD-25	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.347	2.347	ng/kg	J	0.1	0.235
HB-WSD-25	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.655	0.655	ng/kg	EMPC	0.1	0.066
HB-WSD-25	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.65	1.65	ng/kg	J	0.1	0.165
HB-WSD-25	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.128	0.128	ng/kg	EMPC	0.1	0.013
HB-WSD-25	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	0.641	0.641	ng/kg	J	1	0.641
HB-WSD-25	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	0.958	0.958	ng/kg	J	0.03	0.029
HB-WSD-25	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.151	0.0755	ng/kg	U	1	0.076
HB-WSD-25	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	2.816	2.816	ng/kg		0.1	0.282
HB-WSD-25	12/12/2006	1	2	3268-87-9	OCDD	Y	256.139	256.139	ng/kg		0.0003	0.077
HB-WSD-25	12/12/2006	1	2	39001-02-0	OCDF	Y	26.054	26.054	ng/kg		0.0003	0.008
Sample Location TEQ =											2.3	

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-29	12/18/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	59.103	59.103	ng/kg	J	0.01	0.591
HB-WSD-29	12/18/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	14.754	14.754	ng/kg	J	0.01	0.148
HB-WSD-29	12/18/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.022	2.022	ng/kg	J	0.01	0.020
HB-WSD-29	12/18/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	1.345	0.6725	ng/kg	UJ	0.1	0.067
HB-WSD-29	12/18/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.145	3.145	ng/kg	J	0.1	0.315
HB-WSD-29	12/18/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.339	1.339	ng/kg	J	0.1	0.134
HB-WSD-29	12/18/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.742	2.742	ng/kg	J	0.1	0.274
HB-WSD-29	12/18/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.438	0.219	ng/kg	UJ	0.1	0.022
HB-WSD-29	12/18/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.201	1.201	ng/kg	J	1	1.201
HB-WSD-29	12/18/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.415	1.415	ng/kg	J	0.03	0.042
HB-WSD-29	12/18/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	3.313	3.313	ng/kg	J	0.1	0.331
HB-WSD-29	12/18/2006	0	0.5	3268-87-9	OCDD	Y	446.659	446.659	ng/kg	J	0.0003	0.134
HB-WSD-29	12/18/2006	0	0.5	39001-02-0	OCDF	Y	38.734	38.734	ng/kg	J	0.0003	0.012
Sample Location TEQ =												3.3
HB-WSD-29	12/18/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	57.037	57.037	ng/kg		0.01	0.570
HB-WSD-29	12/18/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	5.454	5.454	ng/kg		0.01	0.055
HB-WSD-29	12/18/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.672	0.672	ng/kg	J	0.01	0.007
HB-WSD-29	12/18/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.276	0.276	ng/kg	J	0.1	0.028
HB-WSD-29	12/18/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.186	0.186	ng/kg	J	0.1	0.019
HB-WSD-29	12/18/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.512	1.512	ng/kg	J	0.1	0.151
HB-WSD-29	12/18/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.217	0.1085	ng/kg	U	0.1	0.011
HB-WSD-29	12/18/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	0.212	0.212	ng/kg	J	1	0.212
HB-WSD-29	12/18/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	0.113	0.0565	ng/kg	U	0.03	0.002
HB-WSD-29	12/18/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.2	0.1	ng/kg	U	1	0.100
HB-WSD-29	12/18/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	N	0.219	0.1095	ng/kg	U	0.1	0.011
HB-WSD-29	12/18/2006	0.5	1	3268-87-9	OCDD	Y	416.402	416.402	ng/kg		0.0003	0.125
HB-WSD-29	12/18/2006	0.5	1	39001-02-0	OCDF	Y	15.614	15.614	ng/kg		0.0003	0.005
Sample Location TEQ =												1.3

TABLE 2.37b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-29	12/18/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	49.553	49.553	ng/kg		0.01	0.496
HB-WSD-29	12/18/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.577	0.2885	ng/kg	U	0.01	0.003
HB-WSD-29	12/18/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.182	0.091	ng/kg	U	0.1	0.009
HB-WSD-29	12/18/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	6.272	6.272	ng/kg		0.1	0.627
HB-WSD-29	12/18/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.772	1.772	ng/kg	J	0.1	0.177
HB-WSD-29	12/18/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.584	0.584	ng/kg	J	0.1	0.058
HB-WSD-29	12/18/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	N	0.187	0.0935	ng/kg	U	1	0.094
HB-WSD-29	12/18/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	3.669	3.669	ng/kg	J	0.03	0.110
HB-WSD-29	12/18/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.184	0.092	ng/kg	U	1	0.092
HB-WSD-29	12/18/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	1.474	1.474	ng/kg	J	0.1	0.147
HB-WSD-29	12/18/2006	1	2	3268-87-9	OCDD	Y	323.206	323.206	ng/kg	J	0.0003	0.097
HB-WSD-29	12/18/2006	1	2	39001-02-0	OCDF	Y	12.621	12.621	ng/kg	J	0.0003	0.004
Sample Location TEQ =												1.9

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

EMPC = Estimated Maximum Possible Concentration

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223-241.

TABLE 2.37c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE- SYW-12 SURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-WSD-01	0	0.5	12/14/2006	0.783	mg/kg
Highly Chlorinated PCBs	HB-WSD-01	0.5	1	12/14/2006	0.653	mg/kg
Highly Chlorinated PCBs	HB-WSD-01	1	2	12/14/2006	2.241	mg/kg
Highly Chlorinated PCBs	HB-WSD-02	0	0.5	12/14/2006	0.2238	mg/kg
Highly Chlorinated PCBs	HB-WSD-02	0.5	1	12/14/2006	1.057	mg/kg
Highly Chlorinated PCBs	HB-WSD-02	1	2	12/14/2006	1.554	mg/kg
Highly Chlorinated PCBs	HB-WSD-04	1	2	12/14/2006	0.0894	mg/kg
Highly Chlorinated PCBs	HB-WSD-05	0	0.5	12/14/2006	0.1393	mg/kg
Highly Chlorinated PCBs	HB-WSD-05	0.5	1	12/14/2006	0.1834	mg/kg
Highly Chlorinated PCBs	HB-WSD-05	1	2	12/14/2006	0.0859	mg/kg
Highly Chlorinated PCBs	HB-WSD-06	0	0.5	12/13/2006	0.2169	mg/kg
Highly Chlorinated PCBs	HB-WSD-06	0.5	1	12/13/2006	0.863	mg/kg
Highly Chlorinated PCBs	HB-WSD-06	1	2	12/13/2006	0.1095	mg/kg
Highly Chlorinated PCBs	HB-WSD-07	0	0.5	12/14/2006	0.335	mg/kg
Highly Chlorinated PCBs	HB-WSD-07	0.5	1	12/14/2006	1.032	mg/kg
Highly Chlorinated PCBs	HB-WSD-07	1	2	12/14/2006	0.2101	mg/kg
Highly Chlorinated PCBs	HB-WSD-08	0	0.5	12/13/2006	1.03	mg/kg
Highly Chlorinated PCBs	HB-WSD-08	0.5	1	12/13/2006	0.532	mg/kg
Highly Chlorinated PCBs	HB-WSD-08	1	2	12/13/2006	0.226	mg/kg
Highly Chlorinated PCBs	HB-WSD-09	0	0.5	12/13/2006	0.708	mg/kg
Highly Chlorinated PCBs	HB-WSD-09	0.5	1	12/13/2006	0.534	mg/kg
Highly Chlorinated PCBs	HB-WSD-09	1	2	12/13/2006	0.6	mg/kg
Highly Chlorinated PCBs	HB-WSD-10	0	0.5	12/13/2006	0.2337	mg/kg
Highly Chlorinated PCBs	HB-WSD-10	0.5	1	12/13/2006	0.414	mg/kg
Highly Chlorinated PCBs	HB-WSD-10	1	2	12/13/2006	0.495	mg/kg
Highly Chlorinated PCBs	HB-WSD-11	0	0.5	12/13/2006	1.025	mg/kg
Highly Chlorinated PCBs	HB-WSD-11	0.5	1	12/13/2006	0.0748	mg/kg
Highly Chlorinated PCBs	HB-WSD-11	1	2	12/13/2006	0.391	mg/kg
Highly Chlorinated PCBs	HB-WSD-12	0	0.5	12/13/2006	0.0653	mg/kg
Highly Chlorinated PCBs	HB-WSD-12	0.5	1	12/13/2006	0.758	mg/kg
Highly Chlorinated PCBs	HB-WSD-12	1	2	12/13/2006	0.527	mg/kg
Highly Chlorinated PCBs	HB-WSD-13	0	0.5	12/13/2006	0.533	mg/kg
Highly Chlorinated PCBs	HB-WSD-13	0.5	1	12/13/2006	0.618	mg/kg
Highly Chlorinated PCBs	HB-WSD-13	1	2	12/13/2006	0.455	mg/kg
Highly Chlorinated PCBs	HB-WSD-14	0	0.5	12/12/2006	0.596	mg/kg
Highly Chlorinated PCBs	HB-WSD-14	0.5	1	12/12/2006	1.232	mg/kg
Highly Chlorinated PCBs	HB-WSD-14	1	2	12/12/2006	1.77	mg/kg
Highly Chlorinated PCBs	HB-WSD-15	0	0.5	12/12/2006	0.698	mg/kg
Highly Chlorinated PCBs	HB-WSD-15	0.5	1	12/12/2006	1.323	mg/kg
Highly Chlorinated PCBs	HB-WSD-15	1	2	12/12/2006	1.483	mg/kg
Highly Chlorinated PCBs	HB-WSD-16	0	0.5	12/11/2006	3.47	mg/kg
Highly Chlorinated PCBs	HB-WSD-16	0.5	1	12/11/2006	2.218	mg/kg
Highly Chlorinated PCBs	HB-WSD-16	1	2	12/11/2006	1.32	mg/kg
Highly Chlorinated PCBs	HB-WSD-17	0	0.5	12/12/2006	2.22	mg/kg
Highly Chlorinated PCBs	HB-WSD-17	0.5	1	12/12/2006	1.808	mg/kg

TABLE 2.37c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE- SYW-12 SURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-WSD-17	1	2	12/12/2006	0.947	mg/kg
Highly Chlorinated PCBs	HB-WSD-18	0	0.5	12/12/2006	2.859	mg/kg
Highly Chlorinated PCBs	HB-WSD-18	0.5	1	12/12/2006	1.482	mg/kg
Highly Chlorinated PCBs	HB-WSD-18	1	2	12/12/2006	0.594	mg/kg
Highly Chlorinated PCBs	HB-WSD-20	0	0.5	12/12/2006	0.0939	mg/kg
Highly Chlorinated PCBs	HB-WSD-20	0.5	1	12/12/2006	0.1179	mg/kg
Highly Chlorinated PCBs	HB-WSD-20	1	2	12/12/2006	0.117	mg/kg
Highly Chlorinated PCBs	HB-WSD-22	0	0.5	12/11/2006	0.0311	mg/kg
Highly Chlorinated PCBs	HB-WSD-22	0.5	1	12/11/2006	0.0321	mg/kg
Highly Chlorinated PCBs	HB-WSD-23	0	0.5	12/11/2006	1.683	mg/kg
Highly Chlorinated PCBs	HB-WSD-23	0.5	1	12/11/2006	1.635	mg/kg
Highly Chlorinated PCBs	HB-WSD-23	1	2	12/11/2006	1.012	mg/kg
Highly Chlorinated PCBs	HB-WSD-24	0	0.5	12/11/2006	2.48	mg/kg
Highly Chlorinated PCBs	HB-WSD-24	0.5	1	12/11/2006	0.45	mg/kg
Highly Chlorinated PCBs	HB-WSD-24	1	2	12/11/2006	1.515	mg/kg
Highly Chlorinated PCBs	HB-WSD-25	0	0.5	12/12/2006	0.1494	mg/kg
Highly Chlorinated PCBs	HB-WSD-25	0.5	1	12/12/2006	0.111	mg/kg
Highly Chlorinated PCBs	HB-WSD-25	1	2	12/12/2006	0.1882	mg/kg
Highly Chlorinated PCBs	HB-WSD-26	0	0.5	12/18/2006	0.655	mg/kg
Highly Chlorinated PCBs	HB-WSD-26	0.5	1	12/18/2006	0.349	mg/kg
Highly Chlorinated PCBs	HB-WSD-26	1	2	12/18/2006	0.0531	mg/kg
Highly Chlorinated PCBs	HB-WSD-27	0	0.5	12/11/2006	0.0374	mg/kg
Highly Chlorinated PCBs	HB-WSD-27	0.5	1	12/11/2006	0.0684	mg/kg
Highly Chlorinated PCBs	HB-WSD-27	1	2	12/11/2006	0.0309	mg/kg
Highly Chlorinated PCBs	HB-WSD-29	0	0.5	12/18/2006	0.833	mg/kg
Highly Chlorinated PCBs	HB-WSD-29	0.5	1	12/18/2006	0.0255	mg/kg
Highly Chlorinated PCBs	HB-WSD-30	0	0.5	12/18/2006	0.294	mg/kg
Highly Chlorinated PCBs	HB-WSD-30	0.5	1	12/18/2006	0.0227	mg/kg
Total PCBs	HB-WSD-01	0	0.5	12/14/2006	0.783	mg/kg
Total PCBs	HB-WSD-01	0.5	1	12/14/2006	0.653	mg/kg
Total PCBs	HB-WSD-01	1	2	12/14/2006	2.241	mg/kg
Total PCBs	HB-WSD-02	0	0.5	12/14/2006	0.2238	mg/kg
Total PCBs	HB-WSD-02	0.5	1	12/14/2006	1.057	mg/kg
Total PCBs	HB-WSD-02	1	2	12/14/2006	1.554	mg/kg
Total PCBs	HB-WSD-04	1	2	12/14/2006	0.0894	mg/kg
Total PCBs	HB-WSD-05	0	0.5	12/14/2006	0.1393	mg/kg
Total PCBs	HB-WSD-05	0.5	1	12/14/2006	0.1834	mg/kg
Total PCBs	HB-WSD-05	1	2	12/14/2006	0.0859	mg/kg
Total PCBs	HB-WSD-06	0	0.5	12/13/2006	0.2169	mg/kg
Total PCBs	HB-WSD-06	0.5	1	12/13/2006	0.863	mg/kg
Total PCBs	HB-WSD-06	1	2	12/13/2006	0.1095	mg/kg
Total PCBs	HB-WSD-07	0	0.5	12/14/2006	0.335	mg/kg
Total PCBs	HB-WSD-07	0.5	1	12/14/2006	1.032	mg/kg
Total PCBs	HB-WSD-07	1	2	12/14/2006	0.2101	mg/kg

TABLE 2.37c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE- SYW-12 SURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-WSD-08	0	0.5	12/13/2006	1.03	mg/kg
Total PCBs	HB-WSD-08	0.5	1	12/13/2006	0.532	mg/kg
Total PCBs	HB-WSD-08	1	2	12/13/2006	0.226	mg/kg
Total PCBs	HB-WSD-09	0	0.5	12/13/2006	0.708	mg/kg
Total PCBs	HB-WSD-09	0.5	1	12/13/2006	0.534	mg/kg
Total PCBs	HB-WSD-09	1	2	12/13/2006	0.6	mg/kg
Total PCBs	HB-WSD-10	0	0.5	12/13/2006	0.2337	mg/kg
Total PCBs	HB-WSD-10	0.5	1	12/13/2006	0.414	mg/kg
Total PCBs	HB-WSD-10	1	2	12/13/2006	0.495	mg/kg
Total PCBs	HB-WSD-11	0	0.5	12/13/2006	1.025	mg/kg
Total PCBs	HB-WSD-11	0.5	1	12/13/2006	0.0748	mg/kg
Total PCBs	HB-WSD-11	1	2	12/13/2006	0.391	mg/kg
Total PCBs	HB-WSD-12	0	0.5	12/13/2006	0.0653	mg/kg
Total PCBs	HB-WSD-12	0.5	1	12/13/2006	0.758	mg/kg
Total PCBs	HB-WSD-12	1	2	12/13/2006	0.527	mg/kg
Total PCBs	HB-WSD-13	0	0.5	12/13/2006	0.533	mg/kg
Total PCBs	HB-WSD-13	0.5	1	12/13/2006	0.618	mg/kg
Total PCBs	HB-WSD-13	1	2	12/13/2006	0.455	mg/kg
Total PCBs	HB-WSD-14	0	0.5	12/12/2006	0.596	mg/kg
Total PCBs	HB-WSD-14	0.5	1	12/12/2006	1.232	mg/kg
Total PCBs	HB-WSD-14	1	2	12/12/2006	1.77	mg/kg
Total PCBs	HB-WSD-15	0	0.5	12/12/2006	0.698	mg/kg
Total PCBs	HB-WSD-15	0.5	1	12/12/2006	1.323	mg/kg
Total PCBs	HB-WSD-15	1	2	12/12/2006	1.483	mg/kg
Total PCBs	HB-WSD-16	0	0.5	12/11/2006	3.47	mg/kg
Total PCBs	HB-WSD-16	0.5	1	12/11/2006	2.218	mg/kg
Total PCBs	HB-WSD-16	1	2	12/11/2006	1.32	mg/kg
Total PCBs	HB-WSD-17	0	0.5	12/12/2006	2.22	mg/kg
Total PCBs	HB-WSD-17	0.5	1	12/12/2006	1.808	mg/kg
Total PCBs	HB-WSD-17	1	2	12/12/2006	0.947	mg/kg
Total PCBs	HB-WSD-18	0	0.5	12/12/2006	2.859	mg/kg
Total PCBs	HB-WSD-18	0.5	1	12/12/2006	1.482	mg/kg
Total PCBs	HB-WSD-18	1	2	12/12/2006	0.594	mg/kg
Total PCBs	HB-WSD-20	0	0.5	12/12/2006	0.0939	mg/kg
Total PCBs	HB-WSD-20	0.5	1	12/12/2006	0.1179	mg/kg
Total PCBs	HB-WSD-20	1	2	12/12/2006	0.117	mg/kg
Total PCBs	HB-WSD-22	0	0.5	12/11/2006	0.0311	mg/kg
Total PCBs	HB-WSD-22	0.5	1	12/11/2006	0.0321	mg/kg
Total PCBs	HB-WSD-23	0	0.5	12/11/2006	1.683	mg/kg
Total PCBs	HB-WSD-23	0.5	1	12/11/2006	1.635	mg/kg
Total PCBs	HB-WSD-23	1	2	12/11/2006	1.012	mg/kg
Total PCBs	HB-WSD-24	0	0.5	12/11/2006	2.48	mg/kg
Total PCBs	HB-WSD-24	0.5	1	12/11/2006	0.45	mg/kg
Total PCBs	HB-WSD-24	1	2	12/11/2006	1.515	mg/kg
Total PCBs	HB-WSD-25	0	0.5	12/12/2006	0.1494	mg/kg

TABLE 2.37c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE- SYW-12 SURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-WSD-25	0.5	1	12/12/2006	0.111	mg/kg
Total PCBs	HB-WSD-25	1	2	12/12/2006	0.1882	mg/kg
Total PCBs	HB-WSD-26	0	0.5	12/18/2006	0.655	mg/kg
Total PCBs	HB-WSD-26	0.5	1	12/18/2006	0.349	mg/kg
Total PCBs	HB-WSD-26	1	2	12/18/2006	0.0531	mg/kg
Total PCBs	HB-WSD-27	0	0.5	12/11/2006	0.0374	mg/kg
Total PCBs	HB-WSD-27	0.5	1	12/11/2006	0.0684	mg/kg
Total PCBs	HB-WSD-27	1	2	12/11/2006	0.0309	mg/kg
Total PCBs	HB-WSD-29	0	0.5	12/18/2006	0.833	mg/kg
Total PCBs	HB-WSD-29	0.5	1	12/18/2006	0.0255	mg/kg
Total PCBs	HB-WSD-30	0	0.5	12/18/2006	0.294	mg/kg
Total PCBs	HB-WSD-30	0.5	1	12/18/2006	0.0227	mg/kg

Notes:

* Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.37d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-01	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.021
HB-WSD-01	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.016
Total Chlordane =									0.016
HB-WSD-01	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.021
HB-WSD-01	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.016
Total Chlordane =									0.016
HB-WSD-01	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.05
HB-WSD-01	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									0.05
HB-WSD-02	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0076
HB-WSD-02	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.019
Total Chlordane =									0.0076
HB-WSD-02	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.039
HB-WSD-02	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.029
Total Chlordane =									0.029
HB-WSD-02	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.061
HB-WSD-02	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.046
Total Chlordane =									0.046
HB-WSD-03	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-03	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.0055
HB-WSD-03	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0082
Total Chlordane =									0.0082
HB-WSD-03	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.002
HB-WSD-03	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0012
Total Chlordane =									0.0012
HB-WSD-04	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0023
HB-WSD-04	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0016
Total Chlordane =									0.0016
HB-WSD-04	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.012
HB-WSD-04	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.014
Total Chlordane =									0.014
HB-WSD-04	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.063
HB-WSD-04	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.032
Total Chlordane =									0.032
HB-WSD-05	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0025
HB-WSD-05	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0025
Total Chlordane =									ND
HB-WSD-05	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0022
HB-WSD-05	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.011
Total Chlordane =									0.011
HB-WSD-05	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0021
HB-WSD-05	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0021
Total Chlordane =									ND

TABLE 2.37d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-06	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0044
HB-WSD-06	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0015
Total Chlordane =									0.0015
HB-WSD-06	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-06	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0023
HB-WSD-06	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0019
Total Chlordane =									0.0019
HB-WSD-07	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0049
HB-WSD-07	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0049
Total Chlordane =									ND
HB-WSD-07	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.011
Total Chlordane =									0.011
HB-WSD-07	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.006
Total Chlordane =									0.006
HB-WSD-08	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.011
HB-WSD-08	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.011
Total Chlordane =									ND
HB-WSD-08	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0046
HB-WSD-08	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0064
Total Chlordane =									0.0064
HB-WSD-08	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0047
HB-WSD-08	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0029
Total Chlordane =									0.0029
HB-WSD-09	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0097
HB-WSD-09	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0081
Total Chlordane =									0.0081
HB-WSD-09	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.009
HB-WSD-09	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0068
Total Chlordane =									0.0068
HB-WSD-09	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0046
Total Chlordane =									ND
HB-WSD-10	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0025
HB-WSD-10	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0025
Total Chlordane =									ND
HB-WSD-10	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0024
HB-WSD-10	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0024
Total Chlordane =									ND
HB-WSD-10	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0062
HB-WSD-10	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0052
Total Chlordane =									0.0052
HB-WSD-11	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0029
HB-WSD-11	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0029
Total Chlordane =									ND

TABLE 2.37d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-11	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0021
HB-WSD-11	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0021
Total Chlordane =									ND
HB-WSD-11	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-11	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-12	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-12	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-12	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-12	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0059
Total Chlordane =									0.0059
HB-WSD-13	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0024
HB-WSD-13	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0024
Total Chlordane =									ND
HB-WSD-13	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0081
Total Chlordane =									0.0081
HB-WSD-13	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0038
Total Chlordane =									0.0038
HB-WSD-14	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.012
HB-WSD-14	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-14	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.023
HB-WSD-14	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.023
Total Chlordane =									ND
HB-WSD-14	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.024
HB-WSD-14	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.024
Total Chlordane =									ND
HB-WSD-15	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.012
HB-WSD-15	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-15	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.025
HB-WSD-15	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.025
Total Chlordane =									ND
HB-WSD-15	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.026
HB-WSD-15	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.026
Total Chlordane =									ND
HB-WSD-16	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.063
HB-WSD-16	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.058
Total Chlordane =									0.063
HB-WSD-16	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.063
Total Chlordane =									0.063
HB-WSD-16	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.021
HB-WSD-16	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.021
Total Chlordane =									0.021

TABLE 2.37d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-17	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-17	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-WSD-17	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-17	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-WSD-17	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-17	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-WSD-18	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.075
Total Chlordane =									ND
HB-WSD-18	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-18	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-WSD-18	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.013
HB-WSD-18	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.013
Total Chlordane =									ND
HB-WSD-19	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0042
HB-WSD-19	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0042
Total Chlordane =									ND
HB-WSD-19	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.002
HB-WSD-19	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-WSD-20	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.013
HB-WSD-20	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.013
Total Chlordane =									ND
HB-WSD-20	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.026
HB-WSD-20	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.026
Total Chlordane =									ND
HB-WSD-20	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.032
HB-WSD-20	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.032
Total Chlordane =									ND
HB-WSD-21	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0019
HB-WSD-21	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0019
Total Chlordane =									ND
HB-WSD-21	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0018
HB-WSD-21	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0018
Total Chlordane =									ND
HB-WSD-21	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-21	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-22	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.048
HB-WSD-22	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.048
Total Chlordane =									ND

TABLE 2.37d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-22	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.023
HB-WSD-22	12/11/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.023
Total Chlordane =									ND
HB-WSD-23	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.06
HB-WSD-23	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.047
Total Chlordane =									0.047
HB-WSD-23	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.03
Total Chlordane =									0.03
HB-WSD-23	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.022
HB-WSD-23	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									0.022
HB-WSD-24	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.044
HB-WSD-24	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.056
Total Chlordane =									0.056
HB-WSD-24	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.016
HB-WSD-24	12/11/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.017
Total Chlordane =									0.017
HB-WSD-24	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.034
HB-WSD-24	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.047
Total Chlordane =									0.047
HB-WSD-25	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.012
HB-WSD-25	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-25	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.005
HB-WSD-25	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0033
Total Chlordane =									0.0033
HB-WSD-25	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0048
HB-WSD-25	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0024
Total Chlordane =									0.0024
HB-WSD-26	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.28
HB-WSD-26	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.28
Total Chlordane =									ND
HB-WSD-26	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.067
HB-WSD-26	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.067
Total Chlordane =									ND
HB-WSD-26	12/18/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.038
HB-WSD-26	12/18/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.038
Total Chlordane =									ND
HB-WSD-27	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.13
HB-WSD-27	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.13
Total Chlordane =									ND
HB-WSD-27	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.051
HB-WSD-27	12/11/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.051
Total Chlordane =									ND

TABLE 2.37d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-27	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.046
HB-WSD-27	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.046
Total Chlordane =									ND
HB-WSD-28	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0022
HB-WSD-28	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0022
Total Chlordane =									ND
HB-WSD-28	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-28	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-28	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-28	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-29	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.035
HB-WSD-29	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.035
Total Chlordane =									ND
HB-WSD-29	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0031
HB-WSD-29	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0031
Total Chlordane =									ND
HB-WSD-29	12/18/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0031
HB-WSD-29	12/18/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0031
Total Chlordane =									ND
HB-WSD-30	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.022
HB-WSD-30	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.022
Total Chlordane =									ND
HB-WSD-30	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0064
HB-WSD-30	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0064
Total Chlordane =									ND
HB-WSD-30	12/18/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0032
HB-WSD-30	12/18/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0032
Total Chlordane =									ND

TABLE 2.37e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-WSD-01	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-01	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.007	0.0035
HB-WSD-01	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0058	0.0029
HB-WSD-02	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0057	0.00285
HB-WSD-02	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-02	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0071	0.00355
HB-WSD-03	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-03	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-03	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0058	0.0029
HB-WSD-04	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-04	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0073	0.00365
HB-WSD-04	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-05	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-05	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0066	0.0033
HB-WSD-05	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0062	0.0031
HB-WSD-06	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0065	0.00325
HB-WSD-06	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0012	0.0012
HB-WSD-06	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-07	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-07	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-07	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-08	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-08	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-08	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-09	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-09	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-09	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-10	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0073	0.00365
HB-WSD-10	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.0073	0.0073
HB-WSD-10	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0015	0.0015
HB-WSD-11	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0085	0.00425
HB-WSD-11	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0062	0.0031
HB-WSD-11	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00096	0.00096
HB-WSD-12	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-12	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-12	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.007	0.0035
HB-WSD-13	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-13	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-13	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00077	0.00077
HB-WSD-14	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0016	0.0016
HB-WSD-14	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-14	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-15	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-15	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0075	0.00375
HB-WSD-15	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0077	0.00385
HB-WSD-16	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0086	0.0043
HB-WSD-16	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0011	0.0011
HB-WSD-16	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00086	0.00086
HB-WSD-17	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0014	0.0014
HB-WSD-17	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0058	0.0058
HB-WSD-17	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00089	0.00089
HB-WSD-18	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0013	0.0013
HB-WSD-18	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.001	0.001
HB-WSD-18	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0012	0.0012

TABLE 2.37e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SURFACE SOIL (0-2 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-WSD-19	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0062	0.0031
HB-WSD-19	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0059	0.00295
HB-WSD-20	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00082	0.00082
HB-WSD-20	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00092	0.00092
HB-WSD-20	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0094	0.0047
HB-WSD-21	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00079	0.00079
HB-WSD-21	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00088	0.00088
HB-WSD-21	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0016	0.0016
HB-WSD-22	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0013	0.0013
HB-WSD-22	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00088	0.00088
HB-WSD-23	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00089	0.00089
HB-WSD-23	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0069	0.0069
HB-WSD-23	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0044	0.0044
HB-WSD-24	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0081	0.00405
HB-WSD-24	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0087	0.00435
HB-WSD-24	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0013	0.0013
HB-WSD-25	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0073	0.00365
HB-WSD-25	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.001	0.001
HB-WSD-25	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.002	0.002
HB-WSD-26	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.017	0.0085
HB-WSD-26	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.0098	0.0049
HB-WSD-26	12/18/2006	1	2	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.011	0.0055
HB-WSD-27	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0075	0.00375
HB-WSD-27	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.0074	0.0037
HB-WSD-27	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-28	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0065	0.00325
HB-WSD-28	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0066	0.0033
HB-WSD-28	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-29	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.01	0.005
HB-WSD-29	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0092	0.0046
HB-WSD-29	12/18/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0091	0.00455
HB-WSD-30	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.013	0.0065
HB-WSD-30	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0093	0.00465
HB-WSD-30	12/18/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0095	0.00475

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.38a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
SYW-12 Subsurface Soil	DIOXIN/FURAN (9)															
	1746-01-6	2,3,7,8-TCDD Equivalent	0.000001	0.0002	mg/kg	HB-WSD-15	30/30		1.62E-04			4.26E-06	C	3.90E-06	ca	3.90E-06 Y ASL
	METALS															
	7429-90-5	ALUMINUM	620	14000	mg/kg	HB-WSD-24	103/103	-	1.40E+04			7.82E+03	N	7.61E+03	nc	7.61E+03 Y ASL
	7440-36-0	ANTIMONY	0.19 J	2.1 J	mg/kg	HB-WSD-27	44/103	6.5-13	2.10E+00			3.13E+00	N	3.13E+00	nc	3.13E+00 N BSL
	7440-38-2	ARSENIC	0.77 J	20	mg/kg	HB-WSD-27	99/103	1.3-1.8	2.00E+01		1.60E+01	4.26E-01	C	3.90E-01	ca	3.90E-01 Y TOX
	7440-39-3	BARIIUM	11 J	330	mg/kg	HB-GWS-05	103/103	-	3.30E+02		3.50E+02	1.56E+03	N	5.37E+02	nc	5.37E+02 N BSL
	7440-41-7	BERYLLIUM	0.077 J	0.77 J	mg/kg	HB-WSD-24	103/103	-	7.70E-01		1.40E+01	1.56E+01	N	1.54E+01	nc	1.54E+01 N BSL
	7440-43-9	CADMIUM	0.31 J	100	mg/kg	HB-GWS-05	94/103	1.1-1.8	1.00E+02		2.50E+00	3.91E+00	N	3.70E+00	nc	3.70E+00 Y ASL
	7440-70-2	CALCIUM	22000	400000	mg/kg	HB-SB-57	103/103	-	4.00E+05			NV	NV	NV	N	NUT
	7440-47-3	CHROMIUM ^a	3.2	470 J	mg/kg	HB-GWS-05	103/103	-	4.70E+02			2.35E+01	N	3.01E+01	ca	2.35E+01 Y TOX
	7440-48-4	COBALT	0.4 J	13	mg/kg	HB-WSD-24	103/103	-	1.30E+01			NV	N	9.03E+01	nc	9.03E+01 N BSL
	7440-50-8	COPPER	3.7	450	mg/kg	HB-GWS-05	103/103	-	4.50E+02		2.70E+02	3.13E+02	N	3.13E+02	nc	3.13E+02 Y ASL
	57-12-5	CYANIDE	0.83	3	mg/kg	HB-GWS-05	17/103	0.54-1.7	3.00E+00			1.56E+02	N	1.22E+02	nc	1.22E+02 N BSL
	7439-89-6	IRON	2200 J	31000	mg/kg	HB-WSD-24	103/103	-	3.10E+04			5.48E+03	N	2.35E+03	nc	2.35E+03 Y ASL
	7439-92-1	LEAD	2.1	410	mg/kg	HB-GWS-05	103/103	-	4.10E+02			NV	NV	4.00E+02	nc	4.00E+02 Y ASL
	7439-95-4	MAGNESIUM	2600 J	27000	mg/kg	HB-WSD-28	103/103	-	2.70E+04		2.00E+03	NV	NV	NV	N	NUT
	7439-96-5	MANGANESE	170	630 J	mg/kg	HB-WSD-29	103/103	-	6.30E+02			1.56E+02	N	1.76E+02	nc	1.56E+02 Y ASL
	7439-97-6	MERCURY ^b	0.0047 J	8.6	mg/kg	HB-WSD-18	103/103	-	8.60E+00			2.35E+00	N	2.35E+00	nc	2.35E+00 Y ASL
	22967-92-6	METHYL MERCURY	0.00035	0.0135	mg/kg	HB-WSD-18	58/88	0.000021-0.00415	1.35E-02			7.82E-01	N	6.11E-01	nc	6.11E-01 N BSL
	7440-02-0	NICKEL	2.6 J	110	mg/kg	HB-GWS-05	103/103	-	1.10E+02		1.40E+02	1.56E+02	N	1.56E+02	nc	1.56E+02 N BSL
	7440-09-7	POTASSIUM	170 J	2300	mg/kg	HB-WSD-24	101/103	1400-1400	2.30E+03			NV	NV	NV	N	NUT
	7782-49-2	SELENIUM	0.27 J	2.6	mg/kg	HB-WSD-28	93/103	1.1-2.2	2.60E+00		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-22-4	SILVER	0.13 J	13	mg/kg	HB-GWS-05	77/103	1.1-2.2	1.30E+01		3.60E+01	3.91E+01	N	3.91E+01	nc	3.91E+01 N BSL
	7440-23-5	SODIUM	120 J	2700	mg/kg	HB-SB-52	77/103	110-340	2.70E+03			NV	NV	NV	N	NUT
	7440-62-2	VANADIUM	1.6 J	53	mg/kg	HB-WSD-27	103/103	-	5.30E+01			7.82E+00	N	7.82E+00	nc	7.82E+00 Y ASL
	7440-66-6	ZINC	15	1200	mg/kg	HB-GWS-05	103/103	-	1.20E+03		2.20E+03	2.35E+03	N	2.35E+03	nc	2.35E+03 N BSL
	PCBs															
		LESS CHLORINATED PCBs ^c	0.0288	0.0288	mg/kg	HB-SB-57	1/103	0.0183-0.596	2.88E-02			5.48E-01	N	3.93E-01	nc	3.93E-01 N BSL
		HIGHLY CHLORINATED PCBs ^d	0.0183	3.47	mg/kg	HB-WSD-16	77/101	0.0183-0.323	2.11E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL
		TOTAL PCBs ^e	0.0183	3.47	mg/kg	HB-WSD-16	77/101	0.0183-0.323	2.11E+00			3.19E-01	C	2.22E-02	nc	2.22E-02 Y ASL
	PESTICIDES															
	72-54-8	4,4'-DDD	0.00043 J	0.073 J	mg/kg	HB-WSD-02	12/102	0.0036-0.55	7.30E-02		2.60E+00	2.66E+00	C	2.44E+00	ca	2.44E+00 N BSL
	72-55-9	4,4'-DDE	0.0005 J	0.014 J	mg/kg	HB-WSD-22	5/103	0.0036-0.55	1.40E-02		1.80E+00	1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	50-29-3	4,4'-DDT	0.0025 J	0.1 J	mg/kg	HB-WSD-24	19/90	0.0036-0.55	1.00E-01		1.70E+00	1.88E+00	C	1.72E+00	ca	1.72E+00 N BSL
	319-84-6	ALPHA-BHC	0.00045 J	0.00049 J	mg/kg	HB-WSD-03	2/103	0.0018-0.28	4.90E-04		9.70E-02	1.01E-01	C	9.02E-02	ca	9.02E-02 N BSL
	57-74-9	TOTAL CHLORDANE ^f	0.0007 J	0.063 J	mg/kg	HB-WSD-16	37/103	0.0018-0.28	6.30E-02		9.10E-01	1.82E+00	C	1.62E+00	ca	1.62E+00 N BSL
	60-57-1	DIELDRIN	0.002 J	0.03	mg/kg	HB-WSD-07	10/102	0.0036-0.55	3.00E-02		3.90E-02	3.99E-02	C	3.04E-02	ca	3.04E-02 N BSL
	72-20-8	ENDRIN	0.026 J	0.026 J	mg/kg	HB-WSD-24	1/102	0.0036-0.55	2.60E-02		2.20E+00	2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL
	53494-70-5	ENDRIN KETONE ^g	0.005 J	0.0057 J	mg/kg	HB-WSD-13	2/101	0.0036-0.55	5.70E-03			2.35E+00	N	1.83E+00	nc	1.83E+00 N BSL
	SVOCs															
	92-52-4	1,1'-BIPHENYL	0.047 J	4.9 J	mg/kg	HB-WSD-27	49/104	0.36-11	4.90E+00			3.91E+02	N	3.01E+02	nc	3.01E+02 N BSL
	91-57-6	2-METHYLNAPHTHALENE	0.046 J	16 J	mg/kg	HB-WSD-27	82/104	0.36-9.8	1.60E+01			3.13E+01	N	2.44E+01	nc	2.44E+01 N BSL
	106-47-8	4-CHLOROANILINE	0.059 J	0.2 J	mg/kg	HB-WSD-14	3/104	0.36-25	2.00E-01		3.40E+01	3.13E+01	N	2.44E+01	nc	2.44E+01 N BSL
	106-44-5	4-METHYLPHENOL	0.04 J	1.5 J	mg/kg	HB-GWS-08	27/104	0.36-25	1.50E+00			3.91E+01	N	3.06E+01	nc	3.06E+01 N BSL
	83-32-9	ACENAPHTHENE	0.048 J	33	mg/kg	HB-GWS-08	73/104	0.36-5.6	3.30E+01		1.00E+02	4.69E+02	N	3.68E+02	nc	3.68E+02 N BSL
	208-96-8	ACENAPHTHYLENE	0.046 J	16	mg/kg	HB-GWS-08	90/104	0.36-0.63	1.60E+01		1.00E+02	NV	NV	NV	Y	NTX
	120-12-7	ANTHRACENE	0.047 J	88	mg/kg	HB-WSD-27	93/104	0.36-0.63	8.80E+01		1.00E+02	2.35E+03	N	2.19E+03	nc	2.19E+03 N BSL
	100-52-7	BENZALDEHYDE	0.047 J	1.7 J	mg/kg	HB-WSD-01	41/104	0.36-25	1.70E+00			7.82E+02	N	6.11E+02	nc	6.11E+02 N BSL
	56-55-3	BENZO(A)ANTHRACENE	0.053 J	91	mg/kg	HB-WSD-27	97/104	0.36-0.63	9.10E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	50-32-8	BENZO(A)PYRENE	0.052 J	49	mg/kg	HB-WSD-27	97/104	0.36-0.63	4.90E+01		1.00E+00	2.20E-02	C	6.21E-02	ca	2.20E-02 Y ASL
	205-99-2	BENZO(B)FLUORANTHENE	0.098 J	67	mg/kg	HB-WSD-27	97/104	0.36-0.63	6.70E+01		1.00E+00	2.20E-01	C	6.21E-01	ca	2.20E-01 Y ASL
	191-24-2	BENZO(G,H,I)PERYLENE	0.077 J	15 J	mg/kg	HB-WSD-27	94/104	0.36-0.63	1.50E+01		1.00E+02	NV	NV	NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	0.063 J	24 J	mg/kg	HB-WSD-27	95/104	0.36-0.63	2.40E+01		1.00E+00	2.20E+00	C	6.21E+00	ca	2.20E+00 Y ASL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.041 J	2.2	mg/kg	HB-WSD-04	35/104	0.087-25	2.20E+00			4.56E+01	C	3.47E+01	ca	3.47E+01 N BSL

TABLE 2.38a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0-10 ft bgs)

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Residential Soil (5)	USEPA PRG for Residential Soil (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
	85-68-7	BUTYLBENZYL PHTHALATE	0.051 J	1.2 J	mg/kg	HB-WSD-27	8/104	0.36-25	1.20E+00			1.56E+03	N	1.22E+03	nc	1.22E+03	N	BSL	
	105-60-2	CAPROLACTAM	0.057 J	0.093 J	mg/kg	HB-WSD-23	3/104	0.36-25	9.30E-02			3.91E+03	N	3.06E+03	nc	3.06E+03	N	BSL	
	86-74-8	CARBAZOLE	0.047 J	6.2 J	mg/kg	HB-WSD-27	69/104	0.36-9.8	6.20E+00			3.19E+01	C	2.43E+01	ca	2.43E+01	N	BSL	
	218-01-9	CHRYSENE	0.094 J	89	mg/kg	HB-WSD-27	97/104	0.36-0.63	8.90E+01		1.00E+00	2.20E+01	C	6.21E+01	ca	2.20E+01	Y	ASL	
	53-70-3	DIBENZ(A,H)ANTHRACENE	0.05 J	5.3 J	mg/kg	HB-WSD-27	85/104	0.36-5.6	5.30E+00		3.30E-01	2.20E-02	C	6.21E-02	ca	2.20E-02	Y	ASL	
	132-64-9	DIBENZOFURAN	0.061 J	20 J	mg/kg	HB-WSD-27	65/104	0.36-9.8	2.00E+01		1.40E+01	7.82E+00	N	1.45E+01	nc	7.82E+00	Y	ASL	
	84-74-2	DI-N-BUTYL PHTHALATE	0.15 J	0.15 J	mg/kg	HB-WSD-10	1/104	0.36-25	1.50E-01			7.82E+02	N	6.11E+02	nc	6.11E+02	N	BSL	
	206-44-0	FLUORANTHENE	0.069 J	220	mg/kg	HB-WSD-27	99/104	0.36-0.56	2.20E+02		1.00E+02	3.13E+02	N	2.29E+02	nc	2.29E+02	N	BSL	
	86-73-7	FLUORENE	0.048 J	37	mg/kg	HB-WSD-27	81/104	0.36-4	3.70E+01		1.00E+02	3.13E+02	N	2.75E+02	nc	2.75E+02	N	BSL	
	118-74-1	HEXACHLORO BENZENE	0.048 J	0.24 J	mg/kg	HB-WSD-18	17/104	0.36-25	2.40E-01		3.30E-01	3.99E-01	C	3.04E-01	ca	3.04E-01	N	BSL	
	193-39-5	INDENO(1,2,3-CD)PYRENE	0.052 J	13 J	mg/kg	HB-WSD-27	94/104	0.36-0.63	1.30E+01		5.00E-01	2.20E-01	C	6.21E-01	ca	2.20E-01	Y	ASL	
	91-20-3	NAPHTHALENE	0.041 J	11	mg/kg	HB-SB-54	84/104	0.36-9.8	1.10E+01		1.00E+02	1.56E+02	N	5.59E+00	nc	5.59E+00	Y	ASL	
	85-01-8	PHENANTHRENE	0.073 J	200	mg/kg	HB-WSD-27	97/104	0.36-0.63	2.00E+02		1.00E+02	NV	NV	NV	NV	Y	NTX		
	108-95-2	PHENOL	0.048 J	0.071 J	mg/kg	HB-WSD-16	4/104	0.36-25	7.10E-02		1.00E+02	2.35E+03	N	1.83E+03	nc	1.83E+03	N	BSL	
	129-00-0	PYRENE	0.07 J	190	mg/kg	HB-WSD-27	98/104	0.36-0.6	1.90E+02		1.00E+02	2.35E+02	N	2.32E+02	nc	2.32E+02	N	BSL	
	VOCs																		
	120-82-1	1,2,4-TRICHLORO BENZENE	0.02 J	0.079 J	mg/kg	HB-GWS-05	2/102	0.0054-3.4	7.90E-02				7.82E+01	N	6.22E+00	nc	6.22E+00	N	BSL
	95-50-1	1,2-DICHLORO BENZENE	0.0009 J	0.019 J	mg/kg	HB-GWS-05	9/102	0.0027-1.7	1.90E-02			1.00E+02	7.04E+02	N	6.00E+01	nc	6.00E+01	N	BSL
	541-73-1	1,3-DICHLORO BENZENE	0.0011 J	0.15	mg/kg	HB-GWS-05	3/102	0.0027-1.7	1.50E-01			1.70E+01	2.35E+01	N	5.31E+01	nc	2.35E+01	N	BSL
	106-46-7	1,4-DICHLORO BENZENE	0.00069 J	0.21	mg/kg	HB-GWS-05	33/102	0.0027-1.7	2.10E-01			9.80E+00	2.66E+01	C	3.45E+00	ca	3.45E+00	N	BSL
78-93-3	2-BUTANONE	0.0021 J	0.22	mg/kg	HB-GWS-05	15/103	0.011-6.8	2.20E-01			1.00E+02	4.69E+03	N	2.23E+03	nc	2.23E+03	N	BSL	
67-64-1	ACETONE	0.019 J	0.73	mg/kg	HB-GWS-05	3/103	0.011-6.8	7.30E-01			1.00E+02	7.04E+03	N	1.41E+03	nc	1.41E+03	N	BSL	
98-86-2	ACETOPHENONE	0.048 J	0.76 J	mg/kg	HB-WSD-01	47/104	0.36-25	7.60E-01				1.16E+01	C	NV		1.16E+01	N	BSL	
71-43-2	BENZENE	0.00073 J	0.0043	mg/kg	HB-WSD-13	4/103	0.0027-1.7	4.30E-03			2.90E+00	7.82E+02	N	3.55E+02	ca	3.55E+02	Y	TOX	
75-15-0	CARBON DISULFIDE	0.00072 J	0.019 J	mg/kg	HB-GWS-01	23/103	0.0027-1.7	1.90E-02				1.56E+02	N	1.51E+01	nc	1.51E+01	N	BSL	
108-90-7	CHLORO BENZENE	0.00064 J	0.077	mg/kg	HB-GWS-05	25/103	0.0027-1.7	7.70E-02			1.00E+02	7.82E+01	N	4.29E+00	nc	4.29E+00	N	BSL	
156-59-2	CIS-1,2-DICHLOROETHENE	0.0014 J	0.0014 J	mg/kg	HB-WSD-13	1/103	0.0027-1.7	1.40E-03			5.90E+01	7.82E+02	N	NV	NV	7.82E+02	N	BSL	
110-82-7	CYCLOHEXANE	0.095	0.095	mg/kg	HB-MW-22	1/103	0.0027-1.7	9.50E-02				NV	2.21E+03	nc	2.21E+03	N	BSL		
100-41-4	ETHYLBENZENE	0.00074 J	2.4	mg/kg	HB-SB-52	13/103	0.0027-0.039	2.40E+00			3.00E+01	NV	NV	1.40E+01	nc	1.40E+01	N	BSL	
98-82-8	ISOPROPYLBENZENE	0.0008 J	1.1 J	mg/kg	HB-SB-52	7/102	0.0027-0.31	1.10E+00				7.82E+02	N	3.95E+02	sat	3.95E+02	N	BSL	
79-20-9	METHYL ACETATE	0.0011 J	0.0079 J	mg/kg	HB-SB-57	2/103	0.0027-1.7	7.90E-03				7.82E+02	N	5.72E+01	nc	5.72E+01	N	BSL	
108-87-2	METHYLCYCLOHEXANE	0.0092 J	0.02 J	mg/kg	HB-MW-22	2/103	0.0027-1.7	2.00E-02				NV	2.59E+02	nc	2.59E+02	N	BSL		
75-09-2	METHYLENE CHLORIDE	0.0093 J	0.08 J	mg/kg	HB-MW-26	2/103	0.0054-3.4	8.00E-02			5.10E+01	8.52E+01	C	9.11E+00	ca	9.11E+00	N	BSL	
127-18-4	TETRACHLOROETHENE	0.0006 J	0.0041	mg/kg	HB-WSD-13	2/103	0.0027-1.7	4.10E-03			5.50E+00	1.18E+00	C	4.84E-01	ca	4.84E-01	N	BSL	
108-88-3	TOLUENE	0.00058 J	0.2 J	mg/kg	HB-GWS-07	31/103	0.0027-1.7	2.00E-01			1.00E+02	6.26E+02	N	5.20E+01	nc	5.20E+01	N	BSL	
79-01-6	TRICHLOROETHENE	0.0043	0.0043	mg/kg	HB-WSD-13	1/103	0.0027-1.7	4.30E-03			1.00E+01	1.60E+00	C	5.30E-02	ca	5.30E-02	N	BSL	
1330-20-7	XYLENES, TOTAL	0.00077 J	0.052 J	mg/kg	HB-GWS-01	34/104	0.00285-1.7	5.20E-02			1.00E+02	1.56E+03	N	2.71E+01	nc	2.71E+01	N	BSL	

Footnotes:

- (1) J - estimated value; N - tentatively identified at an estimated value
(2) Concentration used for screening is the maximum detected concentration.
(3) No background screening performed.
(4) Values are from New York Subpart 375-6 Soil Cleanup Objectives (SCO). Values reflect residential restricted use for the protection of human health.
(5) USEPA Region 3 RBCs (USEPA 2007) for residential soil; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.
(6) USEPA Region 9 PRGs (USEPA 2004) for residential soil; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.
(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.
(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level
(9) Based on use of WHO toxicity equivalency factors for dioxins and dioxin-like compounds from Van den Berg et al. (2006); see Table 2.38b.
- = Compound detected in 100% of samples.
NA = Not applicable, minimum and maximum values are calculated.
a = RBC and PRG values for chromium VI utilized.
b = RBC and PRG values for mercury compounds utilized.
c = When detected, reflects summary statistics of Aroclor 1221, 1232, 1016, and 1242. RBC and PRG values for Aroclor-1016 (CAS# 12674112) utilized. Range of detection limits based on Aroclor 1016.
d = When detected, reflects summary statistics of Aroclors 1248, 1254, and 1260. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
e = Reflects summary statistics of all detected Aroclors. RBC and PRG values for Aroclor-1254 (CAS# 11097691) utilized. Range of detection limits based on Aroclor 1254.
f = RBC value for chlordane (CAS# 57749) and PRG value for technical chlordane (CAS# 12789-03-6) utilized.
g = RBC and PRG values for Endrin (CAS# 72208) utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
CAS: Chemical Abstract Service
COPC: Compound of Potential Concern
NUT: Compound is an essential nutrient and is not screened in
NV: No Value
PRG: Preliminary Remediation Goals, USEPA, 2004
RBC: Risk Based Concentration; USEPA, October, 2007
TBC: To Be Considered
USEPA: United States Environmental Protection Agency

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-02	12/14/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	202.332	202.332	ng/kg		0.01	2.023
HB-WSD-02	12/14/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	37.189	37.189	ng/kg		0.01	0.372
HB-WSD-02	12/14/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.021	2.021	ng/kg	J	0.01	0.020
HB-WSD-02	12/14/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.575	2.575	ng/kg	J	0.1	0.258
HB-WSD-02	12/14/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.218	3.218	ng/kg	J	0.1	0.322
HB-WSD-02	12/14/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	13.061	13.061	ng/kg	J	0.1	1.306
HB-WSD-02	12/14/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.394	1.394	ng/kg	J	0.1	0.139
HB-WSD-02	12/14/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.94	6.94	ng/kg	J	0.1	0.694
HB-WSD-02	12/14/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.247	0.1235	ng/kg	UJ	0.1	0.012
HB-WSD-02	12/14/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	4.717	4.717	ng/kg		1	4.717
HB-WSD-02	12/14/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.109	1.109	ng/kg	J	0.03	0.033
HB-WSD-02	12/14/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.206	0.103	ng/kg	U	1	0.103
HB-WSD-02	12/14/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	2.318	2.318	ng/kg		0.1	0.232
HB-WSD-02	12/14/2006	0	0.5	3268-87-9	OCDD	Y	1226.67	1226.67	ng/kg		0.0003	0.368
HB-WSD-02	12/14/2006	0	0.5	39001-02-0	OCDF	Y	79.575	79.575	ng/kg		0.0003	0.024
Sample Location TEQ =												10.6
HB-WSD-02	12/14/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	435.541	435.541	ng/kg		0.01	4.355
HB-WSD-02	12/14/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	253.166	253.166	ng/kg		0.01	2.532
HB-WSD-02	12/14/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.815	8.815	ng/kg		0.01	0.088
HB-WSD-02	12/14/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.653	3.653	ng/kg	J	0.1	0.365
HB-WSD-02	12/14/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	17.476	17.476	ng/kg		0.1	1.748
HB-WSD-02	12/14/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	58.048	58.048	ng/kg	J	0.1	5.805
HB-WSD-02	12/14/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.795	7.795	ng/kg		0.1	0.780
HB-WSD-02	12/14/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	16.455	16.455	ng/kg	J	0.1	1.646
HB-WSD-02	12/14/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.233	0.233	ng/kg	J	0.1	0.023
HB-WSD-02	12/14/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	8.221	8.221	ng/kg		1	8.221
HB-WSD-02	12/14/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	6.786	6.786	ng/kg		0.03	0.204
HB-WSD-02	12/14/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.856	1.856	ng/kg		1	1.856
HB-WSD-02	12/14/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	12.507	12.507	ng/kg		0.1	1.251
HB-WSD-02	12/14/2006	0.5	1	3268-87-9	OCDD	Y	2633.947	2633.947	ng/kg	J	0.0003	0.790
HB-WSD-02	12/14/2006	0.5	1	39001-02-0	OCDF	Y	395.443	395.443	ng/kg		0.0003	0.119
Sample Location TEQ =												29.8
HB-WSD-02	12/14/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	310.144	310.144	ng/kg		0.01	3.101
HB-WSD-02	12/14/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	194.746	194.746	ng/kg		0.01	1.947
HB-WSD-02	12/14/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	6.088	6.088	ng/kg		0.01	0.061
HB-WSD-02	12/14/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.167	3.167	ng/kg	J	0.1	0.317
HB-WSD-02	12/14/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	13.04	13.04	ng/kg		0.1	1.304
HB-WSD-02	12/14/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	48.451	48.451	ng/kg	J	0.1	4.845
HB-WSD-02	12/14/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	5.425	5.425	ng/kg		0.1	0.543
HB-WSD-02	12/14/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	15.308	15.308	ng/kg	J	0.1	1.531
HB-WSD-02	12/14/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.194	0.097	ng/kg	U	0.1	0.010
HB-WSD-02	12/14/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	6.904	6.904	ng/kg		1	6.904
HB-WSD-02	12/14/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	3.894	3.894	ng/kg		0.03	0.117
HB-WSD-02	12/14/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.289	0.1445	ng/kg	U	1	0.145
HB-WSD-02	12/14/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	8.535	8.535	ng/kg		0.1	0.854
HB-WSD-02	12/14/2006	1	2	3268-87-9	OCDD	Y	1592.141	1592.141	ng/kg	J	0.0003	0.478
HB-WSD-02	12/14/2006	1	2	39001-02-0	OCDF	Y	250.845	250.845	ng/kg		0.0003	0.075
Sample Location TEQ =												22.2

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-05	12/14/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	30.893	30.893	ng/kg		0.01	0.309
HB-WSD-05	12/14/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.542	8.542	ng/kg		0.01	0.085
HB-WSD-05	12/14/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.509	0.2545	ng/kg	U	0.1	0.025
HB-WSD-05	12/14/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.864	2.864	ng/kg	J	0.1	0.286
HB-WSD-05	12/14/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.796	0.398	ng/kg	U	0.1	0.040
HB-WSD-05	12/14/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.951	0.4755	ng/kg	U	0.1	0.048
HB-WSD-05	12/14/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	N	0.363	0.1815	ng/kg	U	1	0.182
HB-WSD-05	12/14/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.471	0.471	ng/kg	EMPC	0.03	0.014
HB-WSD-05	12/14/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.261	0.1305	ng/kg	U	1	0.131
HB-WSD-05	12/14/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.006	1.006	ng/kg		0.1	0.101
HB-WSD-05	12/14/2006	0	0.5	3268-87-9	OCDD	Y	200.178	200.178	ng/kg	J	0.0003	0.060
HB-WSD-05	12/14/2006	0	0.5	39001-02-0	OCDF	Y	10.497	10.497	ng/kg		0.0003	0.003
Sample Location TEQ =											1.3	
HB-WSD-05	12/14/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	44.966	44.966	ng/kg		0.01	0.450
HB-WSD-05	12/14/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	20.131	20.131	ng/kg		0.01	0.201
HB-WSD-05	12/14/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.287	1.287	ng/kg	J	0.01	0.013
HB-WSD-05	12/14/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.719	0.719	ng/kg	J	0.1	0.072
HB-WSD-05	12/14/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.299	3.299	ng/kg	J	0.1	0.330
HB-WSD-05	12/14/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	4.337	4.337	ng/kg	J	0.1	0.434
HB-WSD-05	12/14/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.343	1.343	ng/kg	J	0.1	0.134
HB-WSD-05	12/14/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.422	2.422	ng/kg	EMPC	0.1	0.242
HB-WSD-05	12/14/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.512	0.512	ng/kg	EMPC	0.1	0.051
HB-WSD-05	12/14/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	1.455	1.455	ng/kg	J	1	1.455
HB-WSD-05	12/14/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	1.718	1.718	ng/kg	J	0.03	0.052
HB-WSD-05	12/14/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.258	0.129	ng/kg	U	1	0.129
HB-WSD-05	12/14/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	3.994	3.994	ng/kg		0.1	0.399
HB-WSD-05	12/14/2006	0.5	1	3268-87-9	OCDD	Y	313.989	313.989	ng/kg		0.0003	0.094
HB-WSD-05	12/14/2006	0.5	1	39001-02-0	OCDF	Y	35.598	35.598	ng/kg		0.0003	0.011
Sample Location TEQ =											4.1	
HB-WSD-05	12/14/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	6.861	6.861	ng/kg		0.01	0.069
HB-WSD-05	12/14/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	2.378	2.378	ng/kg	J	0.01	0.024
HB-WSD-05	12/14/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.308	0.154	ng/kg	U	0.01	0.002
HB-WSD-05	12/14/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.242	0.121	ng/kg	U	0.1	0.012
HB-WSD-05	12/14/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.652	0.652	ng/kg	J	0.1	0.065
HB-WSD-05	12/14/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	0.667	0.667	ng/kg	J	0.1	0.067
HB-WSD-05	12/14/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	N	0.197	0.0985	ng/kg	U	0.1	0.010
HB-WSD-05	12/14/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	N	0.225	0.1125	ng/kg	U	0.1	0.011
HB-WSD-05	12/14/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.235	0.1175	ng/kg	U	0.1	0.012
HB-WSD-05	12/14/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	N	0.212	0.106	ng/kg	U	1	0.106
HB-WSD-05	12/14/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	N	0.19	0.095	ng/kg	U	0.03	0.003
HB-WSD-05	12/14/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.252	0.126	ng/kg	U	1	0.126
HB-WSD-05	12/14/2006	1	2	51207-31-9	2,3,7,8-TCDF	N	1.217	0.6085	ng/kg	U	0.1	0.061
HB-WSD-05	12/14/2006	1	2	3268-87-9	OCDD	Y	48.681	48.681	ng/kg		0.0003	0.015
HB-WSD-05	12/14/2006	1	2	39001-02-0	OCDF	Y	3.13	3.13	ng/kg	J	0.0003	0.001
Sample Location TEQ =											0.6	

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-07	12/14/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	163.883	163.883	ng/kg		0.01	1.639
HB-WSD-07	12/14/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.73	30.73	ng/kg		0.01	0.307
HB-WSD-07	12/14/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.919	2.919	ng/kg	J	0.01	0.029
HB-WSD-07	12/14/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.346	2.346	ng/kg	J	0.1	0.235
HB-WSD-07	12/14/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.502	7.502	ng/kg		0.1	0.750
HB-WSD-07	12/14/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	11.073	11.073	ng/kg	J	0.1	1.107
HB-WSD-07	12/14/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.545	2.545	ng/kg	J	0.1	0.255
HB-WSD-07	12/14/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	10.24	10.24	ng/kg	J	0.1	1.024
HB-WSD-07	12/14/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.983	0.983	ng/kg	EMPC	0.1	0.098
HB-WSD-07	12/14/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	4.888	4.888	ng/kg		1	4.888
HB-WSD-07	12/14/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.661	4.661	ng/kg		0.03	0.140
HB-WSD-07	12/14/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	0.99	0.99	ng/kg	J	1	0.990
HB-WSD-07	12/14/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	7.925	7.925	ng/kg		0.1	0.793
HB-WSD-07	12/14/2006	0	0.5	3268-87-9	OCDD	Y	1424.109	1424.109	ng/kg	J	0.0003	0.427
HB-WSD-07	12/14/2006	0	0.5	39001-02-0	OCDF	Y	71.604	71.604	ng/kg		0.0003	0.021
Sample Location TEQ =												12.7
HB-WSD-07	12/14/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	555.33	555.33	ng/kg		0.01	5.553
HB-WSD-07	12/14/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	96.129	96.129	ng/kg		0.01	0.961
HB-WSD-07	12/14/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	8.122	8.122	ng/kg		0.01	0.081
HB-WSD-07	12/14/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	5.663	5.663	ng/kg	J	0.1	0.566
HB-WSD-07	12/14/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	17.377	17.377	ng/kg		0.1	1.738
HB-WSD-07	12/14/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	33.299	33.299	ng/kg	J	0.1	3.330
HB-WSD-07	12/14/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	5.41	5.41	ng/kg		0.1	0.541
HB-WSD-07	12/14/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	27.191	27.191	ng/kg	J	0.1	2.719
HB-WSD-07	12/14/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	2.973	2.973	ng/kg	J	0.1	0.297
HB-WSD-07	12/14/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	11.881	11.881	ng/kg		1	11.881
HB-WSD-07	12/14/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	11.1	11.1	ng/kg		0.03	0.333
HB-WSD-07	12/14/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	3.764	3.764	ng/kg		1	3.764
HB-WSD-07	12/14/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	19.742	19.742	ng/kg		0.1	1.974
HB-WSD-07	12/14/2006	0.5	1	3268-87-9	OCDD	Y	4959.239	4959.239	ng/kg	J	0.0003	1.488
HB-WSD-07	12/14/2006	0.5	1	39001-02-0	OCDF	Y	249.411	249.411	ng/kg		0.0003	0.075
Sample Location TEQ =												35.3
HB-WSD-07	12/14/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	131.057	131.057	ng/kg		0.01	1.311
HB-WSD-07	12/14/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	17.02	17.02	ng/kg		0.01	0.170
HB-WSD-07	12/14/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.514	1.514	ng/kg	J	0.01	0.015
HB-WSD-07	12/14/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.844	0.844	ng/kg	J	0.1	0.084
HB-WSD-07	12/14/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.56	2.56	ng/kg	EMPC	0.1	0.256
HB-WSD-07	12/14/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	7.084	7.084	ng/kg		0.1	0.708
HB-WSD-07	12/14/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.904	0.904	ng/kg	EMPC	0.1	0.090
HB-WSD-07	12/14/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.567	5.567	ng/kg		0.1	0.557
HB-WSD-07	12/14/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.575	0.575	ng/kg	EMPC	0.1	0.058
HB-WSD-07	12/14/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	1.992	1.992	ng/kg	J	1	1.992
HB-WSD-07	12/14/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	1.836	1.836	ng/kg	J	0.03	0.055
HB-WSD-07	12/14/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	0.606	0.606	ng/kg	EMPC	1	0.606
HB-WSD-07	12/14/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	2.88	2.88	ng/kg		0.1	0.288
HB-WSD-07	12/14/2006	1	2	39001-02-0	OCDF	Y	40.477	40.477	ng/kg		0.0003	0.012
Sample Location TEQ =												6.2

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-09	12/13/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	330.224	330.224	ng/kg		0.01	3.302
HB-WSD-09	12/13/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	65.094	65.094	ng/kg		0.01	0.651
HB-WSD-09	12/13/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	1.455	0.7275	ng/kg		0.01	0.007
HB-WSD-09	12/13/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	1.2	0.6	ng/kg	U	0.1	0.060
HB-WSD-09	12/13/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.601	12.601	ng/kg		0.1	1.260
HB-WSD-09	12/13/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	23.755	23.755	ng/kg	J	0.1	2.376
HB-WSD-09	12/13/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	5.164	5.164	ng/kg		0.1	0.516
HB-WSD-09	12/13/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	N	1.115	0.5575	ng/kg	U	0.1	0.056
HB-WSD-09	12/13/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.907	0.4535	ng/kg	U	0.1	0.045
HB-WSD-09	12/13/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	7.195	7.195	ng/kg	EMPC	1	7.195
HB-WSD-09	12/13/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	4.121	4.121	ng/kg		0.03	0.124
HB-WSD-09	12/13/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.791	1.791	ng/kg		1	1.791
HB-WSD-09	12/13/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	7.393	7.393	ng/kg		0.1	0.739
HB-WSD-09	12/13/2006	0	0.5	3268-87-9	OCDD	Y	2405.134	2405.134	ng/kg	J	0.0003	0.722
HB-WSD-09	12/13/2006	0	0.5	39001-02-0	OCDF	Y	125.131	125.131	ng/kg		0.0003	0.038
Sample Location TEQ =												18.9
HB-WSD-09	12/13/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	231.681	231.681	ng/kg		0.01	2.317
HB-WSD-09	12/13/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	43.357	43.357	ng/kg		0.01	0.434
HB-WSD-09	12/13/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	3.361	1.6805	ng/kg	U	0.01	0.017
HB-WSD-09	12/13/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	N	1.449	0.7245	ng/kg	U	0.1	0.072
HB-WSD-09	12/13/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	9.724	9.724	ng/kg		0.1	0.972
HB-WSD-09	12/13/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	12.58	12.58	ng/kg	J	0.1	1.258
HB-WSD-09	12/13/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.884	2.884	ng/kg		0.1	0.288
HB-WSD-09	12/13/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	10.476	10.476	ng/kg	J	0.1	1.048
HB-WSD-09	12/13/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.086	0.543	ng/kg	U	0.1	0.054
HB-WSD-09	12/13/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	5.35	5.35	ng/kg		1	5.350
HB-WSD-09	12/13/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	5.93	5.93	ng/kg		0.03	0.178
HB-WSD-09	12/13/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	1.643	1.643	ng/kg		1	1.643
HB-WSD-09	12/13/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	8.196	8.196	ng/kg		0.1	0.820
HB-WSD-09	12/13/2006	0.5	1	3268-87-9	OCDD	Y	1953.408	1953.408	ng/kg	J	0.0003	0.586
HB-WSD-09	12/13/2006	0.5	1	39001-02-0	OCDF	Y	107.509	107.509	ng/kg		0.0003	0.032
Sample Location TEQ =												15.1
HB-WSD-09	12/13/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	214.007	214.007	ng/kg		0.01	2.140
HB-WSD-09	12/13/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	44.856	44.856	ng/kg		0.01	0.449
HB-WSD-09	12/13/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	3.103	3.103	ng/kg		0.01	0.031
HB-WSD-09	12/13/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.253	7.253	ng/kg		0.1	0.725
HB-WSD-09	12/13/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	14.375	14.375	ng/kg	J	0.1	1.438
HB-WSD-09	12/13/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.277	2.277	ng/kg	J	0.1	0.228
HB-WSD-09	12/13/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	10.671	10.671	ng/kg	J	0.1	1.067
HB-WSD-09	12/13/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.966	0.966	ng/kg	J	0.1	0.097
HB-WSD-09	12/13/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	5.282	5.282	ng/kg		1	5.282
HB-WSD-09	12/13/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	5.545	5.545	ng/kg		0.03	0.166
HB-WSD-09	12/13/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	1.522	1.522	ng/kg	EMPC	1	1.522
HB-WSD-09	12/13/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	9.356	9.356	ng/kg		0.1	0.936
HB-WSD-09	12/13/2006	1	2	3268-87-9	OCDD	Y	1798.069	1798.069	ng/kg	J	0.0003	0.539
HB-WSD-09	12/13/2006	1	2	39001-02-0	OCDF	Y	114.266	114.266	ng/kg		0.0003	0.034
Sample Location TEQ =												14.7

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-14	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	431.849	431.849	ng/kg		0.01	4.318
HB-WSD-14	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	78.327	78.327	ng/kg		0.01	0.783
HB-WSD-14	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	6.357	6.357	ng/kg		0.01	0.064
HB-WSD-14	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.979	3.979	ng/kg		0.1	0.398
HB-WSD-14	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	13.335	13.335	ng/kg		0.1	1.334
HB-WSD-14	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	22.887	22.887	ng/kg		0.1	2.289
HB-WSD-14	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	4.002	4.002	ng/kg		0.1	0.400
HB-WSD-14	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	21.317	21.317	ng/kg		0.1	2.132
HB-WSD-14	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.601	0.601	ng/kg	J	0.1	0.060
HB-WSD-14	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	8.879	8.879	ng/kg		1	8.879
HB-WSD-14	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	11.183	11.183	ng/kg		0.03	0.335
HB-WSD-14	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	1.88	1.88	ng/kg		1	1.880
HB-WSD-14	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	10.536	10.536	ng/kg		0.1	1.054
HB-WSD-14	12/12/2006	0	0.5	3268-87-9	OCDD	Y	3643.782	3643.782	ng/kg	J	0.0003	1.093
HB-WSD-14	12/12/2006	0	0.5	39001-02-0	OCDF	Y	231.906	231.906	ng/kg		0.0003	0.070
Sample Location TEQ =												25.1
HB-WSD-14	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	558.12	558.12	ng/kg	J	0.01	5.581
HB-WSD-14	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	118.858	118.858	ng/kg	J	0.01	1.189
HB-WSD-14	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	10.928	10.928	ng/kg	J	0.01	0.109
HB-WSD-14	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	4.824	4.824	ng/kg	J	0.1	0.482
HB-WSD-14	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	28.77	28.77	ng/kg	J	0.1	2.877
HB-WSD-14	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	34.174	34.174	ng/kg	J	0.1	3.417
HB-WSD-14	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.15	7.15	ng/kg	J	0.1	0.715
HB-WSD-14	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	12.308	12.308	ng/kg	J	0.1	1.231
HB-WSD-14	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.547	0.547	ng/kg	J	0.1	0.055
HB-WSD-14	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	12.195	12.195	ng/kg		1	12.195
HB-WSD-14	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	17.302	17.302	ng/kg		0.03	0.519
HB-WSD-14	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	3.208	3.208	ng/kg		1	3.208
HB-WSD-14	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	31.621	31.621	ng/kg		0.1	3.162
HB-WSD-14	12/12/2006	0.5	1	3268-87-9	OCDD	Y	5082.505	5082.505	ng/kg	J	0.0003	1.525
HB-WSD-14	12/12/2006	0.5	1	39001-02-0	OCDF	Y	336.353	336.353	ng/kg		0.0003	0.101
Sample Location TEQ =												36.4
HB-WSD-14	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	716.665	716.665	ng/kg	J	0.01	7.167
HB-WSD-14	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	137.031	137.031	ng/kg		0.01	1.370
HB-WSD-14	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	12.599	12.599	ng/kg		0.01	0.126
HB-WSD-14	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	6.4	6.4	ng/kg	J	0.1	0.640
HB-WSD-14	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	33.084	33.084	ng/kg	J	0.1	3.308
HB-WSD-14	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	44.232	44.232	ng/kg	J	0.1	4.423
HB-WSD-14	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	8.271	8.271	ng/kg	J	0.1	0.827
HB-WSD-14	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	24.564	24.564	ng/kg	J	0.1	2.456
HB-WSD-14	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.672	0.672	ng/kg	J	0.1	0.067
HB-WSD-14	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	17.131	17.131	ng/kg		1	17.131
HB-WSD-14	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	19.627	19.627	ng/kg		0.03	0.589
HB-WSD-14	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	5.202	5.202	ng/kg		1	5.202
HB-WSD-14	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	32.37	32.37	ng/kg		0.1	3.237
HB-WSD-14	12/12/2006	1	2	3268-87-9	OCDD	Y	6620.931	6620.931	ng/kg	J	0.0003	1.986
HB-WSD-14	12/12/2006	1	2	39001-02-0	OCDF	Y	386.447	386.447	ng/kg		0.0003	0.116
Sample Location TEQ =												48.6

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-15	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	753.214	753.214	ng/kg	J	0.01	7.532
HB-WSD-15	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	87.219	87.219	ng/kg		0.01	0.872
HB-WSD-15	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	10.186	10.186	ng/kg		0.01	0.102
HB-WSD-15	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	7.487	7.487	ng/kg		0.1	0.749
HB-WSD-15	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	35.132	35.132	ng/kg		0.1	3.513
HB-WSD-15	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	44.812	44.812	ng/kg		0.1	4.481
HB-WSD-15	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	7.881	7.881	ng/kg		0.1	0.788
HB-WSD-15	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	32.584	32.584	ng/kg		0.1	3.258
HB-WSD-15	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	1.227	0.6135	ng/kg	U	0.1	0.061
HB-WSD-15	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	17.989	17.989	ng/kg		1	17.989
HB-WSD-15	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	28.706	28.706	ng/kg		0.03	0.861
HB-WSD-15	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	4.84	4.84	ng/kg		1	4.840
HB-WSD-15	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	40.053	40.053	ng/kg		0.1	4.005
HB-WSD-15	12/12/2006	0	0.5	3268-87-9	OCDD	Y	6052.757	6052.757	ng/kg	J	0.0003	1.816
HB-WSD-15	12/12/2006	0	0.5	39001-02-0	OCDF	Y	237.15	237.15	ng/kg		0.0003	0.071
Sample Location TEQ =												50.9
HB-WSD-15	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	779.206	779.206	ng/kg		0.01	7.792
HB-WSD-15	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	163.755	163.755	ng/kg		0.01	1.638
HB-WSD-15	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	16.513	16.513	ng/kg		0.01	0.165
HB-WSD-15	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	11.517	11.517	ng/kg		0.1	1.152
HB-WSD-15	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	49.737	49.737	ng/kg	J	0.1	4.974
HB-WSD-15	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	63.221	63.221	ng/kg		0.1	6.322
HB-WSD-15	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	10.67	10.67	ng/kg	J	0.1	1.067
HB-WSD-15	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	36.157	36.157	ng/kg		0.1	3.616
HB-WSD-15	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.903	0.903	ng/kg	J	0.1	0.090
HB-WSD-15	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	22.66	22.66	ng/kg		1	22.660
HB-WSD-15	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	32.845	32.845	ng/kg		0.03	0.985
HB-WSD-15	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	7.062	7.062	ng/kg		1	7.062
HB-WSD-15	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	47.633	47.633	ng/kg		0.1	4.763
HB-WSD-15	12/12/2006	0.5	1	3268-87-9	OCDD	Y	4105.959	4105.959	ng/kg		0.0003	1.232
HB-WSD-15	12/12/2006	0.5	1	39001-02-0	OCDF	Y	496.463	496.463	ng/kg		0.0003	0.149
Sample Location TEQ =												63.7
HB-WSD-15	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	2652.667	2652.667	ng/kg		0.01	26.527
HB-WSD-15	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	468.813	468.813	ng/kg		0.01	4.688
HB-WSD-15	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	37.932	37.932	ng/kg		0.01	0.379
HB-WSD-15	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	28.805	28.805	ng/kg	J	0.1	2.881
HB-WSD-15	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	78.979	78.979	ng/kg	EMPC	0.1	7.898
HB-WSD-15	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	195.928	195.928	ng/kg	J	0.1	19.593
HB-WSD-15	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	24.375	24.375	ng/kg		0.1	2.438
HB-WSD-15	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	83.138	83.138	ng/kg	J	0.1	8.314
HB-WSD-15	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	2.662	2.662	ng/kg	J	0.1	0.266
HB-WSD-15	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	58.19	58.19	ng/kg		1	58.190
HB-WSD-15	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	49.563	49.563	ng/kg		0.03	1.487
HB-WSD-15	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	18.091	18.091	ng/kg		1	18.091
HB-WSD-15	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	64.741	64.741	ng/kg		0.1	6.474
HB-WSD-15	12/12/2006	1	2	3268-87-9	OCDD	Y	15853.936	15853.936	ng/kg	J	0.0003	4.756
HB-WSD-15	12/12/2006	1	2	39001-02-0	OCDF	Y	1313.477	1313.477	ng/kg		0.0003	0.394
Sample Location TEQ =												162.4

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-20	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	83.532	83.532	ng/kg		0.01	0.835
HB-WSD-20	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	9.077	9.077	ng/kg		0.01	0.091
HB-WSD-20	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.539	0.539	ng/kg	EMPC	0.01	0.005
HB-WSD-20	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.403	0.403	ng/kg	EMPC	0.1	0.040
HB-WSD-20	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.589	1.589	ng/kg	J	0.1	0.159
HB-WSD-20	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.308	2.308	ng/kg	J	0.1	0.231
HB-WSD-20	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.635	0.635	ng/kg	J	0.1	0.064
HB-WSD-20	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.289	1.289	ng/kg	J	0.1	0.129
HB-WSD-20	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.08	0.04	ng/kg	U	0.1	0.004
HB-WSD-20	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.327	0.327	ng/kg	EMPC	1	0.327
HB-WSD-20	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.499	0.499	ng/kg	J	0.03	0.015
HB-WSD-20	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.158	0.079	ng/kg	U	1	0.079
HB-WSD-20	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	0.583	0.583	ng/kg	J	0.1	0.058
HB-WSD-20	12/12/2006	0	0.5	3268-87-9	OCDD	Y	594.68	594.68	ng/kg		0.0003	0.178
HB-WSD-20	12/12/2006	0	0.5	39001-02-0	OCDF	Y	29.72	29.72	ng/kg		0.0003	0.009
Sample Location TEQ =											2.2	
HB-WSD-20	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	226.496	226.496	ng/kg		0.01	2.265
HB-WSD-20	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.083	30.083	ng/kg		0.01	0.301
HB-WSD-20	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.537	2.537	ng/kg	J	0.01	0.025
HB-WSD-20	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.032	2.032	ng/kg	J	0.1	0.203
HB-WSD-20	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	7.203	7.203	ng/kg	EMPC	0.1	0.720
HB-WSD-20	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	8.428	8.428	ng/kg		0.1	0.843
HB-WSD-20	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	2.497	2.497	ng/kg	J	0.1	0.250
HB-WSD-20	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	5.385	5.385	ng/kg		0.1	0.539
HB-WSD-20	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.147	0.147	ng/kg	EMPC	0.1	0.015
HB-WSD-20	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	1.678	1.678	ng/kg	J	1	1.678
HB-WSD-20	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	2.195	2.195	ng/kg	EMPC	0.03	0.066
HB-WSD-20	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	0.322	0.322	ng/kg	EMPC	1	0.322
HB-WSD-20	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	1.894	1.894	ng/kg		0.1	0.189
HB-WSD-20	12/12/2006	0.5	1	3268-87-9	OCDD	Y	1552.764	1552.764	ng/kg	J	0.0003	0.466
HB-WSD-20	12/12/2006	0.5	1	39001-02-0	OCDF	Y	82.442	82.442	ng/kg	EMPC	0.0003	0.025
Sample Location TEQ =											7.9	
HB-WSD-20	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	50.081	50.081	ng/kg		0.01	0.501
HB-WSD-20	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	8.551	8.551	ng/kg		0.01	0.086
HB-WSD-20	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.796	0.796	ng/kg	J	0.01	0.008
HB-WSD-20	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.621	0.621	ng/kg	EMPC	0.1	0.062
HB-WSD-20	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.638	1.638	ng/kg	J	0.1	0.164
HB-WSD-20	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	1.739	1.739	ng/kg	J	0.1	0.174
HB-WSD-20	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.631	0.631	ng/kg	J	0.1	0.063
HB-WSD-20	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	0.883	0.883	ng/kg	J	0.1	0.088
HB-WSD-20	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.234	0.117	ng/kg	U	0.1	0.012
HB-WSD-20	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	0.404	0.404	ng/kg	J	1	0.404
HB-WSD-20	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	0.624	0.624	ng/kg	EMPC	0.03	0.019
HB-WSD-20	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.227	0.1135	ng/kg	U	1	0.114
HB-WSD-20	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	N	0.898	0.449	ng/kg	U	0.1	0.045
HB-WSD-20	12/12/2006	1	2	3268-87-9	OCDD	Y	327.841	327.841	ng/kg		0.0003	0.098
HB-WSD-20	12/12/2006	1	2	39001-02-0	OCDF	Y	19.945	19.945	ng/kg		0.0003	0.006
Sample Location TEQ =											1.8	

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-23	12/11/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	370.109	370.109	ng/kg		0.01	3.701
HB-WSD-23	12/11/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	63.501	63.501	ng/kg		0.01	0.635
HB-WSD-23	12/11/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	4.516	4.516	ng/kg	J	0.01	0.045
HB-WSD-23	12/11/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	3.367	3.367	ng/kg	J	0.1	0.337
HB-WSD-23	12/11/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	12.42	12.42	ng/kg		0.1	1.242
HB-WSD-23	12/11/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	21.372	21.372	ng/kg		0.1	2.137
HB-WSD-23	12/11/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	3.327	3.327	ng/kg	EMPC	0.1	0.333
HB-WSD-23	12/11/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	18.03	18.03	ng/kg		0.1	1.803
HB-WSD-23	12/11/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.562	0.281	ng/kg	U	0.1	0.028
HB-WSD-23	12/11/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	6.695	6.695	ng/kg		1	6.695
HB-WSD-23	12/11/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	9.872	9.872	ng/kg		0.03	0.296
HB-WSD-23	12/11/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	Y	2.334	2.334	ng/kg	EMPC	1	2.334
HB-WSD-23	12/11/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	13.919	13.919	ng/kg		0.1	1.392
HB-WSD-23	12/11/2006	0	0.5	3268-87-9	OCDD	Y	3055.34	3055.34	ng/kg	J	0.0003	0.917
HB-WSD-23	12/11/2006	0	0.5	39001-02-0	OCDF	Y	154.389	154.389	ng/kg		0.0003	0.046
Sample Location TEQ =											21.9	
HB-WSD-23	12/11/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	169.883	169.883	ng/kg		0.01	1.699
HB-WSD-23	12/11/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.657	30.657	ng/kg		0.01	0.307
HB-WSD-23	12/11/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	1.856	1.856	ng/kg	J	0.01	0.019
HB-WSD-23	12/11/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.952	0.952	ng/kg	J	0.1	0.095
HB-WSD-23	12/11/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.619	3.619	ng/kg	J	0.1	0.362
HB-WSD-23	12/11/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	9.351	9.351	ng/kg		0.1	0.935
HB-WSD-23	12/11/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.183	1.183	ng/kg	J	0.1	0.118
HB-WSD-23	12/11/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	6.243	6.243	ng/kg		0.1	0.624
HB-WSD-23	12/11/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.351	0.1755	ng/kg	U	0.1	0.018
HB-WSD-23	12/11/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	1.74	1.74	ng/kg	J	1	1.740
HB-WSD-23	12/11/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	2.729	2.729	ng/kg	J	0.03	0.082
HB-WSD-23	12/11/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	Y	0.638	0.638	ng/kg	J	1	0.638
HB-WSD-23	12/11/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	3.628	3.628	ng/kg		0.1	0.363
HB-WSD-23	12/11/2006	0.5	1	3268-87-9	OCDD	Y	1368.891	1368.891	ng/kg		0.0003	0.411
HB-WSD-23	12/11/2006	0.5	1	39001-02-0	OCDF	Y	68.66	68.66	ng/kg		0.0003	0.021
Sample Location TEQ =											7.4	
HB-WSD-23	12/11/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	175.094	175.094	ng/kg		0.01	1.751
HB-WSD-23	12/11/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	30.142	30.142	ng/kg		0.01	0.301
HB-WSD-23	12/11/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.127	2.127	ng/kg	J	0.01	0.021
HB-WSD-23	12/11/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	2.748	2.748	ng/kg	J	0.1	0.275
HB-WSD-23	12/11/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	4.462	4.462	ng/kg		0.1	0.446
HB-WSD-23	12/11/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	9.459	9.459	ng/kg		0.1	0.946
HB-WSD-23	12/11/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.549	1.549	ng/kg	EMPC	0.1	0.155
HB-WSD-23	12/11/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	9.045	9.045	ng/kg		0.1	0.905
HB-WSD-23	12/11/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.257	0.1285	ng/kg	U	0.1	0.013
HB-WSD-23	12/11/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	2.305	2.305	ng/kg	J	1	2.305
HB-WSD-23	12/11/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	2.919	2.919	ng/kg	J	0.03	0.088
HB-WSD-23	12/11/2006	1	2	1746-01-6	2,3,7,8-TCDD	Y	0.679	0.679	ng/kg	J	1	0.679
HB-WSD-23	12/11/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	3.992	3.992	ng/kg		0.1	0.399
HB-WSD-23	12/11/2006	1	2	3268-87-9	OCDD	Y	1130.739	1130.739	ng/kg		0.0003	0.339
HB-WSD-23	12/11/2006	1	2	39001-02-0	OCDF	Y	56.102	56.102	ng/kg		0.0003	0.017
Sample Location TEQ =											8.6	

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-25	12/12/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	40.851	40.851	ng/kg		0.01	0.409
HB-WSD-25	12/12/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	14.532	14.532	ng/kg		0.01	0.145
HB-WSD-25	12/12/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.748	0.748	ng/kg	EMPC	0.01	0.007
HB-WSD-25	12/12/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.3	0.3	ng/kg	EMPC	0.1	0.030
HB-WSD-25	12/12/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	1.763	1.763	ng/kg	J	0.1	0.176
HB-WSD-25	12/12/2006	0	0.5	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.326	2.326	ng/kg	J	0.1	0.233
HB-WSD-25	12/12/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.766	0.766	ng/kg	J	0.1	0.077
HB-WSD-25	12/12/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.584	1.584	ng/kg	EMPC	0.1	0.158
HB-WSD-25	12/12/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.151	0.0755	ng/kg	U	0.1	0.008
HB-WSD-25	12/12/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	0.52	0.52	ng/kg	J	1	0.520
HB-WSD-25	12/12/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	0.911	0.911	ng/kg	J	0.03	0.027
HB-WSD-25	12/12/2006	0	0.5	1746-01-6	2,3,7,8-TCDD	N	0.103	0.0515	ng/kg	U	1	0.052
HB-WSD-25	12/12/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	1.44	1.44	ng/kg		0.1	0.144
HB-WSD-25	12/12/2006	0	0.5	3268-87-9	OCDD	Y	343.457	343.457	ng/kg		0.0003	0.103
HB-WSD-25	12/12/2006	0	0.5	39001-02-0	OCDF	Y	29.272	29.272	ng/kg		0.0003	0.009
Sample Location TEQ =											2.1	
HB-WSD-25	12/12/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	45.622	45.622	ng/kg		0.01	0.456
HB-WSD-25	12/12/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	19.263	19.263	ng/kg		0.01	0.193
HB-WSD-25	12/12/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.956	0.956	ng/kg	J	0.01	0.010
HB-WSD-25	12/12/2006	0.5	1	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.474	0.474	ng/kg	J	0.1	0.047
HB-WSD-25	12/12/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.705	2.705	ng/kg	J	0.1	0.271
HB-WSD-25	12/12/2006	0.5	1	57653-85-7	1,2,3,6,7,8-HXCDD	Y	3.001	3.001	ng/kg	J	0.1	0.300
HB-WSD-25	12/12/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.065	1.065	ng/kg	J	0.1	0.107
HB-WSD-25	12/12/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.075	2.075	ng/kg	J	0.1	0.208
HB-WSD-25	12/12/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.061	0.061	ng/kg	EMPC	0.1	0.006
HB-WSD-25	12/12/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	0.756	0.756	ng/kg	J	1	0.756
HB-WSD-25	12/12/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	Y	1.21	1.21	ng/kg	J	0.03	0.036
HB-WSD-25	12/12/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.167	0.0835	ng/kg	U	1	0.084
HB-WSD-25	12/12/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	Y	2.579	2.579	ng/kg		0.1	0.258
HB-WSD-25	12/12/2006	0.5	1	3268-87-9	OCDD	Y	339.534	339.534	ng/kg		0.0003	0.102
HB-WSD-25	12/12/2006	0.5	1	39001-02-0	OCDF	Y	32.493	32.493	ng/kg		0.0003	0.010
Sample Location TEQ =											2.8	
HB-WSD-25	12/12/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	32.976	32.976	ng/kg		0.01	0.330
HB-WSD-25	12/12/2006	1	2	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	15.156	15.156	ng/kg		0.01	0.152
HB-WSD-25	12/12/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.839	0.839	ng/kg	J	0.01	0.008
HB-WSD-25	12/12/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	Y	0.403	0.403	ng/kg	J	0.1	0.040
HB-WSD-25	12/12/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	2.06	2.06	ng/kg	J	0.1	0.206
HB-WSD-25	12/12/2006	1	2	57653-85-7	1,2,3,6,7,8-HXCDD	Y	2.347	2.347	ng/kg	J	0.1	0.235
HB-WSD-25	12/12/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.655	0.655	ng/kg	EMPC	0.1	0.066
HB-WSD-25	12/12/2006	1	2	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.65	1.65	ng/kg	J	0.1	0.165
HB-WSD-25	12/12/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.128	0.128	ng/kg	EMPC	0.1	0.013
HB-WSD-25	12/12/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	Y	0.641	0.641	ng/kg	J	1	0.641
HB-WSD-25	12/12/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	0.958	0.958	ng/kg	J	0.03	0.029
HB-WSD-25	12/12/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.151	0.0755	ng/kg	U	1	0.076
HB-WSD-25	12/12/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	2.816	2.816	ng/kg		0.1	0.282
HB-WSD-25	12/12/2006	1	2	3268-87-9	OCDD	Y	256.139	256.139	ng/kg		0.0003	0.077
HB-WSD-25	12/12/2006	1	2	39001-02-0	OCDF	Y	26.054	26.054	ng/kg		0.0003	0.008
Sample Location TEQ =											2.3	

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-29	12/18/2006	0	0.5	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	59.103	59.103	ng/kg	J	0.01	0.591
HB-WSD-29	12/18/2006	0	0.5	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	14.754	14.754	ng/kg	J	0.01	0.148
HB-WSD-29	12/18/2006	0	0.5	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	2.022	2.022	ng/kg	J	0.01	0.020
HB-WSD-29	12/18/2006	0	0.5	39227-28-6	1,2,3,4,7,8-HXCDD	N	1.345	0.6725	ng/kg	UJ	0.1	0.067
HB-WSD-29	12/18/2006	0	0.5	70648-26-9	1,2,3,4,7,8-HXCDF	Y	3.145	3.145	ng/kg	J	0.1	0.315
HB-WSD-29	12/18/2006	0	0.5	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.339	1.339	ng/kg	J	0.1	0.134
HB-WSD-29	12/18/2006	0	0.5	19408-74-3	1,2,3,7,8,9-HXCDD	Y	2.742	2.742	ng/kg	J	0.1	0.274
HB-WSD-29	12/18/2006	0	0.5	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.438	0.219	ng/kg	UJ	0.1	0.022
HB-WSD-29	12/18/2006	0	0.5	40321-76-4	1,2,3,7,8-PECDD	Y	1.201	1.201	ng/kg	J	1	1.201
HB-WSD-29	12/18/2006	0	0.5	57117-41-6	1,2,3,7,8-PECDF	Y	1.415	1.415	ng/kg	J	0.03	0.042
HB-WSD-29	12/18/2006	0	0.5	51207-31-9	2,3,7,8-TCDF	Y	3.313	3.313	ng/kg	J	0.1	0.331
HB-WSD-29	12/18/2006	0	0.5	3268-87-9	OCDD	Y	446.659	446.659	ng/kg	J	0.0003	0.134
HB-WSD-29	12/18/2006	0	0.5	39001-02-0	OCDF	Y	38.734	38.734	ng/kg	J	0.0003	0.012
Sample Location TEQ =												3.3
HB-WSD-29	12/18/2006	0.5	1	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	57.037	57.037	ng/kg		0.01	0.570
HB-WSD-29	12/18/2006	0.5	1	67562-39-4	1,2,3,4,6,7,8-HPCDF	Y	5.454	5.454	ng/kg		0.01	0.055
HB-WSD-29	12/18/2006	0.5	1	55673-89-7	1,2,3,4,7,8,9-HPCDF	Y	0.672	0.672	ng/kg	J	0.01	0.007
HB-WSD-29	12/18/2006	0.5	1	70648-26-9	1,2,3,4,7,8-HXCDF	Y	0.276	0.276	ng/kg	J	0.1	0.028
HB-WSD-29	12/18/2006	0.5	1	57117-44-9	1,2,3,6,7,8-HXCDF	Y	0.186	0.186	ng/kg	J	0.1	0.019
HB-WSD-29	12/18/2006	0.5	1	19408-74-3	1,2,3,7,8,9-HXCDD	Y	1.512	1.512	ng/kg	J	0.1	0.151
HB-WSD-29	12/18/2006	0.5	1	72918-21-9	1,2,3,7,8,9-HXCDF	N	0.217	0.1085	ng/kg	U	0.1	0.011
HB-WSD-29	12/18/2006	0.5	1	40321-76-4	1,2,3,7,8-PECDD	Y	0.212	0.212	ng/kg	J	1	0.212
HB-WSD-29	12/18/2006	0.5	1	57117-41-6	1,2,3,7,8-PECDF	N	0.113	0.0565	ng/kg	U	0.03	0.002
HB-WSD-29	12/18/2006	0.5	1	1746-01-6	2,3,7,8-TCDD	N	0.2	0.1	ng/kg	U	1	0.100
HB-WSD-29	12/18/2006	0.5	1	51207-31-9	2,3,7,8-TCDF	N	0.219	0.1095	ng/kg	U	0.1	0.011
HB-WSD-29	12/18/2006	0.5	1	3268-87-9	OCDD	Y	416.402	416.402	ng/kg		0.0003	0.125
HB-WSD-29	12/18/2006	0.5	1	39001-02-0	OCDF	Y	15.614	15.614	ng/kg		0.0003	0.005
Sample Location TEQ =												1.3

TABLE 2.38b
DERIVATION OF TOXIC EQUIVALENTS FOR DIOXINS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 AREA SUBSURFACE SOIL (0-10 ft)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Reported Value	Concentration used for Dioxin Equivalency	Units	Data Qualifier	Dioxin TEF ⁽¹⁾	Calculated Dioxin Equivalency (ng/kg)
HB-WSD-29	12/18/2006	1	2	35822-46-9	1,2,3,4,6,7,8-HPCDD	Y	49.553	49.553	ng/kg		0.01	0.496
HB-WSD-29	12/18/2006	1	2	55673-89-7	1,2,3,4,7,8,9-HPCDF	N	0.577	0.2885	ng/kg	U	0.01	0.003
HB-WSD-29	12/18/2006	1	2	39227-28-6	1,2,3,4,7,8-HXCDD	N	0.182	0.091	ng/kg	U	0.1	0.009
HB-WSD-29	12/18/2006	1	2	70648-26-9	1,2,3,4,7,8-HXCDF	Y	6.272	6.272	ng/kg		0.1	0.627
HB-WSD-29	12/18/2006	1	2	57117-44-9	1,2,3,6,7,8-HXCDF	Y	1.772	1.772	ng/kg	J	0.1	0.177
HB-WSD-29	12/18/2006	1	2	72918-21-9	1,2,3,7,8,9-HXCDF	Y	0.584	0.584	ng/kg	J	0.1	0.058
HB-WSD-29	12/18/2006	1	2	40321-76-4	1,2,3,7,8-PECDD	N	0.187	0.0935	ng/kg	U	1	0.094
HB-WSD-29	12/18/2006	1	2	57117-41-6	1,2,3,7,8-PECDF	Y	3.669	3.669	ng/kg	J	0.03	0.110
HB-WSD-29	12/18/2006	1	2	1746-01-6	2,3,7,8-TCDD	N	0.184	0.092	ng/kg	U	1	0.092
HB-WSD-29	12/18/2006	1	2	51207-31-9	2,3,7,8-TCDF	Y	1.474	1.474	ng/kg	J	0.1	0.147
HB-WSD-29	12/18/2006	1	2	3268-87-9	OCDD	Y	323.206	323.206	ng/kg	J	0.0003	0.097
HB-WSD-29	12/18/2006	1	2	39001-02-0	OCDF	Y	12.621	12.621	ng/kg	J	0.0003	0.004
Sample Location TEQ =												1.9

NOTES:

TCDD/F = Tetra Chlorinated Dibenzo-p-dioxins/Dibenzofurans

PeCDD/F = Penta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HxCDD/F = Hexa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

HpCDD/F = Hepta Chlorinated Dibenzo-p-dioxins/Dibenzofurans

OCDD/F = Octa Chlorinated Dibenzo-p-dioxins/Dibenzofurans

EMPC = Estimated Maximum Possible Concentration

N/A = not applicable

(1) Van den berg, Martin, et al. 2006. The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds. Toxicological Sciences 93(2), 223–241.

TABLE 2.38c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-GWS-01	6	8	12/18/2006	0.02008	mg/kg
Highly Chlorinated PCBs	HB-GWS-04	2	4	12/20/2006	2.343	mg/kg
Highly Chlorinated PCBs	HB-GWS-05	4	6	12/11/2006	2.64	mg/kg
Highly Chlorinated PCBs	HB-GWS-08	8	10	12/13/2006	0.0183	mg/kg
Highly Chlorinated PCBs	HB-MW-22	4	6	12/26/2006	0.0947	mg/kg
Highly Chlorinated PCBs	HB-MW-24	8	10	12/20/2006	0.273	mg/kg
Highly Chlorinated PCBs	HB-MW-25	4	6	12/21/2006	0.0958	mg/kg
Highly Chlorinated PCBs	HB-SB-57	6	8	12/27/2006	0.0147	mg/kg
Highly Chlorinated PCBs	HB-WSD-01	0	0.5	12/14/2006	0.783	mg/kg
Highly Chlorinated PCBs	HB-WSD-01	0.5	1	12/14/2006	0.653	mg/kg
Highly Chlorinated PCBs	HB-WSD-01	1	2	12/14/2006	2.241	mg/kg
Highly Chlorinated PCBs	HB-WSD-02	0	0.5	12/14/2006	0.2238	mg/kg
Highly Chlorinated PCBs	HB-WSD-02	0.5	1	12/14/2006	1.057	mg/kg
Highly Chlorinated PCBs	HB-WSD-02	1	2	12/14/2006	1.554	mg/kg
Highly Chlorinated PCBs	HB-WSD-04	1	2	12/14/2006	0.0894	mg/kg
Highly Chlorinated PCBs	HB-WSD-05	0	0.5	12/14/2006	0.1393	mg/kg
Highly Chlorinated PCBs	HB-WSD-05	0.5	1	12/14/2006	0.1834	mg/kg
Highly Chlorinated PCBs	HB-WSD-05	1	2	12/14/2006	0.0859	mg/kg
Highly Chlorinated PCBs	HB-WSD-06	0	0.5	12/13/2006	0.2169	mg/kg
Highly Chlorinated PCBs	HB-WSD-06	0.5	1	12/13/2006	0.863	mg/kg
Highly Chlorinated PCBs	HB-WSD-06	1	2	12/13/2006	0.1095	mg/kg
Highly Chlorinated PCBs	HB-WSD-07	0	0.5	12/14/2006	0.335	mg/kg
Highly Chlorinated PCBs	HB-WSD-07	0.5	1	12/14/2006	1.032	mg/kg
Highly Chlorinated PCBs	HB-WSD-07	1	2	12/14/2006	0.2101	mg/kg
Highly Chlorinated PCBs	HB-WSD-08	0	0.5	12/13/2006	1.03	mg/kg
Highly Chlorinated PCBs	HB-WSD-08	0.5	1	12/13/2006	0.532	mg/kg
Highly Chlorinated PCBs	HB-WSD-08	1	2	12/13/2006	0.226	mg/kg
Highly Chlorinated PCBs	HB-WSD-09	0	0.5	12/13/2006	0.708	mg/kg
Highly Chlorinated PCBs	HB-WSD-09	0.5	1	12/13/2006	0.534	mg/kg
Highly Chlorinated PCBs	HB-WSD-09	1	2	12/13/2006	0.6	mg/kg
Highly Chlorinated PCBs	HB-WSD-10	0	0.5	12/13/2006	0.2337	mg/kg
Highly Chlorinated PCBs	HB-WSD-10	0.5	1	12/13/2006	0.414	mg/kg
Highly Chlorinated PCBs	HB-WSD-10	1	2	12/13/2006	0.495	mg/kg
Highly Chlorinated PCBs	HB-WSD-11	0	0.5	12/13/2006	1.025	mg/kg
Highly Chlorinated PCBs	HB-WSD-11	0.5	1	12/13/2006	0.0748	mg/kg
Highly Chlorinated PCBs	HB-WSD-11	1	2	12/13/2006	0.391	mg/kg
Highly Chlorinated PCBs	HB-WSD-12	0	0.5	12/13/2006	0.0653	mg/kg
Highly Chlorinated PCBs	HB-WSD-12	0.5	1	12/13/2006	0.758	mg/kg
Highly Chlorinated PCBs	HB-WSD-12	1	2	12/13/2006	0.527	mg/kg
Highly Chlorinated PCBs	HB-WSD-13	0	0.5	12/13/2006	0.533	mg/kg
Highly Chlorinated PCBs	HB-WSD-13	0.5	1	12/13/2006	0.618	mg/kg
Highly Chlorinated PCBs	HB-WSD-13	1	2	12/13/2006	0.455	mg/kg
Highly Chlorinated PCBs	HB-WSD-14	0	0.5	12/12/2006	0.596	mg/kg

TABLE 2.38c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Highly Chlorinated PCBs	HB-WSD-14	0.5	1	12/12/2006	1.232	mg/kg
Highly Chlorinated PCBs	HB-WSD-14	1	2	12/12/2006	1.77	mg/kg
Highly Chlorinated PCBs	HB-WSD-15	0	0.5	12/12/2006	0.698	mg/kg
Highly Chlorinated PCBs	HB-WSD-15	0.5	1	12/12/2006	1.323	mg/kg
Highly Chlorinated PCBs	HB-WSD-15	1	2	12/12/2006	1.483	mg/kg
Highly Chlorinated PCBs	HB-WSD-16	0	0.5	12/11/2006	3.47	mg/kg
Highly Chlorinated PCBs	HB-WSD-16	0.5	1	12/11/2006	2.218	mg/kg
Highly Chlorinated PCBs	HB-WSD-16	1	2	12/11/2006	1.32	mg/kg
Highly Chlorinated PCBs	HB-WSD-17	0	0.5	12/12/2006	2.22	mg/kg
Highly Chlorinated PCBs	HB-WSD-17	0.5	1	12/12/2006	1.808	mg/kg
Highly Chlorinated PCBs	HB-WSD-17	1	2	12/12/2006	0.947	mg/kg
Highly Chlorinated PCBs	HB-WSD-18	0	0.5	12/12/2006	2.859	mg/kg
Highly Chlorinated PCBs	HB-WSD-18	0.5	1	12/12/2006	1.482	mg/kg
Highly Chlorinated PCBs	HB-WSD-18	1	2	12/12/2006	0.594	mg/kg
Highly Chlorinated PCBs	HB-WSD-20	0	0.5	12/12/2006	0.0939	mg/kg
Highly Chlorinated PCBs	HB-WSD-20	0.5	1	12/12/2006	0.1179	mg/kg
Highly Chlorinated PCBs	HB-WSD-20	1	2	12/12/2006	0.117	mg/kg
Highly Chlorinated PCBs	HB-WSD-22	0	0.5	12/11/2006	0.0311	mg/kg
Highly Chlorinated PCBs	HB-WSD-22	0.5	1	12/11/2006	0.0321	mg/kg
Highly Chlorinated PCBs	HB-WSD-23	0	0.5	12/11/2006	1.683	mg/kg
Highly Chlorinated PCBs	HB-WSD-23	0.5	1	12/11/2006	1.635	mg/kg
Highly Chlorinated PCBs	HB-WSD-23	1	2	12/11/2006	1.012	mg/kg
Highly Chlorinated PCBs	HB-WSD-24	0	0.5	12/11/2006	2.48	mg/kg
Highly Chlorinated PCBs	HB-WSD-24	0.5	1	12/11/2006	0.45	mg/kg
Highly Chlorinated PCBs	HB-WSD-24	1	2	12/11/2006	1.515	mg/kg
Highly Chlorinated PCBs	HB-WSD-25	0	0.5	12/12/2006	0.1494	mg/kg
Highly Chlorinated PCBs	HB-WSD-25	0.5	1	12/12/2006	0.111	mg/kg
Highly Chlorinated PCBs	HB-WSD-25	1	2	12/12/2006	0.1882	mg/kg
Highly Chlorinated PCBs	HB-WSD-26	0	0.5	12/18/2006	0.655	mg/kg
Highly Chlorinated PCBs	HB-WSD-26	0.5	1	12/18/2006	0.349	mg/kg
Highly Chlorinated PCBs	HB-WSD-26	1	2	12/18/2006	0.0531	mg/kg
Highly Chlorinated PCBs	HB-WSD-27	0	0.5	12/11/2006	0.0374	mg/kg
Highly Chlorinated PCBs	HB-WSD-27	0.5	1	12/11/2006	0.0684	mg/kg
Highly Chlorinated PCBs	HB-WSD-27	1	2	12/11/2006	0.0309	mg/kg
Highly Chlorinated PCBs	HB-WSD-29	0	0.5	12/18/2006	0.833	mg/kg
Highly Chlorinated PCBs	HB-WSD-29	0.5	1	12/18/2006	0.0255	mg/kg
Highly Chlorinated PCBs	HB-WSD-30	0	0.5	12/18/2006	0.294	mg/kg
Highly Chlorinated PCBs	HB-WSD-30	0.5	1	12/18/2006	0.0227	mg/kg
Less Chlorinated PCBs	HB-SB-57	6	8	12/27/2006	0.0288	mg/kg
Total PCBs	HB-GWS-01	6	8	12/18/2006	0.02008	mg/kg
Total PCBs	HB-GWS-04	2	4	12/20/2006	2.343	mg/kg

TABLE 2.38c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-GWS-05	4	6	12/11/2006	2.64	mg/kg
Total PCBs	HB-GWS-08	8	10	12/13/2006	0.0183	mg/kg
Total PCBs	HB-MW-22	4	6	12/26/2006	0.0947	mg/kg
Total PCBs	HB-MW-24	8	10	12/20/2006	0.273	mg/kg
Total PCBs	HB-MW-25	4	6	12/21/2006	0.0958	mg/kg
Total PCBs	HB-SB-57	6	8	12/27/2006	0.0435	mg/kg
Total PCBs	HB-WSD-01	0	0.5	12/14/2006	0.783	mg/kg
Total PCBs	HB-WSD-01	0.5	1	12/14/2006	0.653	mg/kg
Total PCBs	HB-WSD-01	1	2	12/14/2006	2.241	mg/kg
Total PCBs	HB-WSD-02	0	0.5	12/14/2006	0.2238	mg/kg
Total PCBs	HB-WSD-02	0.5	1	12/14/2006	1.057	mg/kg
Total PCBs	HB-WSD-02	1	2	12/14/2006	1.554	mg/kg
Total PCBs	HB-WSD-04	1	2	12/14/2006	0.0894	mg/kg
Total PCBs	HB-WSD-05	0	0.5	12/14/2006	0.1393	mg/kg
Total PCBs	HB-WSD-05	0.5	1	12/14/2006	0.1834	mg/kg
Total PCBs	HB-WSD-05	1	2	12/14/2006	0.0859	mg/kg
Total PCBs	HB-WSD-06	0	0.5	12/13/2006	0.2169	mg/kg
Total PCBs	HB-WSD-06	0.5	1	12/13/2006	0.863	mg/kg
Total PCBs	HB-WSD-06	1	2	12/13/2006	0.1095	mg/kg
Total PCBs	HB-WSD-07	0	0.5	12/14/2006	0.335	mg/kg
Total PCBs	HB-WSD-07	0.5	1	12/14/2006	1.032	mg/kg
Total PCBs	HB-WSD-07	1	2	12/14/2006	0.2101	mg/kg
Total PCBs	HB-WSD-08	0	0.5	12/13/2006	1.03	mg/kg
Total PCBs	HB-WSD-08	0.5	1	12/13/2006	0.532	mg/kg
Total PCBs	HB-WSD-08	1	2	12/13/2006	0.226	mg/kg
Total PCBs	HB-WSD-09	0	0.5	12/13/2006	0.708	mg/kg
Total PCBs	HB-WSD-09	0.5	1	12/13/2006	0.534	mg/kg
Total PCBs	HB-WSD-09	1	2	12/13/2006	0.6	mg/kg
Total PCBs	HB-WSD-10	0	0.5	12/13/2006	0.2337	mg/kg
Total PCBs	HB-WSD-10	0.5	1	12/13/2006	0.414	mg/kg
Total PCBs	HB-WSD-10	1	2	12/13/2006	0.495	mg/kg
Total PCBs	HB-WSD-11	0	0.5	12/13/2006	1.025	mg/kg
Total PCBs	HB-WSD-11	0.5	1	12/13/2006	0.0748	mg/kg
Total PCBs	HB-WSD-11	1	2	12/13/2006	0.391	mg/kg
Total PCBs	HB-WSD-12	0	0.5	12/13/2006	0.0653	mg/kg
Total PCBs	HB-WSD-12	0.5	1	12/13/2006	0.758	mg/kg
Total PCBs	HB-WSD-12	1	2	12/13/2006	0.527	mg/kg
Total PCBs	HB-WSD-13	0	0.5	12/13/2006	0.533	mg/kg
Total PCBs	HB-WSD-13	0.5	1	12/13/2006	0.618	mg/kg
Total PCBs	HB-WSD-13	1	2	12/13/2006	0.455	mg/kg
Total PCBs	HB-WSD-14	0	0.5	12/12/2006	0.596	mg/kg
Total PCBs	HB-WSD-14	0.5	1	12/12/2006	1.232	mg/kg
Total PCBs	HB-WSD-14	1	2	12/12/2006	1.77	mg/kg

TABLE 2.38c
DERIVATION OF PCB EQUIVALENTS FOR CHLORINATED CHEMICALS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL

Chlorination Level*	Sample Location	Start Depth (ft)	End Depth (ft)	Sample Date	Sum of Location PCB Concentration	Units
Total PCBs	HB-WSD-15	0	0.5	12/12/2006	0.698	mg/kg
Total PCBs	HB-WSD-15	0.5	1	12/12/2006	1.323	mg/kg
Total PCBs	HB-WSD-15	1	2	12/12/2006	1.483	mg/kg
Total PCBs	HB-WSD-16	0	0.5	12/11/2006	3.47	mg/kg
Total PCBs	HB-WSD-16	0.5	1	12/11/2006	2.218	mg/kg
Total PCBs	HB-WSD-16	1	2	12/11/2006	1.32	mg/kg
Total PCBs	HB-WSD-17	0	0.5	12/12/2006	2.22	mg/kg
Total PCBs	HB-WSD-17	0.5	1	12/12/2006	1.808	mg/kg
Total PCBs	HB-WSD-17	1	2	12/12/2006	0.947	mg/kg
Total PCBs	HB-WSD-18	0	0.5	12/12/2006	2.859	mg/kg
Total PCBs	HB-WSD-18	0.5	1	12/12/2006	1.482	mg/kg
Total PCBs	HB-WSD-18	1	2	12/12/2006	0.594	mg/kg
Total PCBs	HB-WSD-20	0	0.5	12/12/2006	0.0939	mg/kg
Total PCBs	HB-WSD-20	0.5	1	12/12/2006	0.1179	mg/kg
Total PCBs	HB-WSD-20	1	2	12/12/2006	0.117	mg/kg
Total PCBs	HB-WSD-22	0	0.5	12/11/2006	0.0311	mg/kg
Total PCBs	HB-WSD-22	0.5	1	12/11/2006	0.0321	mg/kg
Total PCBs	HB-WSD-23	0	0.5	12/11/2006	1.683	mg/kg
Total PCBs	HB-WSD-23	0.5	1	12/11/2006	1.635	mg/kg
Total PCBs	HB-WSD-23	1	2	12/11/2006	1.012	mg/kg
Total PCBs	HB-WSD-24	0	0.5	12/11/2006	2.48	mg/kg
Total PCBs	HB-WSD-24	0.5	1	12/11/2006	0.45	mg/kg
Total PCBs	HB-WSD-24	1	2	12/11/2006	1.515	mg/kg
Total PCBs	HB-WSD-25	0	0.5	12/12/2006	0.1494	mg/kg
Total PCBs	HB-WSD-25	0.5	1	12/12/2006	0.111	mg/kg
Total PCBs	HB-WSD-25	1	2	12/12/2006	0.1882	mg/kg
Total PCBs	HB-WSD-26	0	0.5	12/18/2006	0.655	mg/kg
Total PCBs	HB-WSD-26	0.5	1	12/18/2006	0.349	mg/kg
Total PCBs	HB-WSD-26	1	2	12/18/2006	0.0531	mg/kg
Total PCBs	HB-WSD-27	0	0.5	12/11/2006	0.0374	mg/kg
Total PCBs	HB-WSD-27	0.5	1	12/11/2006	0.0684	mg/kg
Total PCBs	HB-WSD-27	1	2	12/11/2006	0.0309	mg/kg
Total PCBs	HB-WSD-29	0	0.5	12/18/2006	0.833	mg/kg
Total PCBs	HB-WSD-29	0.5	1	12/18/2006	0.0255	mg/kg
Total PCBs	HB-WSD-30	0	0.5	12/18/2006	0.294	mg/kg
Total PCBs	HB-WSD-30	0.5	1	12/18/2006	0.0227	mg/kg

Notes:

* Less Chlorinated PCBs were defined as Aroclors 1221, 1232, 1016, and 1242. Highly Chlorinated PCBs were defined as Aroclors 1248, 1254, 1260, and higher if reported. Total PCBs are the sum of all detected Aroclors.

TABLE 2.38d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-GWS-01	12/18/2006	6	8	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.011
HB-GWS-01	12/18/2006	6	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.011
Total Chlordane =									ND
HB-GWS-03	12/19/2006	6	8	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.021
HB-GWS-03	12/19/2006	6	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.021
Total Chlordane =									ND
HB-GWS-04	12/20/2006	2	4	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.1
HB-GWS-04	12/20/2006	2	4	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.1
Total Chlordane =									ND
HB-GWS-05	12/11/2006	4	6	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.14
HB-GWS-05	12/11/2006	4	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.14
Total Chlordane =									ND
HB-GWS-07	12/14/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.025
HB-GWS-07	12/14/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.025
Total Chlordane =									ND
HB-GWS-08	12/13/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.062
HB-GWS-08	12/13/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.062
Total Chlordane =									ND
HB-MW-22	12/26/2006	4	6	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.043
HB-MW-22	12/26/2006	4	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.043
Total Chlordane =									ND
HB-MW-23	12/26/2006	8	10	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0007
HB-MW-23	12/26/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0024
Total Chlordane =									0.0007
HB-MW-24	12/20/2006	8	10	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0041
HB-MW-24	12/20/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.014
Total Chlordane =									0.0041
HB-MW-25	12/21/2006	4	6	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.053
HB-MW-25	12/21/2006	4	6	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.053
Total Chlordane =									ND
HB-MW-26	12/21/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0042
HB-MW-26	12/21/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0042
Total Chlordane =									ND
HB-SB-52	12/14/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-SB-52	12/14/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-SB-54	12/12/2006	8	10	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.028
HB-SB-54	12/12/2006	8	10	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.028
Total Chlordane =									ND
HB-SB-56	12/11/2006	6	8	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.014
HB-SB-56	12/11/2006	6	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.014
Total Chlordane =									ND
HB-SB-57	12/27/2006	6	8	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0029
HB-SB-57	12/27/2006	6	8	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0029
Total Chlordane =									ND

TABLE 2.38d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-01	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.021
HB-WSD-01	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.016
Total Chlordane =									0.016
HB-WSD-01	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.021
HB-WSD-01	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.016
Total Chlordane =									0.016
HB-WSD-01	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.05
HB-WSD-01	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.05
Total Chlordane =									0.05
HB-WSD-02	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0076
HB-WSD-02	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.019
Total Chlordane =									0.0076
HB-WSD-02	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.039
HB-WSD-02	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.029
Total Chlordane =									0.029
HB-WSD-02	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.061
HB-WSD-02	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.046
Total Chlordane =									0.046
HB-WSD-03	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-03	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.0055
HB-WSD-03	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0082
Total Chlordane =									0.0082
HB-WSD-03	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.002
HB-WSD-03	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0012
Total Chlordane =									0.0012
HB-WSD-04	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0023
HB-WSD-04	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0016
Total Chlordane =									0.0016
HB-WSD-04	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.012
HB-WSD-04	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.014
Total Chlordane =									0.014
HB-WSD-04	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.063
HB-WSD-04	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.032
Total Chlordane =									0.032
HB-WSD-05	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0025
HB-WSD-05	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0025
Total Chlordane =									ND
HB-WSD-05	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0022
HB-WSD-05	12/14/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.011
Total Chlordane =									0.011
HB-WSD-05	12/14/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0021
HB-WSD-05	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0021
Total Chlordane =									ND
HB-WSD-06	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0044
HB-WSD-06	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0015
Total Chlordane =									0.0015

TABLE 2.38d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-06	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-06	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0023
HB-WSD-06	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0019
Total Chlordane =									0.0019
HB-WSD-07	12/14/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0049
HB-WSD-07	12/14/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0049
Total Chlordane =									ND
HB-WSD-07	12/14/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.011
Total Chlordane =									0.011
HB-WSD-07	12/14/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.006
Total Chlordane =									0.006
HB-WSD-08	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.011
HB-WSD-08	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.011
Total Chlordane =									ND
HB-WSD-08	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0046
HB-WSD-08	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0064
Total Chlordane =									0.0064
HB-WSD-08	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0047
HB-WSD-08	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0029
Total Chlordane =									0.0029
HB-WSD-09	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0097
HB-WSD-09	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0081
Total Chlordane =									0.0081
HB-WSD-09	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.009
HB-WSD-09	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0068
Total Chlordane =									0.0068
HB-WSD-09	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0046
Total Chlordane =									ND
HB-WSD-10	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0025
HB-WSD-10	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0025
Total Chlordane =									ND
HB-WSD-10	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0024
HB-WSD-10	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0024
Total Chlordane =									ND
HB-WSD-10	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0062
HB-WSD-10	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0052
Total Chlordane =									0.0052
HB-WSD-11	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0029
HB-WSD-11	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0029
Total Chlordane =									ND
HB-WSD-11	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0021
HB-WSD-11	12/13/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0021
Total Chlordane =									ND

TABLE 2.38d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-11	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-11	12/13/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-12	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-12	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-12	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-12	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0059
Total Chlordane =									0.0059
HB-WSD-13	12/13/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0024
HB-WSD-13	12/13/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0024
Total Chlordane =									ND
HB-WSD-13	12/13/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0081
Total Chlordane =									0.0081
HB-WSD-13	12/13/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0038
Total Chlordane =									0.0038
HB-WSD-14	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.012
HB-WSD-14	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-14	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.023
HB-WSD-14	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.023
Total Chlordane =									ND
HB-WSD-14	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.024
HB-WSD-14	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.024
Total Chlordane =									ND
HB-WSD-15	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.012
HB-WSD-15	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-15	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.025
HB-WSD-15	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.025
Total Chlordane =									ND
HB-WSD-15	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.026
HB-WSD-15	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.026
Total Chlordane =									ND
HB-WSD-16	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.063
HB-WSD-16	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.058
Total Chlordane =									0.063
HB-WSD-16	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.063
Total Chlordane =									0.063
HB-WSD-16	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.021
HB-WSD-16	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.021
Total Chlordane =									0.021
HB-WSD-17	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-17	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-WSD-17	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-17	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND

TABLE 2.38d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-17	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-17	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-WSD-18	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.075
Total Chlordane =									ND
HB-WSD-18	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.029
HB-WSD-18	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									ND
HB-WSD-18	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.013
HB-WSD-18	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.013
Total Chlordane =									ND
HB-WSD-19	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0042
HB-WSD-19	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0042
Total Chlordane =									ND
HB-WSD-19	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.002
HB-WSD-19	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.002
Total Chlordane =									ND
HB-WSD-20	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.013
HB-WSD-20	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.013
Total Chlordane =									ND
HB-WSD-20	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.026
HB-WSD-20	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.026
Total Chlordane =									ND
HB-WSD-20	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.032
HB-WSD-20	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.032
Total Chlordane =									ND
HB-WSD-21	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0019
HB-WSD-21	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0019
Total Chlordane =									ND
HB-WSD-21	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0018
HB-WSD-21	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0018
Total Chlordane =									ND
HB-WSD-21	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-21	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-22	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.048
HB-WSD-22	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.048
Total Chlordane =									ND
HB-WSD-22	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.023
HB-WSD-22	12/11/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.023
Total Chlordane =									ND
HB-WSD-23	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.06
HB-WSD-23	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.047
Total Chlordane =									0.047
HB-WSD-23	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y		mg/kg	0.03
Total Chlordane =									0.03

TABLE 2.38d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-23	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.022
HB-WSD-23	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.029
Total Chlordane =									0.022
HB-WSD-24	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.044
HB-WSD-24	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y		mg/kg	0.056
Total Chlordane =									0.056
HB-WSD-24	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.016
HB-WSD-24	12/11/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.017
Total Chlordane =									0.017
HB-WSD-24	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.034
HB-WSD-24	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.047
Total Chlordane =									0.047
HB-WSD-25	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.012
HB-WSD-25	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.012
Total Chlordane =									ND
HB-WSD-25	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.005
HB-WSD-25	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0033
Total Chlordane =									0.0033
HB-WSD-25	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	Y	J	mg/kg	0.0048
HB-WSD-25	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	Y	J	mg/kg	0.0024
Total Chlordane =									0.0024
HB-WSD-26	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.28
HB-WSD-26	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.28
Total Chlordane =									ND
HB-WSD-26	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.067
HB-WSD-26	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.067
Total Chlordane =									ND
HB-WSD-26	12/18/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.038
HB-WSD-26	12/18/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.038
Total Chlordane =									ND
HB-WSD-27	12/11/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.13
HB-WSD-27	12/11/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.13
Total Chlordane =									ND
HB-WSD-27	12/11/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.051
HB-WSD-27	12/11/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.051
Total Chlordane =									ND
HB-WSD-27	12/11/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.046
HB-WSD-27	12/11/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.046
Total Chlordane =									ND
HB-WSD-28	12/12/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0022
HB-WSD-28	12/12/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0022
Total Chlordane =									ND
HB-WSD-28	12/12/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-28	12/12/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND

TABLE 2.38d
DERIVATION OF TOTAL CHLORDANE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value
HB-WSD-28	12/12/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.0023
HB-WSD-28	12/12/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.0023
Total Chlordane =									ND
HB-WSD-29	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.035
HB-WSD-29	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.035
Total Chlordane =									ND
HB-WSD-29	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0031
HB-WSD-29	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0031
Total Chlordane =									ND
HB-WSD-29	12/18/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0031
HB-WSD-29	12/18/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0031
Total Chlordane =									ND
HB-WSD-30	12/18/2006	0	0.5	5103-71-9	ALPHA-CHLORDANE	N	UJ	mg/kg	0.022
HB-WSD-30	12/18/2006	0	0.5	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	UJ	mg/kg	0.022
Total Chlordane =									ND
HB-WSD-30	12/18/2006	0.5	1	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0064
HB-WSD-30	12/18/2006	0.5	1	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0064
Total Chlordane =									ND
HB-WSD-30	12/18/2006	1	2	5103-71-9	ALPHA-CHLORDANE	N	U	mg/kg	0.0032
HB-WSD-30	12/18/2006	1	2	12789-03-6	CONSTITUENTS OF CHLORDANE (ALPHA, BETA, AND GAMMA)	N	U	mg/kg	0.0032
Total Chlordane =									ND

TABLE 2.38e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-GWS-01	12/18/2006	6	8	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.052	0.052
HB-GWS-03	12/19/2006	6	8	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.0068	0.0068
HB-GWS-04	12/20/2006	2	4	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.06	0.03
HB-GWS-05	12/11/2006	4	6	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.009	0.009
HB-GWS-07	12/14/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.73	0.365
HB-GWS-08	12/13/2006	8	10	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.028	0.028
HB-GWS-08	12/13/2006	8	10	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.91	0.455
HB-MW-22	12/26/2006	4	6	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0085	0.0085
HB-MW-23	12/26/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.007	0.0035
HB-MW-24	12/20/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0081	0.00405
HB-MW-25	12/21/2006	4	6	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.024	0.024
HB-MW-26	12/21/2006	8	10	1330-20-7	XYLENES, TOTAL	N	J	mg/kg	0.074	0.037
HB-SB-52	12/14/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	3.4	1.7
HB-SB-54	12/12/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.82	0.41
HB-SB-56	12/11/2006	6	8	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0085	0.00425
HB-SB-57	12/27/2006	6	8	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00096	0.00096
HB-WSD-01	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-01	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.007	0.0035
HB-WSD-01	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0058	0.0029
HB-WSD-02	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0057	0.00285
HB-WSD-02	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-02	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0071	0.00355
HB-WSD-03	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-03	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-03	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0058	0.0029
HB-WSD-04	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-04	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0073	0.00365
HB-WSD-04	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-05	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-05	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0066	0.0033
HB-WSD-05	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0062	0.0031
HB-WSD-06	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0065	0.00325
HB-WSD-06	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0012	0.0012
HB-WSD-06	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-07	12/14/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-07	12/14/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-07	12/14/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-08	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-08	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-08	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-09	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-09	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-09	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-10	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0073	0.00365
HB-WSD-10	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y		mg/kg	0.0073	0.0073
HB-WSD-10	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0015	0.0015
HB-WSD-11	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0085	0.00425
HB-WSD-11	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0062	0.0031
HB-WSD-11	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00096	0.00096
HB-WSD-12	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0069	0.00345
HB-WSD-12	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-12	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.007	0.0035
HB-WSD-13	12/13/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-13	12/13/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034

TABLE 2.38e
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SUBSURFACE SOIL (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-WSD-13	12/13/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00077	0.00077
HB-WSD-14	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0016	0.0016
HB-WSD-14	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-14	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-15	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0072	0.0036
HB-WSD-15	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0075	0.00375
HB-WSD-15	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0077	0.00385
HB-WSD-16	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0086	0.0043
HB-WSD-16	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0011	0.0011
HB-WSD-16	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00086	0.00086
HB-WSD-17	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0014	0.0014
HB-WSD-17	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0058	0.0058
HB-WSD-17	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00089	0.00089
HB-WSD-18	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0013	0.0013
HB-WSD-18	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.001	0.001
HB-WSD-18	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0012	0.0012
HB-WSD-19	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0062	0.0031
HB-WSD-19	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0059	0.00295
HB-WSD-20	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00082	0.00082
HB-WSD-20	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00092	0.00092
HB-WSD-20	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0094	0.0047
HB-WSD-21	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00079	0.00079
HB-WSD-21	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00088	0.00088
HB-WSD-21	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0016	0.0016
HB-WSD-22	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0013	0.0013
HB-WSD-22	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00088	0.00088
HB-WSD-23	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.00089	0.00089
HB-WSD-23	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0069	0.0069
HB-WSD-23	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0044	0.0044
HB-WSD-24	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0081	0.00405
HB-WSD-24	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0087	0.00435
HB-WSD-24	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.0013	0.0013
HB-WSD-25	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0073	0.00365
HB-WSD-25	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.001	0.001
HB-WSD-25	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	Y	J	mg/kg	0.002	0.002
HB-WSD-26	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.017	0.0085
HB-WSD-26	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.0098	0.0049
HB-WSD-26	12/18/2006	1	2	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.011	0.0055
HB-WSD-27	12/11/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0075	0.00375
HB-WSD-27	12/11/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.0074	0.0037
HB-WSD-27	12/11/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0067	0.00335
HB-WSD-28	12/12/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0065	0.00325
HB-WSD-28	12/12/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0066	0.0033
HB-WSD-28	12/12/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0068	0.0034
HB-WSD-29	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.01	0.005
HB-WSD-29	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0092	0.0046
HB-WSD-29	12/18/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0091	0.00455
HB-WSD-30	12/18/2006	0	0.5	1330-20-7	XYLENES, TOTAL	N	UJ	mg/kg	0.013	0.0065
HB-WSD-30	12/18/2006	0.5	1	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0093	0.00465
HB-WSD-30	12/18/2006	1	2	1330-20-7	XYLENES, TOTAL	N	U	mg/kg	0.0095	0.00475

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.39a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)			
SYW-12 Area - Shallow Ground Water	METALS																		
	7429-90-5	ALUMINUM	0.042 J	57	mg/L	HB-GWS-09	17/18	0.1-0.1	5.70E+01		2.00E-01	3.65E+00	N	3.65E+00	nc	3.65E+00	Y	ASL	
	7440-36-0	ANTIMONY	0.0018 J	0.0058 J	mg/L	HB-GWS-08	2/18	0.06-0.06	5.80E-03		6.00E-03	1.46E-03	N	1.46E-03	nc	1.46E-03	Y	ASL	
	7440-38-2	ARSENIC	0.0048 J	0.059	mg/L	HB-GWS-08	7/18	0.01-0.01	5.90E-02		1.00E-02	4.46E-05	C	4.46E-05	ca	4.46E-05	Y	TOX	
	7440-39-3	BARIUM	0.1	1.7	mg/L	HB-GWS-09	18/18	-	1.70E+00		2.00E+00	7.30E-01	N	2.55E-01	nc	2.55E-01	Y	ASL	
	7440-41-7	BERYLLIUM	0.0011 J	0.0036 J	mg/L	HB-GWS-09	3/18	0.01-0.01	3.60E-03		4.00E-03	7.30E-03	N	7.30E-03	nc	7.30E-03	N	BSL	
	7440-43-9	CADMIUM	0.0024 J	0.027	mg/L	HB-GWS-08	7/18	0.01-0.01	2.70E-02		5.00E-03	1.83E-03	N	1.82E-03	nc	1.82E-03	Y	ASL	
	7440-70-2	CALCIUM	220	3600	mg/L	HB-GWS-09	18/18	-	3.60E+03			NV	NV	NV	NV	N	NUT		
	7440-47-3	CHROMIUM ^a	0.0022 J	0.33	mg/L	HB-GWS-08	13/18	0.01-0.01	3.30E-01		1.00E-01	1.10E-02	N	1.09E-02	nc	1.09E-02	Y	TOX	
	7440-48-4	COBALT	0.0071 J	0.05 J	mg/L	HB-GWS-09	4/18	0.05-0.05	5.00E-02			NV	7.30E-02	nc	7.30E-02	N	BSL		
	7440-50-8	COPPER	0.0023 J	0.74	mg/L	HB-GWS-09	14/18	0.01-0.01	7.40E-01		1.30E+00	1.46E-01	N	1.46E-01	nc	1.46E-01	Y	ASL	
	57-12-5	CYANIDE	0.016	0.028	mg/L	HB-GWS-04	2/18	0.01-0.01	2.80E-02		2.00E-01	7.30E-02	N	7.30E-02	nc	7.30E-02	N	BSL	
	7439-89-6	IRON	3.8	120	mg/L	HB-GWS-08	18/18	-	1.20E+02		3.00E-01	2.56E+00	N	1.09E+00	nc	1.09E+00	Y	ASL	
	7439-92-1	LEAD	0.005 J	1.7	mg/L	HB-GWS-09	13/18	0.01-0.01	1.70E+00		1.50E-02	NV	NV	1.50E-02	Y	ASL			
	7439-95-4	MAGNESIUM	9.9	110	mg/L	HB-MW-23	18/18	-	1.10E+02			NV	NV	NV	NV	N	NUT		
	7439-96-5	MANGANESE	0.27	3.3	mg/L	HB-GWS-09	18/18	-	3.30E+00		5.00E-02	7.30E-02	N	8.76E-02	nc	7.30E-02	Y	ASL	
	7439-97-6	MERCURY ^b	0.00003 J	0.0087	mg/L	HB-GWS-08	11/18	0.0002-0.0002	8.70E-03		2.00E-03	3.65E-04	N	3.65E-04	nc	3.65E-04	Y	ASL	
	7440-02-0	NICKEL	0.0016 J	0.2	mg/L	HB-GWS-08	18/18	-	2.00E-01			7.30E-02	N	7.30E-02	nc	7.30E-02	Y	ASL	
	7440-09-7	POTASSIUM	2 J	69 J	mg/L	HB-MW-24	18/18	-	6.90E+01			NV	NV	NV	NV	N	NUT		
	7782-49-2	SELENIUM	0.0052 J	0.022	mg/L	HB-GWS-09	4/18	0.01-0.01	2.20E-02		5.00E-02	1.83E-02	N	1.82E-02	nc	1.82E-02	Y	ASL	
	7440-22-4	SILVER	0.0011 J	0.011	mg/L	HB-GWS-08, HB-GWS-09	6/18	0.01-0.01	1.10E-02		1.00E-01	1.83E-02	N	1.82E-02	nc	1.82E-02	N	BSL	
	7440-23-5	SODIUM	150	3400 J	mg/L	HB-MW-23	18/18	-	3.40E+03			NV	NV	NV	NV	N	NUT		
	7440-28-0	THALLIUM	0.023	0.023	mg/L	HB-GWS-09	1/18	0.02-0.02	2.30E-02		2.00E-03	2.56E-04	N	2.41E-04	nc	2.41E-04	Y	ASL	
	7440-62-2	VANADIUM	0.0014 J	0.14	mg/L	HB-GWS-09	13/18	0.05-0.05	1.40E-01			3.65E-03	N	3.65E-03	nc	3.65E-03	Y	ASL	
	7440-66-6	ZINC	0.014 J	1.9	mg/L	HB-GWS-08	12/18	0.02-0.02	1.90E+00		5.00E+00	1.10E+00	N	1.09E+00	nc	1.09E+00	Y	ASL	
	PESTICIDES																		
	50-29-3	4,4'-DDT		0.018 J	0.018 J	ug/l	HB-B-04W	1/17	0.1-1.1	1.80E-02			1.97E-01	C	1.98E-01	ca	1.97E-01	N	BSL
	SVOCs																		
	92-52-4	1,1'-BIPHENYL	1.2 J	8.9 J	ug/L	HB-B-04W	2/18	10-11	8.90E+00			3.04E+01	N	3.04E+01	nc	3.04E+01	N	BSL	
	91-57-6	2-METHYLNAPHTHALENE	1.1 J	5.5 J	ug/l	HB-GWS-08	5/18	10-11	5.50E+00			1.83E+01	N	NV	1.83E+01	N	BSL		
	106-44-5	4-METHYLPHENOL	2 J	4.1 J	ug/l	HB-GWS-01	3/18	10-11	4.10E+00			1.83E+01	N	1.82E+01	nc	1.82E+01	N	BSL	
	100-02-7	4-NITROPHENOL	1.1 J	1.1 J	ug/l	HB-MW-25	1/18	50-55	1.10E+00			NV	NV	NV	NV	Y	NTX		
	83-32-9	ACENAPHTHENE	1.4 J	41	ug/l	HB-B-04W	7/18	10-11	4.10E+01			3.65E+01	N	3.65E+01	nc	3.65E+01	Y	ASL	
	208-96-8	ACENAPHTHYLENE	1.4 J	17	ug/l	HB-GWS-08	6/18	10-11	1.70E+01			NV	NV	NV	NV	Y	NTX		
	120-12-7	ANTHRACENE	1.2 J	7.9 J	ug/l	HB-GWS-08	6/18	10-11	7.90E+00		3.00E+00	3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL	
	1912-24-9	ATRAZINE	53	53	ug/L	HB-GWS-05	1/18	10-11	5.30E+01			3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL	
	100-52-7	BENZALDEHYDE	3.9 J	37	ug/L	HB-GWS-05	2/18	10-11	3.70E+01			3.65E+02	N	3.65E+02	nc	3.65E+02	N	BSL	
	56-55-3	BENZ(A)ANTHRACENE	1.4 J	13	ug/l	HB-GWS-08	6/18	10-11	1.30E+01			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL	
	50-32-8	BENZO(A)PYRENE	1.4 J	18	ug/l	HB-GWS-08	7/18	10-11	1.80E+01		2.00E-01	3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL	
	205-99-2	BENZO(B)FLUORANTHENE	1.6 J	20	ug/l	HB-GWS-08	7/18	10-11	2.00E+01			3.00E-01	C	9.21E-01	ca	3.00E-01	Y	ASL	
	191-24-2	BENZO(G,H,I)PERYLENE	1.5 J	7.3 J	ug/l	HB-GWS-08	5/18	10-11	7.30E+00			NV	NV	NV	NV	Y	NTX		
	207-08-9	BENZO(K)FLUORANTHENE	1.1 J	6.9 J	ug/l	HB-GWS-08	4/18	10-11	6.90E+00			3.35E+00	C	3.36E+00	ca	3.35E+00	Y	ASL	
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1.1 J	5.9 J	ug/l	HB-GWS-05	3/18	10-17	5.90E+00		6.00E+00	3.00E+00	C	9.21E+00	ca	3.00E+00	Y	ASL	
	105-60-2	CAPROLACTAM	42	42	ug/L	HB-GWS-05	1/18	10-11	4.20E+01			3.04E-01	C	3.03E-01	ca	3.03E-01	Y	ASL	
	86-74-8	CARBAZOLE	1.2 J	3.5 J	ug/l	HB-B-04W	2/18	10-11	3.50E+00			3.00E-03	C	9.21E-03	ca	3.00E-03	Y	ASL	
	218-01-9	CHRYSENE	1.4 J	14	ug/l	HB-GWS-08	7/18	10-11	1.40E+01			3.65E+00	N	1.22E+00	nc	1.22E+00	Y	ASL	
	53-70-3	DIBENZ(A,H)ANTHRACENE	2.1 J	2.1 J	ug/l	HB-GWS-08	1/18	10-11	2.10E+00			1.46E+02	N	1.46E+02	nc	1.46E+02	N	BSL	
	132-64-9	DIBENZOFURAN	1.2 J	2.2 J	ug/l	HB-B-04W	2/18	10-11	2.20E+00			3.04E+01	N	3.04E+01	nc	3.04E+01	N	BSL	
	206-44-0	FLUORANTHENE	1.7 J	16	ug/l	HB-GWS-08	8/18	10-11	1.60E+01			2.43E+01	N	2.43E+01	nc	2.43E+01	N	BSL	
	86-73-7	FLUORENE	1.3 J	12	ug/l	HB-B-04W	5/18	10-11	1.20E+01			3.00E-02	C	9.21E-02	ca	3.00E-02	Y	ASL	
	193-39-5	INDENO(1,2,3-CD)PYRENE	1.2 J	5 J	ug/l	HB-GWS-08	5/18	10-11	5.00E+00			1.83E+03	N	1.82E+03	nc	1.82E+03	N	BSL	
	91-20-3	NAPHTHALENE	1.2 J	170	ug/l	HB-B-04W	9/18	10-11	1.70E+02			6.51E-01	N	6.20E-01	nc	6.20E-01	Y	ASL	
	85-01-8	PHENANTHRENE	1.5 J	17	ug/l	HB-B-04W	8/18	10-11	1.70E+01			NV	NV	NV	NV	Y	NTX		
	108-95-2	PHENOL	2.6 J	20	ug/l	HB-GWS-01	2/18	10-11	2.00E+01			1.10E+03	N	1.09E+03	nc	1.09E+03	N	BSL	
	129-00-0	PYRENE	1 J	22	ug/l	HB-GWS-08	9/18	10-11	2.20E+01			1.83E+01	N	1.83E+01	nc	1.83E+01	Y	ASL	

TABLE 2.39a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SHALLOW GROUND WATER
GEDDES AND SYRACUSE, NEW YORK

Scenario: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	USEPA RBC for Tap Water (5)	USEPA PRG for Tap Water (6)	Screening Toxicity Value (7)	COPC Flag (Y/N)	Rationale for Selection or Deletion (8)
	VOCs															
	75-34-3	1,1-DICHLOROETHANE	0.1 J	1.04	ug/l	HB-GWS-01	2/18	0.5-12.5	1.04E+00			8.96E+01	N	8.11E+01	nc	8.11E+01 N BSL
	95-50-1	1,2-DICHLOROBENZENE	0.12 J	0.21 J	ug/l	HB-MW-26	4/18	0.5-12.5	2.10E-01		6.00E+02	2.68E+01	N	3.70E+01	nc	2.68E+01 N BSL
	106-46-7	1,4-DICHLOROBENZENE	0.1 J	0.34 J	ug/l	HB-MW-25	3/18	0.5-12.5	3.40E-01		7.50E+01	2.81E-01	C	5.02E-01	ca	2.81E-01 Y ASL
	78-93-3	2-BUTANONE	1.38 J	4.1 J	ug/l	HB-GWS-09	2/18	10-250	4.10E+00			6.97E+02	N	6.97E+02	nc	6.97E+02 N BSL
	67-64-1	ACETONE	1.31 J	1.57 J	ug/l	HB-MW-24	3/18	10-250	1.57E+00			5.48E+02	N	5.48E+02	nc	5.48E+02 N BSL
	98-86-2	ACETOPHENONE	4.2 J	38	ug/L	HB-GWS-05	2/18	10-11	3.80E+01			6.08E+01	N	NV		6.08E+01 N BSL
	71-43-2	BENZENE	0.9	0.9	ug/l	HB-GWS-01	1/18	0.5-12.5	9.00E-01		5.00E+00	1.24E+03	N	1.03E+03	nc	1.03E+03 Y TOX
	75-15-0	CARBON DISULFIDE	0.11 J	0.63	ug/l	HB-GWS-09	8/18	0.5-12.5	6.30E-01			3.36E-01	C	3.54E-01	ca	3.36E-01 Y ASL
	108-90-7	CHLOROBENZENE	0.17 J	0.39 J	ug/l	HB-GWS-04	5/18	0.5-12.5	3.90E-01		1.00E+02	1.04E+02	N	1.04E+02	nc	1.04E+02 N BSL
	156-59-2	CIS-1,2-DICHLOROETHENE	1.12	1.12	ug/l	HB-GWS-01	1/18	0.5-12.5	1.12E+00		7.00E+01	8.96E+00	N	1.06E+01	nc	8.96E+00 N BSL
	110-82-7	CYCLOHEXANE	0.55	0.55	ug/l	HB-MW-22	1/18	0.5-12.5	5.50E-01			6.08E+00	N	6.08E+00	nc	6.08E+00 N BSL
	100-41-4	ETHYLBENZENE	0.27 J	14.8	ug/l	HB-B-04W	3/18	0.5-0.5	1.48E+01		7.00E+02	1.34E+02	N	1.34E+02	nc	1.34E+02 N BSL
	98-82-8	ISOPROPYLBENZENE	0.1 J	5.25 J	ug/L	HB-B-04W	4/18	0.5-0.5	5.25E+00			6.58E+01	N	6.58E+01	nc	6.58E+01 N BSL
	1634-04-4	METHYL TERT-BUTYL ETHER	0.16 J	0.35 J	ug/l	HB-MW-22	3/18	0.5-12.5	3.50E-01			2.64E+00	C	1.10E+01	ca	2.64E+00 N BSL
	108-88-3	TOLUENE	0.1 J	10.4	ug/l	HB-GWS-01	7/18	0.5-0.5	1.04E+01		1.00E+03	2.27E+02	N	7.23E+01	nc	7.23E+01 N BSL
	1330-20-7	XYLENES, TOTAL	0.11 J	15.2 J	ug/l	HB-B-04W	6/18	0.5-0.5	1.52E+01		1.00E+04	2.13E+01	N	2.06E+01	nc	2.06E+01 N BSL

Footnotes:

*Sample start depth less than or equal to 10 ft bgs.

(1) J - estimated value; N - tentatively identified at an estimated value

(2) Concentration used for screening is the maximum detected concentration.

(3) N/A - No background screening performed.

(4) United States Environmental Protection Agency. 2008. National Primary and Secondary Drinking Water Regulations.

(5) USEPA Region 3 RBCs (USEPA 2007) for tap water; C = Cancer RBC; N = Noncancer RBC; NV = No value in Region 3 RBC data set. Noncancer RBCs adjusted by multiplying RBC by 0.1.

(6) USEPA Region 9 PRGs (USEPA 2004) for tap water; ca = Cancer PRG; nc = Noncancer PRG; NV = No value in Region 9 PRG data set. Noncancer PRGs adjusted by multiplying PRG by 0.1.

(7) The Screening Toxicity Value represents the minimum of the Region 3 RBC and the Region 9 PRG.

(8) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level

- = Compound detected in 100% of samples.

a = RBC and PRG values for chromium VI utilized.

b = Where mercury is not speciated, RBC and PRG values for methyl mercury utilized.

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements

CAS: Chemical Abstract Service

COPC: Compound of Potential Concern

NUT: Compound is an essential nutrient and not screened in

NV: No Value

PRG: Preliminary Remediation Goals, USEPA, 2004

RBC: Risk Based Concentration; USEPA, October, 2007

TBC: To Be Considered

USEPA: United States Environmental Protection Agency

TABLE 2.39b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL WASTEBED B/HARBOR BROOK SITE - SYW-12 SHALLOW GROUND WATER (0-10 FT BGS)

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-B-04W	3/7/2007	6	11	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	15.2	15.2
HB-B-08W	3/5/2007	6	11	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.58	0.58
HB-B-10	3/7/2007	6	11	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-01	12/18/2006	8	10	1330-20-7	XYLENES, TOTAL	Y		ug/l	1.68	1.68
HB-GWS-02	12/18/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-03	12/19/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-04	12/20/2006	8	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.38	0.38
HB-GWS-05	12/11/2006	10	12	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-06	12/15/2006	8	10	1330-20-7	XYLENES, TOTAL	N	UJ	ug/l	1	0.5
HB-GWS-07	12/14/2006	8	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.11	0.11
HB-GWS-08	12/13/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-09	12/12/2006	10	12	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-22	3/5/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-23	3/5/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-24	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-25	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-26	3/5/2007	5	15	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.45	0.45
HB-MW-27	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5

Notes:

a - Total Xylene value utilized in the risk assessment.

TABLE 2.40a
OCCURRENCE, DISTRIBUTION, AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - SYW-12 SHALLOW GROUND WATER: VAPOR INTRUSION
GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0-10 ft bgs)*

Exposure Point	CAS Number	Chemical	Minimum Detected Concentration (1)	Maximum Detected Concentration (1)	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (2)	Background Value (3)	Potential ARAR/TBC Value (4)	Target Groundwater Concentration Corresponding to Target Indoor Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor = 0.001 and Partitioning Across the Water Table Obeys Henry's Law (5)	Screening Toxicity Value	COPC Flag (Y/N)	Rationale for Selection or Deletion (6)
SYW-12 Area - Shallow Ground Water	SVOCs														
	92-52-4	1,1'-BIPHENYL	1.2 J	8.9 J	ug/l	HB-B-04W	2/18	10-11	8.90E+00				NV	Y	NTX
	91-57-6	2-METHYLNAPHTHALENE	1.1 J	5.5 J	ug/l	HB-GWS-08	5/18	10-11	5.50E+00			3.30E+02	nc	3.30E+02	BSL
	106-44-5	4-METHYLPHENOL	2 J	4.1 J	ug/l	HB-GWS-01	3/18	10-11	4.10E+00				NV	Y	NTX
	100-02-7	4-NITROPHENOL	1.1 J	1.1 J	ug/l	HB-MW-25	1/18	50-55	1.10E+00				NV	Y	NTX
	83-32-9	ACENAPHTHENE	1.4 J	41	ug/l	HB-B-04W	7/18	10-11	4.10E+01			**	nc	**	INC
	208-96-8	ACENAPHTHYLENE	1.4 J	17	ug/l	HB-GWS-08	6/18	10-11	1.70E+01				NV	Y	NTX
	120-12-7	ANTHRACENE	1.2 J	7.9 J	ug/l	HB-GWS-08	6/18	10-11	7.90E+00				NV	Y	NTX
	1912-24-9	ATRAZINE	53	53	ug/l	HB-GWS-05	1/18	10-11	5.30E+01		3.00E+00		NV	Y	NTX
	56-55-3	BENZ(A)ANTHRACENE	1.4 J	13	ug/l	HB-GWS-08	6/18	10-11	1.30E+01				NV	Y	NTX
	50-32-8	BENZO(A)PYRENE	1.4 J	18	ug/l	HB-GWS-08	7/18	10-11	1.80E+01				NV	Y	NTX
	205-99-2	BENZO(B)FLUORANTHENE	1.6 J	20	ug/l	HB-GWS-08	7/18	10-11	2.00E+01		2.00E-01	**	c	**	INC
	191-24-2	BENZO(G,H,I)PERYLENE	1.5 J	7.3 J	ug/l	HB-GWS-08	5/18	10-11	7.30E+00				NV	Y	NTX
	207-08-9	BENZO(K)FLUORANTHENE	1.1 J	6.9 J	ug/l	HB-GWS-08	4/18	10-11	6.90E+00				NV	Y	NTX
	117-81-7	BIS(2-ETHYLHEXYL)PHthalATE	1.1 J	5.9 J	ug/l	HB-GWS-05	3/18	10-17	5.90E+00		6.00E+00		NV	Y	NTX
	105-60-2	CAPROLACTAM	42	42	ug/l	HB-GWS-05	1/18	10-11	4.20E+01				NV	Y	NTX
	86-74-8	CARBAZOLE	1.2 J	3.5 J	ug/l	HB-B-04W	2/18	10-11	3.50E+00				NV	Y	NTX
	218-01-9	CHRYSENE	1.4 J	14	ug/l	HB-GWS-08	7/18	10-11	1.40E+01			**	c	**	INC
	53-70-3	DIBENZ(A,H)ANTHRACENE	2.1 J	2.1 J	ug/l	HB-GWS-08	1/18	10-11	2.10E+00				NV	Y	NTX
	132-64-9	DIBENZOFURAN	1.2 J	2.2 J	ug/l	HB-B-04W	2/18	10-11	2.20E+00			**	nc	**	INC
	206-44-0	FLUORANTHENE	1.7 J	16	ug/l	HB-GWS-08	8/18	10-11	1.60E+01				NV	Y	NTX
	86-73-7	FLUORENE	1.3 J	12	ug/l	HB-B-04W	5/18	10-11	1.20E+01			**	nc	**	INC
	193-39-5	INDENO(1,2,3-CD)PYRENE	1.2 J	5 J	ug/l	HB-GWS-08	5/18	10-11	5.00E+00				NV	Y	NTX
	91-20-3	NAPHTHALENE	1.2 J	170	ug/l	HB-B-04W	9/18	10-11	1.70E+02			1.50E+01	nc	1.50E+01	ASL
	85-01-8	PHENANTHRENE	1.5 J	17	ug/l	HB-B-04W	8/18	10-11	1.70E+01				NV	Y	NTX
	108-95-2	PHENOL	2.6 J	20	ug/l	HB-GWS-01	2/18	10-11	2.00E+01				NV	Y	NTX
	129-00-0	PYRENE	1 J	22	ug/l	HB-GWS-08	9/18	10-11	2.20E+01			**	nc	**	INC
	VOCs														
	75-34-3	1,1-DICHLOROETHANE	0.1 J	1.04	ug/l	HB-GWS-01	2/18	0.5-12.5	1.04E+00			2.20E+02	nc	2.20E+02	BSL
	95-50-1	1,2-DICHLOROENZENE	0.12 J	0.21 J	ug/l	HB-MW-26	4/18	0.5-12.5	2.10E-01		6.00E+02	2.60E+02	nc	2.60E+02	BSL
	106-46-7	1,4-DICHLOROENZENE	0.1 J	0.34 J	ug/l	HB-MW-25	3/18	0.5-12.5	3.40E-01		7.50E+01	8.20E+02	nc	8.20E+02	BSL
	78-93-3	2-BUTANONE	1.38 J	4.1 J	ug/l	HB-GWS-09	2/18	10-250	4.10E+00			4.40E+04	nc	4.40E+04	BSL
	67-64-1	ACETONE	1.31 J	1.57 J	ug/l	HB-MW-24	3/18	10-250	1.57E+00			2.20E+04	nc	2.20E+04	BSL
	98-86-2	ACETOPHENONE	4.2 J	38	ug/l	HB-GWS-05	2/18	10-11	3.80E+01			8.00E+05	nc	8.00E+05	BSL
	100-52-7	BENZALDEHYDE	3.9 J	37	ug/l	HB-GWS-05	2/18	10-11	3.70E+01			3.60E+04	nc	3.60E+04	BSL
	71-43-2	BENZENE	0.9	0.9	ug/l	HB-GWS-01	1/18	0.5-12.5	9.00E-01		5.00E+00	1.37E+01	c	1.37E+01	TOX
	75-15-0	CARBON DISULFIDE	0.11 J	0.63	ug/l	HB-GWS-09	8/18	0.5-12.5	6.30E-01			5.60E+01	nc	5.60E+01	BSL
	108-90-7	CHLOROENZENE	0.17 J	0.39 J	ug/l	HB-GWS-04	5/18	0.5-12.5	3.90E-01		1.00E+02	3.90E+01	nc	3.90E+01	BSL
	156-59-2	CIS-1,2-DICHLOROETHENE	1.12	1.12	ug/l	HB-GWS-01	1/18	0.5-12.5	1.12E+00		7.00E+01		NV	Y	NTX
	110-82-7	CYCLOHEXANE	0.55	0.55	ug/l	HB-MW-22	1/18	0.5-12.5	5.50E-01				NV	Y	NTX
	100-41-4	ETHYLBENZENE	0.27 J	14.8	ug/l	HB-B-04W	3/18	0.5-0.5	1.48E+01		7.00E+02	3.01E+01	c	3.01E+01	BSL
	98-82-8	ISOPROPYLBENZENE	0.1 J	5.25 J	ug/l	HB-B-04W	4/18	0.5-0.5	5.25E+00				NV	Y	NTX
	1634-04-4	METHYL TERT-BUTYL ETHER	0.16 J	0.35 J	ug/l	HB-MW-22	3/18	0.5-12.5	3.50E-01				NV	Y	NTX
	108-88-3	TOLUENE	0.1 J	10.4	ug/l	HB-GWS-01	7/18	0.5-0.5	1.04E+01		1.00E+03	1.50E+02	nc	1.50E+02	BSL
	1330-20-7	XYLENES, TOTAL ^a	0.11 J	15.2 J	ug/l	HB-B-04W	6/18	0.5-0.5	1.52E+01		1.00E+04	2.20E+03	nc	2.20E+03	BSL

Footnotes:

- * Sample start depth less than or equal to 10 ft bgs.
 ** Target soil gas concentration exceeds maximum possible vapor concentration (pathway incomplete)
 (1) J - estimated value
 (2) Concentration used for screening is the maximum detected concentration.
 (3) N/A - No background screening performed.
 (4) Primary and Secondary Drinking Water Regulations
 (5) USEPA - OSWER Draft Guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils (Subsurface Vapor Intrusion Guidance) Tables. November 2002. ca = Cancer; nc = Noncancer. Screening criteria correspond to a cancer risk of 10-6 and a noncancer hazard of 0.1. For USEPA (2002) criteria that defaulted to MCLs, criteria were derived (in italics) from USEPA (2009) RSL residential air concentration based on an attenuation factor of 10 and the Henry's Law constant for each compound at 25 deg C.
 (6) Selection Rationale: ASL - Above Screening Level; TOX - Class A Carcinogen; NTX - No Toxicity Information. Deletion Rationale: BSL - Below Screening Level; INC - Pathway Incomplete

Definitions:

ARAR: Applicable or Relevant and Appropriate Requirements
 CAS: Chemical Abstract Service
 COPC: Compound of Potential Concern
 NV: No Value
 TBC: To Be Considered
 USEPA: United States Environmental Protection Agency

TABLE 2.40b
DERIVATION OF TOTAL XYLENE CONCENTRATIONS
HONEYWELL, WASTEBED B/HARBOR BROOK SITE - SYW-12 SHALLOW GROUND WATER: VAPOR INTRUSION

Sample Location	Sample Date	Start Depth (ft)	End Depth (ft)	CAS Number	Chemical	Detect (Y/N)	Data Qualifier	Units	Reported Value	Total Xylene Value ^a
HB-B-04W	3/7/2007	6	11	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	15.2	15.2
HB-B-08W	3/5/2007	6	11	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.58	0.58
HB-B-10	3/7/2007	6	11	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-01	12/18/2006	8	10	1330-20-7	XYLENES, TOTAL	Y		ug/l	1.68	1.68
HB-GWS-02	12/18/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-03	12/19/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-04	12/20/2006	8	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.38	0.38
HB-GWS-05	12/11/2006	10	12	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-06	12/15/2006	8	10	1330-20-7	XYLENES, TOTAL	N	UJ	ug/l	1	0.5
HB-GWS-07	12/14/2006	8	10	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.11	0.11
HB-GWS-08	12/13/2006	8	10	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-GWS-09	12/12/2006	10	12	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-22	3/5/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-23	3/5/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-24	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-25	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5
HB-MW-26	3/5/2007	5	15	1330-20-7	XYLENES, TOTAL	Y	J	ug/l	0.45	0.45
HB-MW-27	3/7/2007	4	14	1330-20-7	XYLENES, TOTAL	N	U	ug/l	1	0.5

Notes:

a - Total Xylene value utilized in the risk assessment.

RAGS Table 3 Series

TABLE 3.1a
EXPOSURE POINT CONCENTRATION SUMMARY- EXPOSURE UNIT 1 - SITE WIDE SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Site Wide	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	89.16	516.4	575.5	5.16E+02	ng/kg	% UCL	99% Chebyshev (Mean, Sd) UCL
	METALS									
	7429-90-5	ALUMINUM	mg/kg	6651	7128	24400	7.13E+03	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-36-0	ANTIMONY	mg/kg	0.65	0.67	4.9	6.66E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-38-2	ARSENIC	mg/kg	8.30	9.18	34.4	9.18E+00	mg/kg	% UCL	95% KM (BCA) UCL
	7440-39-3	BARIUM	mg/kg	256.1	268.6	4880	2.69E+02	mg/kg	% UCL	95% H-UCL
	7440-43-9	CADMIUM	mg/kg	18.05	24.07	110	2.41E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	67.96	113.4	391	1.13E+02	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	7440-50-8	COPPER	mg/kg	117.9	203.7	744	2.04E+02	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	7439-89-6	IRON	mg/kg	13271	14049	30000	1.40E+04	mg/kg	% UCL	95% Student's-t UCL
	7439-92-1	LEAD	mg/kg	270.1	400.6	2320	2.70E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	289.4	306.6	722	3.07E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-97-6	MERCURY	mg/kg	5.207	8.244	64.3	8.24E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-22-4	SILVER	mg/kg	21.35	10.03	91.9	1.00E+01	mg/kg	% UCL	95% KM (t) UCL
	7440-28-0	THALLIUM	mg/kg	0.97	0.70	2.3	7.02E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	20.23	20.98	49.1	2.10E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.28	0.90	2	8.97E-01	mg/kg	% UCL	95% Adjusted Gamma UCL
		HIGHLY CHLORINATED PCBs ^b	mg/kg	1.10	1.43	6	1.43E+00	mg/kg	% UCL	95% Approximate Gamma UCL
		TOTAL PCBs ^c	mg/kg	1.14	1.48	6	1.48E+00	mg/kg	% UCL	95% Approximate Gamma UCL
	PESTICIDES									
	60-57-1	DIELDRIN	mg/kg	0.11	0.011	0.2	1.11E-02	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	3.13	8.70	130	8.70E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	3.09	5.47	37	5.47E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	7.21	15.24	120	1.52E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	7.30	14.88	100	1.49E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	6.59	12.86	81	1.29E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	4.97	9.82	69	9.82E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	5.72	11.96	94	1.20E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	7.33	15.04	110	1.50E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	2.08	3.27	22	3.27E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	2.22	3.28	53	3.28E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	mg/kg	14.5	33.07	310	3.31E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	118-74-1	HEXACHLOROBENZENE	mg/kg	1.85	0.65	11	6.52E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	4.59	9.08	64	9.08E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	7.79	15.74	300	1.57E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	10.29	22.74	210	2.27E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

TABLE 3.1a
EXPOSURE POINT CONCENTRATION SUMMARY- EXPOSURE UNIT 1 - SITE WIDE SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Site Wide	VOCs									
	87-61-6	1,2,3-TRICHLOROBENZENE	mg/kg	4.18	2.38	8.3	2.38E+00	mg/kg	% UCL	99% KM (Chebyshev) UCL
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	7.87	2.72	53	2.72E+00	mg/kg	% UCL	95% KM (t) UCL
	95-50-1	1,2-DICHLOROBENZENE	mg/kg	9.98	5.42	210	5.42E+00	mg/kg	% UCL	95% KM (t) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	14.08	19.67	350	1.97E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	0.33	0.35	4.2	3.54E-01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.0042	0.0026	0.009	2.57E-03	mg/kg	% UCL	95% KM (t) UCL
	OTHER									
	112-40-3	DODECANE	mg/kg	845	813.5	1100	8.14E+02	mg/kg	% UCL	95% KM (t) UCL

Footnotes:

a = Aroclor-1016, -1221, -1232, -1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248, -1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Chebyshev (Mean, Sd) UCL = $(1-\alpha)100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Chebyshev (MVUE) UCL = $(1-\alpha)100\%$ UCL of the Mean of a Lognormal Population Based Upon the Chebyshev Theorem (Using the MVUE of the Mean and its Standard Error - parametric)

Gamma UCL = Computation of UCL of the mean of a Gamma, $G(k,\theta)$ distribution (parametric).

H-UCL = $(1-\alpha)100\%$ UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier Estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.1b
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SUBSURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Site-Wide	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	89.16	516.4	575.5	5.16E+02	ng/kg	% UCL	99% Chebyshev (Mean, Sd) UCL
	METALS									
	7429-90-5	ALUMINUM	mg/kg	6635	7047	24900	7.05E+03	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-36-0	ANTIMONY	mg/kg	0.67	0.63	4.9	6.29E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-38-2	ARSENIC	mg/kg	9.77	11.08	103	1.11E+01	mg/kg	% UCL	95% KM (BCA) UCL
	7440-39-3	BARIUM	mg/kg	271	517.5	4880	5.18E+02	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	7440-43-9	CADMIUM	mg/kg	15.05	20.2	110	2.02E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	62.15	100.3	391	1.00E+02	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	7440-50-8	COPPER	mg/kg	112.2	182.6	744	1.83E+02	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	7439-89-6	IRON	mg/kg	13103	13848	34400	1.38E+04	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-92-1	LEAD	mg/kg	240.8	326.1	2320	2.41E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	290.5	307.2	1390	3.07E+02	mg/kg	% UCL	95% Student's-t UCL
	7439-97-6	MERCURY	mg/kg	6.48	11.7	97	1.17E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-22-4	SILVER	mg/kg	18.53	15.21	102	1.52E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-28-0	THALLIUM	mg/kg	2.73	1.26	38.5	1.26E+00	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	19.07	19.75	49.1	1.98E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.63	4.93	3	3.00E+00	mg/kg	Max	Insufficient Data*
		HIGHLY CHLORINATED PCBs ^b	mg/kg	1.03	2.82	6	2.82E+00	mg/kg	% UCL	95% H-UCL
		TOTAL PCBs ^c	mg/kg	1.13	2.01	6	2.01E+00	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	PESTICIDES									
	60-57-1	DIELDRIN	mg/kg	0.072	0.0097	0.2	9.72E-03	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	105-67-9	2,4-DIMETHYLPHENOL	mg/kg	30.59	3.288	190	3.29E+00	mg/kg	% UCL	95% KM (t) UCL
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	85.28	238.5	3800	2.39E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	34METPH	3&4-METHYLPHENOL	mg/kg	47.83	9.40	500	9.40E+00	mg/kg	% UCL	95% KM (t) UCL
	83-32-9	ACENAPHTHENE	mg/kg	34.23	75.83	1400	7.58E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	11.68	38.52	850	3.85E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	120-12-7	ANTHRACENE	mg/kg	40.66	140.6	3000	1.41E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	23.49	92.41	2000	9.24E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	17.87	65.3	1400	6.53E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	21.17	86.01	1900	8.60E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	7.38	20.22	380	2.02E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	10.85	35.91	740	3.59E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	86-74-8	CARBAZOLE	mg/kg	21.59	65.31	1500	6.53E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	21.03	79.21	1700	7.92E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	3.336	7.04	130	7.04E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	42.94	103.8	1800	1.04E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	mg/kg	61.98	265.8	5800	2.66E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	86-73-7	FLUORENE	mg/kg	66.05	144.7	2700	1.45E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	118-74-1	HEXACHLORO BENZENE	mg/kg	2.35	0.73	13	7.31E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	7.24	20.95	410	2.10E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	440.5	1012	21000	1.01E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	104.8	439.2	9300	4.39E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	129-00-0	PYRENE	mg/kg	48.9	212.6	4700	2.13E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

TABLE 3.1b
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SUBSURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Site-Wide	VOCs									
	95-50-1	1,2-DICHLOROBENZENE	mg/kg	67.61	92.97	2700	9.30E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	87-61-6	1,2,3-TRICHLOROBENZENE	mg/kg	7.24	1.24	18	1.24E+00	mg/kg	% UCL	95% KM (t) UCL
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	19.88	15.21	350	1.52E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	95-63-6	1,2,4-TRIMETHYLBENZENE	mg/kg	28.53	42.62	390	4.26E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	108-67-8	1,3,5-TRIMETHYLBENZENE	mg/kg	16.75	8.60	160	8.60E+00	mg/kg	% UCL	95% KM (t) UCL
	541-73-1	1,3-DICHLOROBENZENE	mg/kg	5.84	1.40	41	1.40E+00	mg/kg	% UCL	95% KM (t) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	70.97	113.7	3400	1.14E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	5.83	8.88	190	8.88E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	74-83-9	BROMOMETHANE	mg/kg	0.76	NA	0.76	7.60E-01	mg/kg	Max	Insufficient Data*
	108-90-7	CHLOROBENZENE	mg/kg	6.68	8.12	120	8.12E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	1.31	1.17	8.7	1.17E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	108-88-3	TOLUENE	mg/kg	15.82	22.33	450	2.23E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	1330-20-7	XYLENES, TOTAL	mg/kg	37.5	74.76	860	7.48E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	OTHER									
	112-40-3	DODECANE	mg/kg	845	813.5	1100	8.14E+02	mg/kg	% UCL	95% KM (t) UCL

Footnotes:

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d= Max - maximum detected concentration; %UCL - % upper confidence limit.

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Chebyshev (Mean, Sd) UCL = $(1 - \alpha) 100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Gamma UCL = Computation of UCL of the mean of a Gamma, $G(k, \theta)$ distribution (parametric).

H-UCL = $(1 - \alpha) 100\%$ UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

Table 3.1c
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SURFACE SEDIMENTS
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment (0 - 1 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site-Wide	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	28.35	65.59	458.6	6.56E+01	ng/kg	% UCL	Use 95% H-UCL
	METALS									
	7440-38-2	ARSENIC	mg/kg	6.05	6.60	22	6.60E+00	mg/kg	% UCL	95% KM (BCA) UCL
	7440-43-9	CADMIUM	mg/kg	1.61	1.50	19.2	1.50E+00	mg/kg	% UCL	95% KM (t) UCL
	7440-47-3	CHROMIUM	mg/kg	40.97	48.52	534	4.85E+01	mg/kg	% UCL	Use 95% H-UCL
	7439-89-6	IRON	mg/kg	9826	10863	21300	1.09E+04	mg/kg	% UCL	Use 95% Student's-t UCL
	7439-92-1	LEAD	mg/kg	95.60	114.8	479	9.56E+01	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	243.6	265.5	664	2.66E+02	mg/kg	% UCL	Use 95% Student's-t UCL
	7439-97-6	MERCURY	mg/kg	2.40	5.31	52	5.31E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-28-0	THALLIUM	mg/kg	1.97	0.62	4.9	6.23E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	13.30	13.69	27.1	1.37E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		HIGHLY CHLORINATED PCBs ^a	mg/kg	0.50	0.76	4.7	7.59E-01	mg/kg	% UCL	Use 95% H-UCL
		TOTAL PCBs ^b	mg/kg	0.51	0.76	4.7	7.58E-01	mg/kg	% UCL	Use 95% H-UCL
	PESTICIDES									
	60-57-1	DIELDRIN	mg/kg	0.04	0.02	0.069	1.60E-02	mg/kg	% UCL	95% KM (t) UCL
	53494-70-5	ENDRIN KETONE	mg/kg	0.09	0.05	0.15	4.93E-02	mg/kg	% UCL	95% KM (t) UCL
	1024-57-3	HEPTACHLOR EPOXIDE	mg/kg	0.02	0.01	0.063	7.65E-03	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	90-12-0	1-METHYLNAPHTHALENE	mg/kg	2.027	3.73	4.6	3.73E+00	mg/kg	% UCL	Use 95% Student's-t UCL
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	12.20	29.13	210	2.91E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	3.18	3.74	51	3.74E+00	mg/kg	% UCL	95% KM (BCA) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	15.91	60.84	460	2.75E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	16.30	62.88	480	6.29E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	21.81	94.36	720	9.44E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	11.24	39.78	280	3.98E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	10.08	35.26	270	3.53E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	mg/kg	13.39	37.79	290	3.78E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	86-74-8	CARBAZOLE	mg/kg	4.65	12.38	93	1.24E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	20.50	86.73	650	8.67E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	4.30	10.17	72	1.09E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	10.42	21.75	100	2.18E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	mg/kg	34.10	134.4	990	1.34E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	118-74-1	HEXACHLOROBENZENE	mg/kg	0.11	0.13	0.53	1.25E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	9.85	32.73	230	3.27E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	32.55	59.97	240	6.00E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	33.47	112.4	780	1.12E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	129-00-0	PYRENE	mg/kg	40.35	173.2	1300	1.73E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

Table 3.1c
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SURFACE SEDIMENTS
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment (0 - 1 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site-Wide	VOCs									
	95-50-1	1,2-DICHLOROBENZENE	mg/kg	9.27	13.16	120	1.32E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	87-61-6	1,2,3-TRICHLOROBENZENE	mg/kg	0.79	0.535	3.7	5.35E-01	mg/kg	% UCL	95% KM (t) UCL
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	2.22	0.52	8.1	5.17E-01	mg/kg	% UCL	95% KM (t) UCL
	108-70-3	1,3,5-TRICHLOROBENZENE	mg/kg	2.09	16.83	15	1.68E+01	mg/kg	% UCL	99% KM (Chebyshev) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	14.52	22.45	160	2.25E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	1.79	3.91	29	3.91E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	108-90-7	CHLOROBENZENE	mg/kg	8.15	21.57	240	2.16E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	544-76-3	N-HEXADACANE	mg/kg	0.68	NA	0.83	8.30E-01	mg/kg	Max	Max
	75-09-2	METHYLENE CHLORIDE	mg/kg	2.41	0.41	9.5	4.10E-01	mg/kg	% UCL	95% KM (t) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.01	0.01	0.017	9.23E-03	mg/kg	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	mg/kg	4.06	9.85	88	9.85E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	1330-20-7	XYLENES, TOTAL	mg/kg	10.25	34.80	314	3.48E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

Footnotes:

a = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

c = Max - maximum detected concentration; %UCL - % upper confidence limit. KM - Kaplan-Meier method.

Chebyshev UCL or Student's-t UCL for nonparametric distribution based on ProUCL (V. 4.0) recommendation; Gamma UCL for gamma distribution; Nonparametric Bootstrap UCLs introduce some standard error and may not be reproduced precisely.

NA = UCL could not be calculated because there were too few samples or too few detected samples among many non-detected samples

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95% UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

H-UCL = (1- α)100% UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

Table 3.1d
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE UPPER SEDIMENTS
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment (0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site-Wide	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	24.28	55.72	458.6	5.57E+01	mg/kg	% UCL	Use 95% H-UCL
	METALS									
	7440-38-2	ARSENIC	mg/kg	6.04	2.76	22	2.76E+00	mg/kg	% UCL	95% KM (t) UCL
	7440-39-3	BARIUM	mg/kg	126.0	143.8	752	1.44E+02	mg/kg	% UCL	95% KM (BCA) UCL
	7440-43-9	CADMIUM	mg/kg	1.45	1.12	19.2	1.12E+00	mg/kg	% UCL	95% KM (t) UCL
	7440-47-3	CHROMIUM	mg/kg	40.51	45.60	534	4.56E+01	mg/kg	% UCL	95% KM (BCA) UCL
	7439-89-6	IRON	mg/kg	9279	11388	21300	1.14E+04	mg/kg	% UCL	Use 95% Chebyshev (Mean, Sd) UCL
	7439-92-1	LEAD	mg/kg	111.3	154.6	753	1.11E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	274.8	331.5	723	3.32E+02	mg/kg	% UCL	Use 95% Chebyshev (Mean, Sd) UCL
	7439-97-6	MERCURY	mg/kg	2.36	5.34	52	5.34E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-28-0	THALLIUM	mg/kg	0.85	0.48	4.9	4.81E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	11.55	12.01	27.1	1.20E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		HIGHLY CHLORINATED PCBs ^a	mg/kg	0.41	0.69	3.7	6.91E-01	mg/kg	% UCL	Use 95% H-UCL
		TOTAL PCBs ^b	mg/kg	0.42	0.70	3.7	6.95E-01	mg/kg	% UCL	Use 95% H-UCL
	PESTICIDES									
	319-86-8	DELTA-BHC	mg/kg	0.0045	NA	0.0045	4.50E-03	mg/kg	Max	Max
	53494-70-5	ENDRIN KETONE	mg/kg	0.0850	0.05	0.15	4.71E-02	mg/kg	% UCL	95% KM (t) UCL
	1024-57-3	HEPTACHLOR EPOXIDE	mg/kg	0.0170	0.006	0.063	6.00E-03	mg/kg	% UCL	95% KM (t) UCL
	60-57-1	DIELDRIN	mg/kg	0.03	0.01	0.069	1.32E-02	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	90-12-0	1-METHYLNAPHTHALENE	mg/kg	2.03	3.73	4.6	3.73E+00	mg/kg	% UCL	Use 95% Student's-t UCL
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	442.0	2226	32000	2.23E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	83-32-9	ACENAPHTHENE	mg/kg	96.41	454.4	6500	4.54E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	124.80	557.0	8300	5.57E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	120-12-7	ANTHRACENE	mg/kg	73.89	357.3	5100	3.57E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	33.88	144.6	1900	1.84E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	13.14	42.55	480	4.26E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	17.71	62.37	720	6.24E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	8.99	26.39	280	2.64E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	8.45	23.99	270	2.40E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	mg/kg	12.30	18.88	290	1.89E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	86-74-8	CARBAZOLE	mg/kg	25.92	115.2	1700	1.15E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	33.72	137.7	1700	1.38E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	3.52	5.37	72	7.51E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	151.1	748.7	11000	7.49E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	mg/kg	116.2	620.6	8300	6.21E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	86-73-7	FLUORENE	mg/kg	161.4	832.5	12000	8.33E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	118-74-1	HEXACHLOROBENZENE	mg/kg	0.11	0.09	0.53	8.59E-02	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	8.06	22.06	230	2.21E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	1147	6160	97000	6.16E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	285.5	1702	23000	1.70E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	129-00-0	PYRENE	mg/kg	91.71	440.6	5700	4.41E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

Table 3.1d
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE UPPER SEDIMENTS
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment (0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site-Wide	VOCs									
	95-50-1	1,2-DICHLOROBENZENE	mg/kg	9.01	9.34	120	9.34E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	87-61-6	1,2,3-TRICHLOROBENZENE	mg/kg	0.79	0.54	3.7	5.35E-01	mg/kg	% UCL	95% KM (t) UCL
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	2.22	0.38	8.1	3.76E-01	mg/kg	% UCL	95% KM (t) UCL
	108-70-3	1,3,5-TRICHLOROBENZENE	mg/kg	2.09	16.83	15	1.68E+01	mg/kg	% UCL	99% KM (Chebyshev) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	13.87	15.97	160	1.60E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	591-78-6	2-HEXANONE	mg/kg	442	2226	32000	2.23E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	14.46	63.92	1100	6.39E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	108-90-7	CHLOROBENZENE	mg/kg	7.69	14.81	240	1.48E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	100-41-4	ETHYLBENZENE	mg/kg	26.69	107.9	1800	1.08E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	544-76-3	N-HEXADACANE	mg/kg	0.68	NA	0.83	8.30E-01	mg/kg	Max	Max
	75-09-2	METHYLENE CHLORIDE	mg/kg	2.79	0.92	30	9.17E-01	mg/kg	% UCL	95% KM (t) UCL
	100-42-5	STYRENE	mg/kg	122.0	106.6	1700	1.07E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.02	0.01	0.029	1.46E-02	mg/kg	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	mg/kg	56.91	257.70	4400	2.58E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	1330-20-7	XYLENES, TOTAL	mg/kg	118.6	641.3	10500	6.41E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

Footnotes:

a = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

c = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because there were too few samples or too few detected samples among many non-detected samples

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95% UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

Chebyshev (Mean, Sd) UCL = $(1-\alpha)100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Chebyshev (MVUE) UCL = $(1-\alpha)100\%$ UCL of the Mean of a Lognormal Population Based Upon the Chebyshev Theorem (Using the MVUE of the Mean and its Standard Error - parametric)

KM (BCA) UCL = UCL based upon Kaplan-Meier Estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

KM (Percentile Bootstrap) UCL = $(1-\alpha)100\%$ UCL of the Mean Based Upon Simple Percentile Bootstrap Method (non-parametric)

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

95% H-UCL = $(1-\alpha)100\%$ UCL of the mean based upon H-statistic (H-UCL) (parametric).

TABLE 3.1e
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SHALLOW GROUND WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water
(0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site-Wide	METALS									
	7429-90-5	ALUMINUM	mg/L	1.842	3.592	15.1	3.59E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-36-0	ANTIMONY	mg/L	0.0031	0.0024	0.0049	2.43E-03	mg/kg	% UCL	95% KM (t) UCL
	7440-38-2	ARSENIC	mg/L	0.0083	0.0064	0.0181	6.40E-03	mg/kg	% UCL	95% KM (t) UCL
	7440-39-3	BARIUM	mg/L	1.016	3.572	20.3	3.57E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-43-9	CADMIUM	mg/L	0.0051	0.0019	0.012	1.91E-03	mg/kg	% UCL	95% KM (t) UCL
	7440-47-3	CHROMIUM	mg/L	0.020	0.017	0.053	1.74E-02	mg/kg	% UCL	95% KM (Percentile Bootstrap) UCL
	57-12-5	CYANIDE	mg/L	0.037	0.027	0.12	2.68E-02	mg/kg	% UCL	95% KM (t) UCL
	7439-89-6	IRON	mg/L	4.59	11	43	1.10E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7439-92-1	LEAD	mg/L	0.018	0.014	0.10	1.75E-02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/L	0.585	1.268	5.11	1.27E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7439-97-6	MERCURY	mg/L	0.0023	0.0014	0.0088	1.36E-03	mg/kg	% UCL	95% KM (t) UCL
	7440-22-4	SILVER	mg/L	0.0082	0.0025	0.025	2.49E-03	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/L	0.0073	0.0100	0.028	9.99E-03	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		HIGHLY CHLORINATED PCBs ^a	ug/L	0.07	NA	0.07	7.00E-02	ug/L	Max	Insufficient Data*
		TOTAL PCBs ^b	ug/L	0.07	NA	0.07	7.00E-02	ug/L	Max	Insufficient Data*
	PESTICIDES									
	50-29-3	4,4'-DDT	ug/L	20	NA	20	2.00E+01	mg/kg	Max	Insufficient Data*
	SVOCs									
	92-52-4	1,1'-BIPHENYL	ug/L	26.62	21.91	83	2.19E+01	mg/kg	% UCL	95% KM (t) UCL
	120-83-2	2,4-DICHLOROPHENOL	ug/L	33.57	13.76	75	1.38E+01	mg/kg	% UCL	95% KM (t) UCL
	105-67-9	2,4-DIMETHYLPHENOL	ug/L	2015	847.5	7500	8.48E+02	mg/kg	% UCL	95% KM (t) UCL
	91-57-6	2-METHYLNAPHTHALENE	ug/L	476.8	1304	9800	1.30E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	95-48-7	2-METHYLPHENOL	ug/L	1600	1788	8000	1.79E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	88-75-5	2-NITROPHENOL	ug/L	2.8	3.134	3	3.00E+00	mg/kg	Max	Insufficient Data*
	34METPH	3&4-METHYLPHENOL	ug/L	2277	5550	16000	5.55E+03	mg/kg	% UCL	99% KM (Chebyshev) UCL
	106-44-5	4-METHYLPHENOL	ug/L	1806	2558	12000	2.56E+03	mg/kg	% UCL	95% KM (BCA) UCL
	100-02-7	4-NITROPHENOL	ug/L	5.5	9.68	8	8.00E+00	mg/kg	Max	Insufficient Data*
	83-32-9	ACENAPHTHENE	ug/L	140.2	218.4	2200	2.18E+02	mg/kg	% UCL	95% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	ug/L	280.9	145.8	2700	1.46E+02	mg/kg	% UCL	95% KM (t) UCL
	120-12-7	ANTHRACENE	ug/L	242.9	101.8	2000	1.02E+02	mg/kg	% UCL	95% KM (t) UCL
	56-55-3	BENZ(A)ANTHRACENE	ug/L	150.7	39.54	690	3.95E+01	mg/kg	% UCL	95% KM (t) UCL
	50-32-8	BENZO(A)PYRENE	ug/L	129.3	33	310	3.30E+01	mg/kg	% UCL	95% KM (t) UCL
	205-99-2	BENZO(B)FLUORANTHENE	ug/L	87	24.18	240	2.42E+01	mg/kg	% UCL	95% KM (t) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	ug/L	27	NA	27	2.70E+01	mg/kg	Max	Insufficient Data*
	207-08-9	BENZO(K)FLUORANTHENE	ug/L	183.5	66.73	340	6.67E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	ug/L	21.26	19.74	110	1.97E+01	mg/kg	% UCL	95% KM (t) UCL
	86-74-8	CARBAZOLE	ug/L	101.6	66.51	840	6.65E+01	mg/kg	% UCL	95% KM (t) UCL
	218-01-9	CHRYSENE	ug/L	110.1	33.6	590	3.36E+01	mg/kg	% UCL	95% KM (t) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	ug/L	10	NA	10	1.00E+01	mg/kg	Max	Insufficient Data*
	132-64-9	DIBENZOFURAN	ug/L	220.4	442.3	3400	4.42E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	ug/L	226.5	411.6	3200	4.12E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	86-73-7	FLUORENE	ug/L	240.7	406.8	4200	4.07E+02	mg/kg	% UCL	95% KM (Chebyshev) UCL
	87-68-3	HEXACHLOROBUTADIENE	ug/L	1	NA	1	1.00E+00	mg/kg	Max	Insufficient Data*

TABLE 3.1e
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SHALLOW GROUND WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water
(0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site-Wide	193-39-5	INDENO(1,2,3-CD)PYRENE	ug/L	68.5	32.14	110	3.21E+01	mg/kg	% UCL	95% KM (t) UCL
	91-20-3	NAPHTHALENE	ug/L	2549	5371	35000	5.37E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	ug/L	396.9	1058	8300	1.06E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	108-95-2	PHENOL	ug/L	1402	3268	18000	3.27E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	129-00-0	PYRENE	ug/L	157.4	248.3	1900	2.48E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	VOCs									
	87-61-6	1,2,3-TRICHLOROBENZENE	ug/L	10	26.83	19	1.90E+01	mg/kg	Max	Insufficient Data*
	120-82-1	1,2,4-TRICHLOROBENZENE	ug/L	122.2	58.48	230	5.85E+01	mg/kg	% UCL	95% KM (t) UCL
	95-63-6	1,2,4-TRIMETHYLBENZENE	ug/L	135.6	162.2	420	1.62E+02	mg/kg	% UCL	95% KM (BCA) UCL
	95-50-1	1,2-DICHLOROBENZENE	ug/L	1435	1156	6480	1.16E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	108-67-8	1,3,5-TRIMETHYLBENZENE	ug/L	72.54	78.96	250	7.90E+01	mg/kg	% UCL	95% KM (t) UCL
	541-73-1	1,3-DICHLOROBENZENE	ug/L	19.6	7.02	62	7.02E+00	mg/kg	% UCL	95% KM (t) UCL
	106-46-7	1,4-DICHLOROBENZENE	ug/L	779.4	847.2	4500	8.47E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	591-78-6	2-HEXANONE	ug/L	0.97	2.34	2.6	2.34E+00	mg/kg	% UCL	95% KM (t) UCL
	71-43-2	BENZENE	ug/L	626	1112	3900	1.11E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	75-27-4	BROMODICHLOROMETHANE	ug/L	0.6	NA	0.6	6.00E-01	mg/kg	Max	Insufficient Data*
	108-90-7	CHLOROBENZENE	ug/L	461.9	205.2	3080	2.05E+02	mg/kg	% UCL	95% KM (t) UCL
	67-66-3	CHLOROFORM	ug/L	8.27	2.11	27	2.11E+00	mg/kg	% UCL	95% KM (t) UCL
	100-41-4	ETHYLBENZENE	ug/L	75.7	100.5	350	1.01E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	98-82-8	ISOPROPYLBENZENE	ug/L	15.7	9.972	68	9.97E+00	mg/kg	% UCL	95% KM (t) UCL
	75-09-2	METHYLENE CHLORIDE	ug/L	15.25	25	25	2.50E+01	mg/kg	% UCL	95% KM (BCA) UCL
	99-87-6	P-ISOPROPYLTOLUENE	ug/L	10.4	28.35	20	2.00E+01	mg/kg	Max	Insufficient Data*
	135-98-8	SEC-BUTYLBENZENE	ug/L	60.5	133.9	120	1.20E+02	mg/kg	Max	Insufficient Data*
	100-42-5	STYRENE	ug/L	337.1	271	850	2.71E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	127-18-4	TETRACHLOROETHENE	ug/L	0.57	0.47	1.7	4.65E-01	mg/kg	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	ug/L	1085	1848	5740	1.85E+03	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	75-01-4	VINYL CHLORIDE	ug/L	2.43	1.41	4.1	1.41E+00	mg/kg	% UCL	95% KM (t) UCL
	1330-20-7	XYLENES, TOTAL	ug/L	573.8	2119	3380	2.12E+03	mg/kg	% UCL	99% KM (Chebyshev) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

c = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95% UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (Percentile Bootstrap) UCL - (1-α) 100% UCL of the Mean Based Upon Simple Percentile Bootstrap Method (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

TABLE 3.1f
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SURFACE WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
Site-Wide	METALS									
	7440-36-0	ANTIMONY	mg/L	0.0020	0.0018	0.0028	1.83E-03	mg/L	% UCL	95% KM (t) UCL
	7440-38-2	ARSENIC	mg/L	0.0060	0.0033	0.012	3.26E-03	mg/L	% UCL	95% KM (t) UCL
	7440-47-3	CHROMIUM	mg/L	0.0063	0.0060	0.016	5.96E-03	mg/L	% UCL	95% KM (t) UCL
	7439-89-6	IRON	mg/L	1.36	5.81	12.28	5.81E+00	mg/L	% UCL	99% KM (Chebyshev) UCL
	7439-92-1	LEAD	mg/L	0.014	0.010	0.036	1.42E-02	mg/L	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/L	0.14	0.41	1.8	4.10E-01	mg/L	% UCL	97.5% KM (Chebyshev) UCL
	7439-97-6	MERCURY	mg/L	0.00012	0.000095	0.00048	9.55E-05	mg/L	% UCL	95% KM (t) UCL
	7440-28-0	THALLIUM	mg/L	0.0038	NA	0.0038	3.80E-03	mg/L	Max	Insufficient Data*
	7440-62-2	VANADIUM	mg/L	0.0016	0.0016	0.0037	1.58E-03	mg/L	% UCL	95% KM (t) UCL
	7440-66-6	ZINC	mg/L	0.20	0.35	1.41	3.46E-01	mg/L	% UCL	97.5% KM (Chebyshev) UCL
	SVOCs									
	95-63-6	1,2,4-TRIMETHYLBENZENE	ug/L	13.8	23.4	67	2.34E+01	ug/L	% UCL	95% KM (BCA) UCL
	108-67-8	1,3,5-TRIMETHYLBENZENE	ug/L	6.58	8.52	26	8.52E+00	ug/L	% UCL	95% KM (t) UCL
	105-67-9	2,4-DIMETHYLPHENOL	ug/L	37.65	51.79	190	5.18E+01	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	91-57-6	2-METHYLNAPHTHALENE	ug/L	88.37	62.8	300	6.28E+01	ug/L	% UCL	95% KM (t) UCL
	83-32-9	ACENAPHTHENE	ug/L	25.2	15.67	49	1.57E+01	ug/L	% UCL	95% KM (t) UCL
	208-96-8	ACENAPHTHYLENE	ug/L	22.42	13.1	55	1.31E+01	ug/L	% UCL	95% KM (t) UCL
	56-55-3	BENZ(A)ANTHRACENE	ug/L	2.2	3.75	4	3.75E+00	ug/L	% UCL	95% KM (t) UCL
	50-32-8	BENZO(A)PYRENE	ug/L	1.73	2.19	2	2.00E+00	ug/L	Max	Max
	205-99-2	BENZO(B)FLUORANTHENE	ug/L	1.93	2.86	3	2.86E+00	ug/L	% UCL	95% KM (t) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	ug/L	1.9	NA	1.9	1.90E+00	ug/L	Max	Insufficient Data*
	207-08-9	BENZO(K)FLUORANTHENE	ug/L	1.6	NA	1.6	1.60E+00	ug/L	Max	Insufficient Data*
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	ug/L	4	5.94	7.8	5.94E+00	ug/L	% UCL	95% KM (t) UCL
	86-74-8	CARBAZOLE	ug/L	24.94	14.51	56	1.45E+01	ug/L	% UCL	95% KM (t) UCL
	218-01-9	CHRYSENE	ug/L	1.96	2.86	4	2.86E+00	ug/L	% UCL	95% KM (t) UCL
	132-64-9	DIBENZOFURAN	ug/L	30.35	18.43	73	1.84E+01	ug/L	% UCL	95% KM (t) UCL
	86-73-7	FLUORENE	ug/L	19.95	13.55	42	1.36E+01	ug/L	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	ug/L	1.4	NA	1.4	1.40E+00	ug/L	Max	Insufficient Data*
	91-20-3	NAPHTHALENE	ug/L	520	1096	2200	1.10E+03	ug/L	% UCL	99% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	ug/L	25.52	18.5	83	1.85E+01	ug/L	% UCL	95% KM (t) UCL
	129-00-0	PYRENE	ug/L	6.97	5.79	28	5.79E+00	ug/L	% UCL	95% KM (BCA) UCL

TABLE 3.1f
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - SITE WIDE SURFACE WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
Site-Wide	VOCs									
	106-46-7	1,4-DICHLOROBENZENE	ug/L	8	NA	8	8.00E+00	ug/L	Max	Insufficient Data*
	34METPH	3&4-METHYLPHENOL	ug/L	132.1	58.81	280	5.88E+01	ug/L	% UCL	95% KM (t) UCL
	71-43-2	BENZENE	ug/L	53.54	44.53	200	4.45E+01	ug/L	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	ug/L	77.02	223.2	410	2.23E+02	ug/L	% UCL	99% KM (Chebyshev) UCL
	1330-20-7	XYLENES, TOTAL	ug/L	72.51	473.6	470	4.70E+02	ug/L	% UCL	99% KM (Chebyshev) UCL
	OTHER									
	25321-22-6	DICHLOROBENZENES	ug/L	7.24	NA	9.68	9.68E+00	ug/L	Max	Insufficient Data*

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Max - maximum detected concentration; %UCL - % upper confidence limit. KM - Kaplan-Meier method.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

TABLE 3.1g
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 1 - FISH TISSUE
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Fish Tissue
Exposure Medium: Fish Tissue

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Fish Fillet Tissue from Onodaga Lake	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	mg/kg	0.000010	0.000020	0.000046	2.00E-05	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	METALS									
	7440-36-0	ANTIMONY	mg/kg	0.56	0.99	2.1	9.90E-01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-38-2	ARSENIC	mg/kg	0.33	0.080	1.05	8.00E-02	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-47-3	CHROMIUM	mg/kg	0.49	0.57	0.73	5.70E-01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	57-12-5	CYANIDE	mg/kg	1.73	5.69	14.3	5.70E+00	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7439-96-5	MANGANESE	mg/kg	0.99	3.23	5.51	3.20E+00	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	22967-92-6	MERCURY (AS METHYLMERCURY)	mg/kg	1.05	1.08	5.07	1.10E+00	mg/kg	% UCL	95% D'Agostino (Y) UCL
	7782-49-2	SELENIUM	mg/kg	1.08	1.47	2.2	1.50E+00	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-62-2	VANADIUM	mg/kg	0.48	0.63	0.97	6.30E-01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-66-6	ZINC	mg/kg	30.5	43.7	73.8	4.40E+01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.274	0.479	1.98	4.80E-01	mg/kg	% UCL	95% D'Agostino (Y) UCL
		HIGHLY CHLORINATED PCBs ^b	mg/kg	0.421	0.577	1.92	5.80E-01	mg/kg	% UCL	95% D'Agostino (Y) UCL
		TOTAL PCBs ^c	mg/kg	0.673	0.913	3.9	9.10E-01	mg/kg	% UCL	95% D'Agostino (Y) UCL
	PESTICIDES									
	3484-82-6	2,4'-DDE	mg/kg	0.00376	0.00413	0.024	4.10E-03	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	72-54-8	4,4'-DDD	mg/kg	0.0112	0.0133	0.17	1.30E-02	mg/kg	% UCL	95% D'Agostino (Y) UCL
	72-55-9	4,4'-DDE	mg/kg	0.0286	0.0344	0.4	3.40E-02	mg/kg	% UCL	95% D'Agostino (Y) UCL
	50-29-3	4,4'-DDT	mg/kg	0.00808	0.00949	0.082	9.50E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	309-00-2	ALDRIN	mg/kg	0.00232	0.00253	0.003	2.50E-03	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	319-86-8	DELTA-BHC	mg/kg	0.00231	0.00252	0.0028	2.50E-03	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	60-57-1	DIELDRIN	mg/kg	0.00379	0.00382	0.042	3.80E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	1024-57-3	HEPTACHLOR EPOXIDE	mg/kg	0.00355	0.00414	0.01	4.10E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	12789-03-6	CHLORDANE, TOTAL	mg/kg	0.00839	0.00973	0.061	9.70E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	SVOCs									
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	mg/kg	0.7	NA	2.3	2.30E+00	mg/kg	Max	Insufficient Data*
	118-74-1	HEXACHLOROBENZENE	mg/kg	0.0118	0.013	0.17	1.30E-02	mg/kg	% UCL	95% D'Agostino (Y) UCL

Footnotes:

Table modified from RAGS Table 3.1 Onondaga Lake Human Health Risk Assessment prepared by TAMS Consultants, Inc. & YEC, Inc.

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

* = Insufficient data to calculate a UCL, according to the Onondaga Lake Human Health Risk Assessment. In these cases, the EPC is obtained by using the maximum detected concentration.

D'Agostino (Y) UCL = Distributional fit for sample sets containing greater than 50 samples.

Shapiro-Wilk (W) UCL = Distributional fit for sample sets containing less than 50 samples.

TABLE 3.2
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 2 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, and DSA #2	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	129.3	434.6	575.5	4.35E+02	ng/kg	% UCL	Use 95% Adjusted Gamma UCL
	METALS									
	7429-90-5	ALUMINUM	mg/kg	6738	7502	24400	7.50E+03	mg/kg	% UCL	95% Student's-t UCL
	7440-38-2	ARSENIC	mg/kg	8.2	9.091	21.4	9.09E+00	mg/kg	% UCL	95% Student's-t UCL
	7440-39-3	BARIUM	mg/kg	399.3	450.8	4880	4.51E+02	mg/kg	% UCL	95% H-UCL
	7440-43-9	CADMIUM	mg/kg	25.36	44.57	110	4.46E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	94.76	123.7	391	1.24E+02	mg/kg	% UCL	95% H-UCL
	7440-50-8	COPPER	mg/kg	176.2	328.1	744	3.28E+02	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	7439-89-6	IRON	mg/kg	11956	12957	24400	1.30E+04	mg/kg	% UCL	95% Student's-t UCL
	7439-92-1	LEAD	mg/kg	332.9	432.4	1800	3.33E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	305.2	328.5	722	3.29E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-97-6	MERCURY	mg/kg	8.09	13.73	64.3	1.37E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-22-4	SILVER	mg/kg	24.35	19.35	91.9	1.94E+01	mg/kg	% UCL	95% KM (BCA) UCL
	7440-28-0	THALLIUM	mg/kg	0.97	0.81	2.3	8.07E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	20.62	21.48	49.1	2.15E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.31	0.72	2	7.21E-01	mg/kg	% UCL	95% Chebyshev (MVUE) UCL
		HIGHLY CHLORINATED PCBs ^b	mg/kg	1.24	1.65	6	1.65E+00	mg/kg	% UCL	95% Approximate Gamma UCL
		TOTAL PCBs PCBs ^c	mg/kg	1.24	1.65	6	1.65E+00	mg/kg	% UCL	95% Approximate Gamma UCL
	PESTICIDES									
	60-57-1	DIELDRIN	mg/kg	0.2	NA	0.2	2.00E-01	mg/kg	Max	Insufficient Data*
	SVOCs									
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	4.6	16.35	130	1.64E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	1.54	3.91	37	3.91E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	1.44	1.66	6.9	1.66E+00	mg/kg	% UCL	95% KM (BCA) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	1.58	1.77	6.4	1.77E+00	mg/kg	% UCL	95% KM (BCA) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	2.09	2.39	9.5	2.39E+00	mg/kg	% UCL	95% KM (BCA) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	1.05	1.17	4.7	1.17E+00	mg/kg	% UCL	95% KM (BCA) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	1.04	1.19	5	1.19E+00	mg/kg	% UCL	95% KM (BCA) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	0.35	0.38	1.4	3.78E-01	mg/kg	% UCL	95% KM (t) UCL
	132-64-9	DIBENZOFURAN	mg/kg	2.50	2.82	53	2.82E+00	mg/kg	% UCL	95% KM (BCA) UCL
	118-74-1	HEXACHLORO BENZENE	mg/kg	1.87	0.98	11	9.83E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	0.91	1.03	4.1	1.03E+00	mg/kg	% UCL	95% KM (BCA) UCL
	91-20-3	NAPHTHALENE	mg/kg	12.44	27.46	300	2.75E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	3.91	11.96	120	1.20E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL

TABLE 3.2
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 2 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, and DSA #2	VOCs									
	87-61-6	1,2,3TRICHLOROBENZENE	mg/kg	4.18	NA	8.3	8.30E+00	mg/kg	Max	Insufficient Data*
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	8.97	4.67	53	4.67E+00	mg/kg	% UCL	95% KM (t) UCL
	95-50-1	1,2-DICHLOROBENZENE	mg/kg	10.59	20.26	210	2.03E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	15.92	34.82	350	3.48E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	0.55	0.69	4.2	6.85E-01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.002	NA	0.002	2.00E-03	mg/kg	Max	Insufficient Data*

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95% UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Chebyshev (Mean, Sd) UCL = $(1-\alpha)100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Chebyshev (MVUE) UCL = $(1-\alpha)100\%$ UCL of the Mean of a Lognormal Population Based Upon the Chebyshev Theorem (Using the MVUE of the Mean and its Standard Error - parametric).

Gamma UCL = Computation of UCL of the mean of a Gamma, $G(k,\theta)$ distribution (parametric).

H-UCL = $(1-\alpha)100\%$ UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.3a
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 3 - SURFACE SEDIMENT
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface Sediment
(0 - 1 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
I-690 Drainage Ditch	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	5.243	9.198	19.8	9.20E+00	ng/kg	% UCL	95% Approximate Gamma UCL
	METALS									
	7440-38-2	ARSENIC	mg/kg	3.483	3.816	5.2	3.82E+00	mg/kg	% UCL	95% KM (t) UCL
	7440-47-3	CHROMIUM	mg/kg	75.48	156	534	1.56E+02	mg/kg	% UCL	95% Chebyshev (MVUE) UCL
	7439-89-6	IRON	mg/kg	9388	14322	21300	1.43E+04	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-96-5	MANGANESE	mg/kg	234.3	287	365	2.87E+02	mg/kg	% UCL	95% Student's-t UCL
	7439-97-6	MERCURY	mg/kg	0.406	1.129	0.75	7.50E-01	mg/kg	Max	Max
	7440-62-2	VANADIUM	mg/kg	15.2	16.22	24	1.62E+01	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	16.13	16	45	1.60E+01	mg/kg	% UCL	95% KM (t) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	3.48	3.75	11	3.75E+00	mg/kg	% UCL	95% KM (t) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	1.10	1.37	2	1.37E+00	mg/kg	% UCL	95% KM (t) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	1.11	1.35	2	1.35E+00	mg/kg	% UCL	95% KM (t) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	1.23	1.47	2.1	1.47E+00	mg/kg	% UCL	95% KM (t) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	1.02	1.21	1.8	1.21E+00	mg/kg	% UCL	95% KM (t) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	0.082	0.11	0.096	9.60E-02	mg/kg	Max	Max
	132-64-9	DIBENZOFURAN	mg/kg	3.36	3.79	13	3.79E+00	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	0.81	0.96	1.5	9.56E-01	mg/kg	% UCL	95% KM (t) UCL
	91-20-3	NAPHTHALENE	mg/kg	63.94	60.9	150	6.09E+01	mg/kg	% UCL	95% KM (t) UCL
	85-01-8	PHENANTHRENE	mg/kg	4.90	10.44	18	1.04E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.011	NA	0.017	1.70E-02	mg/kg	Max	Insufficient Data*
	VOCs									
	71-43-2	BENZENE	mg/kg	0.824	0.872	2	8.72E-01	mg/kg	% UCL	95% KM (t) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Chebyshev (MVUE) UCL = (1-α) 100% UCL of the Mean of a Lognormal Population Based Upon the Chebyshev Theorem (Using the MVUE of the Mean and its Standard Error - parametric)

Gamma UCL = Computation of UCL of the mean of a Gamma, G(k,θ) distribution (parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.3b
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 3 - SURFACE WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
I-690 Drainage Ditch	METALS									
	7440-47-3	CHROMIUM	mg/L	0.012	0.012	0.016	1.23E-02	mg/L	% UCL	95% KM (t) UCL
	7439-89-6	IRON	mg/L	1.20	1.67	3.13	1.67E+00	mg/L	% UCL	95% KM (t) UCL
	7439-92-1	LEAD	mg/L	0.012	0.016	0.026	1.18E-02	mg/L	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/L	0.109	0.109	0.152	1.09E-01	mg/L	% UCL	95% KM (t) UCL
	7439-97-6	MERCURY	mg/L	0.00019	0.00023	0.00048	2.29E-04	mg/L	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/L	0.003	0.0044	0.0037	3.70E-03	mg/L	Max	Max
	7440-66-6	ZINC	mg/L	1.05	1.05	1.41	1.05E+00	mg/L	% UCL	95% KM (t) UCL
	SVOCs									
	91-57-6	2-METHYLNAPHTHALENE	ug/L	67.8	93.94	160	9.39E+01	ug/L	% UCL	95% KM (t) UCL
	34METPH	3&4-METHYLPHENOL	ug/L	119.2	151.6	210	1.52E+02	ug/L	% UCL	95% KM (t) UCL
	208-96-8	ACENAPHTHYLENE	ug/L	7.43	9.78	11	9.78E+00	ug/L	% UCL	95% KM (t) UCL
	86-74-8	CARBAZOLE	ug/L	19	23.13	30	2.31E+01	ug/L	% UCL	95% KM (t) UCL
	132-64-9	DIBENZOFURAN	ug/L	16.66	22.17	35	2.22E+01	ug/L	% UCL	95% KM (t) UCL
	86-73-7	FLUORENE	ug/L	12.72	17.25	27	1.73E+01	ug/L	% UCL	95% KM (t) UCL
	91-20-3	NAPHTHALENE	ug/L	615	799.9	1400	8.00E+02	ug/L	% UCL	95% KM (t) UCL
	85-01-8	PHENANTHRENE	ug/L	13.78	18.26	27	1.83E+01	ug/L	% UCL	95% KM (t) UCL
	VOCs									
	95-63-6	1,2,4-TRIMETHYLBENZENE	ug/L	41.5	NA	67	6.70E+01	ug/L	Max	Insufficient Data*
	108-67-8	1,3,5-TRIMETHYLBENZENE	ug/L	16	NA	26	2.60E+01	ug/L	Max	Insufficient Data*
	71-43-2	BENZENE	ug/L	50.32	73.75	130	7.38E+01	ug/L	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	ug/L	112.4	159.5	270	1.60E+02	ug/L	% UCL	95% KM (t) UCL
	1330-20-7	XYLENES, TOTAL	ug/L	188.5	NA	300	3.00E+02	ug/L	Max	Insufficient Data*

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

TABLE 3.4
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 4 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Railroad Area	METALS									
	7429-90-5	ALUMINUM	mg/kg	8421	9390	13600	9.39E+03	mg/kg	% UCL	95% Student's-t UCL
	7440-38-2	ARSENIC	mg/kg	8.14	13.81	22.7	1.38E+01	mg/kg	% UCL	95% Chebyshev (Mean, Sd) UCL
	7440-39-3	BARIUM	mg/kg	96.79	287.2	879	2.87E+02	mg/kg	% UCL	95% Chebyshev (Mean, Sd) UCL
	7440-47-3	CHROMIUM	mg/kg	16.93	19.39	33.2	1.94E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-89-6	IRON	mg/kg	16021	17257	20900	1.73E+04	mg/kg	% UCL	95% Student's-t UCL
	7439-92-1	LEAD	mg/kg	72.71	503.9	849	7.27E+01	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	356.7	386.9	522	3.87E+02	mg/kg	% UCL	95% Student's-t UCL
	7439-97-6	MERCURY	mg/kg	0.36	1.005	2	1.01E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-62-2	VANADIUM	mg/kg	18.45	20.68	30.9	2.07E+01	mg/kg	% UCL	95% Student's-t UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.003	NA	0.003	3.00E-03	mg/kg	Max	Insufficient Data*
		HIGHLY CHLORINATED PCBs ^b	mg/kg	0.020	0.058	0.069	5.82E-02	mg/kg	% UCL	95% Approximate Gamma UCL
		TOTAL PCBs ^c	mg/kg	0.020	0.058	0.069	5.82E-02	mg/kg	% UCL	95% Approximate Gamma UCL
	PESTICIDES									
	60-57-1	DIELDRIN	mg/kg	0.0078	NA	0.05	5.00E-02	mg/kg	Max	Insufficient Data*
	SVOCs									
	208-96-8	ACENAPHTHYLENE	mg/kg	0.13	0.17	0.2	1.73E-01	mg/kg	% UCL	95% KM (t) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	0.31	0.41	1.1	4.08E-01	mg/kg	% UCL	95% KM (BCA) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	0.32	0.40	0.95	3.97E-01	mg/kg	% UCL	95% KM (BCA) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	0.39	0.50	1.7	5.02E-01	mg/kg	% UCL	95% KM (BCA) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	0.19	0.24	0.48	2.41E-01	mg/kg	% UCL	95% KM (t) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	0.27	0.32	0.81	3.17E-01	mg/kg	% UCL	95% KM (Percentile Bootstrap) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	0.11	0.14	0.19	1.37E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	0.21	0.25	0.47	2.49E-01	mg/kg	% UCL	95% KM (t) UCL
	85-01-8	PHENANTHRENE	mg/kg	0.37	0.47	1.5	4.73E-01	mg/kg	% UCL	95% KM (BCA) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.005	0.0041	0.008	4.08E-03	mg/kg	% UCL	95% KM (t) UCL

TABLE 3.4
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 4 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
	VOCs									
	71-43-2	BENZENE	mg/kg	0.001	NA	0.001	1.00E-03	mg/kg	Max	Insufficient Data*

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Chebyshev (Mean, Sd) UCL = $(1-\alpha) 100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Gamma UCL = Computation of UCL of the mean of a Gamma, $G(k,\theta)$ distribution (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (Percentile Bootstrap) UCL - $(1-\alpha) 100\%$ UCL of the Mean Based Upon Simple Percentile Bootstrap Method (non-parametric)

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.5
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 5 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Penn-Can Property	METALS									
	7429-90-5	ALUMINUM	mg/kg	5804	6653	9220	6.65E+03	mg/kg	% UCL	95% Student's-t UCL
	7440-36-0	ANTIMONY	mg/kg	1.00	1.75	4.9	1.75E+00	mg/kg	% UCL	95% KM (t) UCL
	7440-38-2	ARSENIC	mg/kg	11.29	15.11	34.4	1.51E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-47-3	CHROMIUM	mg/kg	19.15	35.46	93.4	3.55E+01	mg/kg	% UCL	95% Chebyshev (Mean, Sd) UCL
	7439-89-6	IRON	mg/kg	15909	18281	30000	1.83E+04	mg/kg	% UCL	95% Student's-t UCL
	7439-92-1	LEAD	mg/kg	72.77	97.42	263	7.28E+01	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	284.7	314.3	402	3.14E+02	mg/kg	% UCL	95% Student's-t UCL
	7439-97-6	MERCURY	mg/kg	1.35	2.39	7.9	2.39E+00	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-28-0	THALLIUM	mg/kg	1	NA	1	1.00E+00	mg/kg	Max	Insufficient Data*
	7440-62-2	VANADIUM	mg/kg	20.16	23.14	44.1	2.31E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	PCBs									
		HIGHLY CHLORINATED PCBs ^a	mg/kg	0.64	5.98	6	5.98E+00	mg/kg	% UCL	99% Chebyshev (Mean, Sd) UCL
		TOTAL PCBs ^b	mg/kg	0.64	5.98	6	5.98E+00	mg/kg	% UCL	99% Chebyshev (Mean, Sd) UCL
	PESTICIDES									
	1031-07-8	ENDOSULFAN SULFATE	mg/kg	0.13	NA	0.13	1.30E-01	mg/kg	Max	Insufficient Data*
	7421-93-4	ENDRIN ALDEHYDE	mg/kg	0.098	0.067	0.15	6.65E-02	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	34METPH	3&4-METHYLPHENOL	mg/kg	0.044	NA	0.044	4.40E-02	mg/kg	Max	Insufficient Data*
	208-96-8	ACENAPHTHYLENE	mg/kg	7.22	14.37	30	1.44E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	20.8	35.77	120	3.58E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	20.31	34.96	100	3.50E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	18.62	30.27	81	3.03E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	13.41	29.85	69	2.99E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	16.47	38.02	94	3.80E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	20.56	34.65	110	3.47E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	4.77	9.95	22	9.95E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	3.025	6.16	19	6.16E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	mg/kg	43.59	76.77	310	7.68E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	12.64	28.11	64	2.81E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	4.14	7.25	23	7.25E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	24.66	43.31	210	4.33E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.0043	0.0038	0.009	3.78E-03	mg/kg	% UCL	95% KM (t) UCL

TABLE 3.5
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 5 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
	VOCs									
	71-43-2	BENZENE	mg/kg	0.0103	0.00808	0.052	8.08E-03	mg/kg	% UCL	95% KM (t) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

c = Max - maximum detected concentration; %UCL - % upper confidence limit. KM - Kaplan-Meier method.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

95% H-UCL = (1-α)100% UCL of the mean based upon H-statistic (H-UCL) (parametric).

Chebyshev (Mean, Sd) UCL = (1-α)100% UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Gamma UCL = Computation of UCL of the mean of a Gamma, G(k,θ) distribution (parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.6a
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 6 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	89.16	516.4	575.5	5.16E+02	ng/kg	% UCL	99% Chebyshev (Mean, Sd) UCL
	METALS									
	7429-90-5	ALUMINUM	mg/kg	6548	7208	24400	7.21E+03	mg/kg	% UCL	95% Student's-t UCL
	7440-38-2	ARSENIC	mg/kg	7.62	8.36	21.4	8.36E+00	mg/kg	% UCL	95% KM (BCA) UCL
	7440-39-3	BARIUM	mg/kg	344.8	366.3	4880	3.66E+02	mg/kg	% UCL	95% H-UCL
	7440-43-9	CADMIUM	mg/kg	20.76	35.72	110	3.57E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	94.13	117.3	391	1.17E+02	mg/kg	% UCL	95% H-UCL
	7440-50-8	COPPER	mg/kg	162.6	246.2	744	2.46E+02	mg/kg	% UCL	95% Chebyshev (Mean, Sd) UCL
	7439-89-6	IRON	mg/kg	12027	12975	24400	1.30E+04	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-92-1	LEAD	mg/kg	373.8	670.7	2320	3.74E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	276	298.6	722	2.99E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-97-6	MERCURY	mg/kg	7.25	11.7	64.3	1.17E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-22-4	SILVER	mg/kg	22.25	15.09	91.9	1.51E+01	mg/kg	% UCL	95% KM (t) UCL
	7440-28-0	THALLIUM	mg/kg	0.97	0.80	2.3	7.99E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	20.79	21.64	49.1	2.16E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.31	0.72	2	7.21E-01	mg/kg	% UCL	95% Chebyshev (MVUE) UCL
		HIGHLY CHLORINATED PCBs ^b	mg/kg	1.18	1.53	6	1.53E+00	mg/kg	% UCL	95% Approximate Gamma UCL
		TOTAL PCBs ^c	mg/kg	1.18	1.53	6	1.53E+00	mg/kg	% UCL	95% Approximate Gamma UCL
	PESDTICIDES									
	60-57-1	DIELDRIN	mg/kg	0.16	0.11	0.2	1.14E-01	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	3.93	12.73	130	1.27E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	2.13	3.93	37	3.93E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	4.51	7.53	32	7.53E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	4.69	9.04	32	9.04E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	4.27	6.60	27	6.60E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	3.35	5.25	24	5.25E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	3.45	5.68	25	5.68E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	4.84	8.02	34	8.02E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	1.22	1.64	6.2	1.64E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	2.33	4.12	53	4.12E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	118-74-1	HEXACHLOROBENZENE	mg/kg	1.85	0.95	11	9.54E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	2.88	4.58	20	4.58E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	10.48	23.56	300	2.36E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	8.05	19.16	120	1.92E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

TABLE 3.6a
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 6 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1	VOCs									
	87-61-6	1,2,3-TRICHLOROBENZENE	mg/kg	4.18	3.88	8.3	3.88E+00	mg/kg	% UCL	99% KM (Chebyshev) UCL
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	7.87	4.22	53	4.22E+00	mg/kg	% UCL	95% KM (t) UCL
	95-50-1	1,2-DICHLOROBENZENE	mg/kg	9.98	8.38	210	8.38E+00	mg/kg	% UCL	95% KM (t) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	14.66	30.32	350	3.03E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	0.43	0.53	4.2	5.34E-01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.02	NA	0.38	3.80E-01	mg/kg	Max	Insufficient Data*
	OTHER									
	112-40-3	DODECANE	ma/ka	845	813.5	1100	8.14E+02	ma/ka	% UCL	95% KM (t) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254,-1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Chebyshev (Mean, Sd) UCL = $(1-\alpha)100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Chebyshev (MVUE) UCL = $(1-\alpha)100\%$ UCL of the Mean of a Lognormal Population Based Upon the Chebyshev Theorem (Using the MVUE of the Mean and its Standard Error - parametric).

Gamma UCL = Computation of UCL of the mean of a Gamma, $G(k,\theta)$ distribution (parametric).

H-UCL = $(1-\alpha)100\%$ UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.6b
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 6 - SURFACE SEDIMENT
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Sediment
Exposure Medium: Sediment (0-1 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	46.42	111.6	458.6	1.12E+02	ng/kg	% UCL	95% Chebyshev (MVUE) UCL
	Metals									
	7440-38-2	ARSENIC	mg/kg	6.95	10.29	22	1.03E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-43-9	CADMIUM	mg/kg	1.74	4.67	19.2	4.67E+00	mg/kg	% UCL	99% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	39.59	67.73	211	6.77E+01	mg/kg	% UCL	Use 95% Chebyshev (Mean, Sd) UCL
	7439-89-6	IRON	mg/kg	10419	11447	21200	1.14E+04	mg/kg	% UCL	Use 95% Student's-t UCL
	7439-92-1	LEAD	mg/kg	117.6	146.8	479	1.18E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	232.2	246.7	366	2.47E+02	mg/kg	% UCL	Use 95% Student's-t UCL
	7439-97-6	MERCURY	mg/kg	3.36	7.85	52	7.85E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-28-0	THALLIUM	mg/kg	1.97	0.75	4.9	7.54E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	13.32	14.58	27.1	1.46E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		HIGHLY CHLORINATED PCBs ^a	mg/kg	0.58	0.77	4.7	7.73E-01	mg/kg	% UCL	Use 95% H-UCL
		TOTAL PCBs ^b	mg/kg	0.58	0.77	4.7	7.73E-01	mg/kg	% UCL	Use 95% H-UCL
	Pesticides									
	60-57-1	DIELDRIN	mg/kg	0.04	0.02	0.069	1.93E-02	mg/kg	% UCL	95% KM (t) UCL
	53494-70-5	ENDRIN KETONE	mg/kg	0.09	0.05	0.15	5.37E-02	mg/kg	% UCL	95% KM (t) UCL
	1024-57-3	HEPTACHLOR EPOXIDE	mg/kg	0.02	0.01	0.063	9.94E-03	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	15.36	35.83	210	3.58E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	4.03	5.57	51	5.57E+00	mg/kg	% UCL	95% KM (BCA) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	23.84	151.5	460	1.52E+02	mg/kg	% UCL	99% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	24.78	106.3	480	1.06E+02	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	35.13	238.5	720	2.39E+02	mg/kg	% UCL	99% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	15.86	66.30	280	6.63E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	14.71	58.58	270	5.86E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	mg/kg	17.02	61.77	290	6.18E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	86-74-8	CARBAZOLE	mg/kg	6.21	19.81	93	1.98E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	32.95	217.6	650	2.18E+02	mg/kg	% UCL	99% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	6.44	12.86	72	1.29E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	16.11	28.59	100	2.86E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	mg/kg	54.17	131	990	1.31E+02	mg/kg	% UCL	Use 95% Chebyshev (MVUE) UCL
	118-74-1	HEXACHLOROBENZENE	mg/kg	0.11	0.13	0.53	1.25E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	13.73	54.43	230	5.44E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	33.48	69.03	240	6.90E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	52.76	248.80	780	2.49E+02	mg/kg	% UCL	Use 97.5% Chebyshev (MVUE) UCL
	129-00-0	PYRENE	mg/kg	66.03	440.50	1300	4.41E+02	mg/kg	% UCL	99% KM (Chebyshev) UCL

TABLE 3.6b
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 6 - SURFACE SEDIMENT
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Sediment
Exposure Medium: Sediment (0-1 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1	VOCs									
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	2.22	0.83	8.1	8.31E-01	mg/kg	% UCL	95% KM (t) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	16.33	52.70	160	5.27E+01	mg/kg	% UCL	99% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	2.12	8.25	29	8.25E+00	mg/kg	% UCL	99% KM (Chebyshev) UCL
	108-90-7	CHLOROBENZENE	mg/kg	8.15	48.32	240	4.83E+01	mg/kg	% UCL	99% KM (Chebyshev) UCL
	75-09-2	METHYLENE CHLORIDE	mg/kg	4.00	0.73	9.5	7.32E-01	mg/kg	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	mg/kg	4.56	20.91	88	2.09E+01	mg/kg	% UCL	99% KM (Chebyshev) UCL
	1330-20-7	XYLENES, TOTAL	mg/kg	11.48	67.72	314	6.77E+01	mg/kg	% UCL	99% KM (Chebyshev) UCL

Footnotes:

Chebyshev UCL or Student's-t UCL for nonparametric distribution based on ProUCL (V. 4.0) recommendation; Gamma UCL for gamma distribution; Nonparametric Bootstrap UCLs introduce some standard error and may not be reproduced precisely.

a = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

c = Max - maximum detected concentration; %UCL - % upper confidence limit.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95% UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

Chebyshev (Mean, Sd) UCL = $(1-\alpha)100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Chebyshev (MVUE) UCL = $(1-\alpha)100\%$ UCL of the Mean of a Lognormal Population Based Upon the Chebyshev Theorem (Using the MVUE of the Mean and its Standard Error - parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

95% H-UCL = $(1-\alpha)100\%$ UCL of the mean based upon H-statistic (H-UCL) (parametric).

TABLE 3.6c
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 6 - SURFACE WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1	METALS									
	7440-36-0	ANTIMONY	mg/L	0.0020	0.0022	0.0028	2.15E-03	mg/L	% UCL	95% KM (t) UCL
	7440-38-2	ARSENIC	mg/L	0.0018	NA	0.0018	1.80E-03	mg/L	Max	Insufficient Data*
	7440-47-3	CHROMIUM	mg/L	0.0050	0.0055	0.006	5.47E-03	mg/L	% UCL	95% KM (t) UCL
	7439-89-6	IRON	mg/L	1.03	4.95	12.28	4.95E+00	mg/L	% UCL	97.5% KM (Chebyshev) UCL
	7439-92-1	LEAD	mg/L	0.012	0.0084	0.028	1.23E-02	mg/L	Avg	USEPA Guidance for Lead Exposure
	7439-97-6	MERCURY	mg/L	0.00015	0.00011	0.00047	1.12E-04	mg/L	% UCL	95% KM (t) UCL
	7440-28-0	THALLIUM	mg/L	0.0038	NA	0.0038	3.80E-03	mg/L	Max	Insufficient Data*
	SVOCs									
	105-67-9	2,4-DIMETHYLPHENOL	ug/L	101	125.1	190	1.25E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	91-57-6	2-METHYLNAPHTHALENE	ug/L	111.7	104.1	300	1.04E+02	ug/L	% UCL	95% KM (t) UCL
	34METPH	3&4-METHYLPHENOL	ug/L	230	201.3	280	2.01E+02	ug/L	% UCL	95% KM (t) UCL
	83-32-9	ACENAPHTHENE	ug/L	38.6	33.48	49	3.35E+01	ug/L	% UCL	95% KM (t) UCL
	208-96-8	ACENAPHTHYLENE	ug/L	37.78	19.97	55	2.00E+01	ug/L	% UCL	95% KM (t) UCL
	56-55-3	BENZ(A)ANTHRACENE	ug/L	2.5	5.12	4	4.00E+00	ug/L	Max	Max
	50-32-8	BENZO(A)PYRENE	ug/L	1.6	2.30	2	2.00E+00	ug/L	Max	Max
	205-99-2	BENZO(B)FLUORANTHENE	ug/L	2.1	3.67	3	3.00E+00	ug/L	Max	Max
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	ug/L	4.5	NA	7.8	7.80E+00	ug/L	Max	Insufficient Data*
	86-74-8	CARBAZOLE	ug/L	35.85	19.68	56	1.97E+01	ug/L	% UCL	95% KM (t) UCL
	218-01-9	CHRYSENE	ug/L	2.23	3.78	4	3.78E+00	ug/L	% UCL	95% KM (t) UCL
	132-64-9	DIBENZOFURAN	ug/L	49.05	25.89	73	2.59E+01	ug/L	% UCL	95% KM (t) UCL
	86-73-7	FLUORENE	ug/L	27.36	18.55	42	1.86E+01	ug/L	% UCL	95% KM (t) UCL
	91-20-3	NAPHTHALENE	ug/L	569.5	1724	2200	1.72E+03	ug/L	% UCL	99% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	ug/L	41.27	30.18	83	3.02E+01	ug/L	% UCL	95% KM (t) UCL
	129-00-0	PYRENE	ug/L	9.3	8.126	28	8.13E+00	ug/L	% UCL	95% KM (t) UCL

TABLE 3.6c
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 6 - SURFACE WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Water
Exposure Medium: Surface Water

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1	VOCs									
	106-46-7	1,4-DICHLOROBENZENE	ug/L	8	NA	8	8.00E+00	ug/L	Max	Insufficient Data*
	71-43-2	BENZENE	ug/L	74.61	73.73	200	7.37E+01	ug/L	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	ug/L	82.39	389.7	410	3.90E+02	ug/L	% UCL	99% KM (Chebyshev) UCL
	1330-20-7	XYLENES, TOTAL	ug/L	68.33	8700	470	4.70E+02	ug/L	Max	Insufficient Data*
	OTHER									
	25321-22-6	DICHLOROBENZENES	ug/L	9.68	NA	9.68	9.68E+00	ug/L	Max	Insufficient Data*

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Max - maximum detected concentration; %UCL - % upper confidence limit. KM - Kaplan-Meier method.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

TABLE 3.6d
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 6 - FISH TISSUE
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Fish Tissue
Exposure Medium: Fish Tissue

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Fish Fillet Tissue from Onodaga Lake	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	mg/kg	0.000010	0.000020	0.000046	2.00E-05	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	METALS									
	7440-36-0	ANTIMONY	mg/kg	0.56	0.99	2.1	9.90E-01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-38-2	ARSENIC	mg/kg	0.33	0.080	1.05	8.00E-02	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-47-3	CHROMIUM	mg/kg	0.49	0.57	0.73	5.70E-01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	57-12-5	CYANIDE	mg/kg	1.73	5.69	14.3	5.70E+00	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7439-96-5	MANGANESE	mg/kg	0.99	3.23	5.51	3.20E+00	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	22967-92-6	MERCURY (AS METHYLMERCURY)	mg/kg	1.05	1.08	5.07	1.10E+00	mg/kg	% UCL	95% D'Agostino (Y) UCL
	7782-49-2	SELENIUM	mg/kg	1.08	1.47	2.2	1.50E+00	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-62-2	VANADIUM	mg/kg	0.48	0.63	0.97	6.30E-01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	7440-66-6	ZINC	mg/kg	30.5	43.7	73.8	4.40E+01	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.27	0.48	1.98	4.80E-01	mg/kg	% UCL	95% D'Agostino (Y) UCL
		HIGHLY CHLORINATED PCBs ^b	mg/kg	0.42	0.58	1.92	5.80E-01	mg/kg	% UCL	95% D'Agostino (Y) UCL
		TOTAL PCBs ^c	mg/kg	0.67	0.91	3.9	9.10E-01	mg/kg	% UCL	95% D'Agostino (Y) UCL
	PESTICIDES									
	3424826	2,4'-DDE	mg/kg	0.0038	0.0041	0.024	4.10E-03	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	72-54-8	4,4'-DDD	mg/kg	0.011	0.013	0.17	1.30E-02	mg/kg	% UCL	95% D'Agostino (Y) UCL
	72-55-9	4,4'-DDE	mg/kg	0.029	0.034	0.4	3.40E-02	mg/kg	% UCL	95% D'Agostino (Y) UCL
	50-29-3	4,4'-DDT	mg/kg	0.0081	0.0095	0.082	9.50E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	309-00-2	ALDRIN	mg/kg	0.0023	0.0025	0.003	2.50E-03	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	319-86-8	DELTA-BHC	mg/kg	0.0023	0.0025	0.0028	2.50E-03	mg/kg	% UCL	95% Shapiro-Wilk (W) UCL
	60-57-1	DIELDRIN	mg/kg	0.0038	0.0038	0.042	3.80E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	1024-57-3	HEPTACHLOR EPOXIDE	mg/kg	0.0036	0.0041	0.01	4.10E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	12789-03-6	CHLORDANE, TOTAL	mg/kg	0.0084	0.0097	0.061	9.70E-03	mg/kg	% UCL	95% D'Agostino (Y) UCL
	SVOCs									
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	mg/kg	0.7	NA	2.3	2.30E+00	mg/kg	Max	Insufficient Data*
	118-74-1	HEXACHLOROBENZENE	mg/kg	0.012	0.013	0.17	1.30E-02	mg/kg	% UCL	95% D'Agostino (Y) UCL

Footnotes:

Table modified from Table 3.1 Onondaga Lake Human Health Risk Assessment prepared by TAMS Consultants, Inc. & YEC, Inc.

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

* = Insufficient data to calculate a UCL, according to the Onondaga Lake Human Health Risk Assessment. In these cases, the EPC is obtained by using the maximum detected concentration.

D'Agostino (Y) UCL = Distributional fit for sample sets containing greater than 50 samples.

Shapiro-Wilk (W) UCL = Distributional fit for sample sets containing less than 50 samples.

TABLE 3.7
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 7 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Penn-Can Property, Lakeshore Area, DSA #1, DSA #2, AOS #1, and AOS #2	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	89.16	516.4	575.5	5.16E+02	ng/kg	% UCL	99% Chebyshev (Mean, Sd) UCL
	METALS									
	7429-90-5	ALUMINUM	mg/kg	6342	6853	24400	6.85E+03	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-36-0	ANTIMONY	mg/kg	0.72	0.75	4.9	7.52E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-38-2	ARSENIC	mg/kg	8.33	9.24	34.4	9.24E+00	mg/kg	% UCL	95% KM (BCA) UCL
	7440-39-3	BARIUM	mg/kg	283.9	301.4	4880	3.01E+02	mg/kg	% UCL	95% H-UCL
	7440-43-9	CADMIUM	mg/kg	18.8	28.04	110	2.80E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	76.86	95.74	391	9.57E+01	mg/kg	% UCL	95% H-UCL
	7440-50-8	COPPER	mg/kg	132.8	230.8	744	2.31E+02	mg/kg	% UCL	97.5% Chebyshev (Mean, Sd) UCL
	7439-89-6	IRON	mg/kg	12792	13715	30000	1.37E+04	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-92-1	LEAD	mg/kg	304.5	461.2	2320	3.05E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	277.7	296.3	722	2.96E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-97-6	MERCURY	mg/kg	5.88	9.51	64.3	9.51E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-22-4	SILVER	mg/kg	21.35	11.74	91.9	1.17E+01	mg/kg	% UCL	95% KM (t) UCL
	7440-28-0	THALLIUM	mg/kg	0.97	0.74	2.3	7.38E-01	mg/kg	% UCL	95% KM (t) UCL
	7440-62-2	VANADIUM	mg/kg	20.56	21.37	49.1	2.14E+01	mg/kg	% UCL	95% KM (t) UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.31	0.72	2	7.21E-01	mg/kg	% UCL	95% Chebyshev (MVUE) UCL
		HIGHLY CHLORINATED PCBs ^b	mg/kg	1.10	1.43	6	1.43E+00	mg/kg	% UCL	95% Approximate Gamma UCL
		TOTAL PCBs ^c	mg/kg	1.10	1.43	6	1.43E+00	mg/kg	% UCL	95% Approximate Gamma UCL
	PESTICIDES									
	60-57-1	DIELDRIN	mg/kg	0.16	0.11	0.2	1.13E-01	mg/kg	% UCL	95% KM (t) UCL
	SVOCs									
	91-57-6	2-METHYLNAPHTHALENE	mg/kg	3.67	10.3	130	1.03E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	mg/kg	3.35	6.42	37	6.42E+00	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	8.33	17.81	120	1.78E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	8.36	17.35	100	1.74E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	7.59	12.61	81	1.26E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	5.75	11.47	69	1.15E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	6.46	13.99	94	1.40E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	8.44	17.51	110	1.75E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	2.29	3.22	22	3.22E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	132-64-9	DIBENZOFURAN	mg/kg	2.57	3.91	53	3.91E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	mg/kg	16.66	38.55	310	3.86E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	118-74-1	HEXACHLOROBENZENE	mg/kg	1.85	0.82	11	8.15E-01	mg/kg	% UCL	95% KM (t) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	5.21	10.61	64	1.06E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	8.81	18.71	300	1.87E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	11.93	26.54	210	2.65E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL

TABLE 3.7
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 7 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
Penn-Can Property, Lakeshore Area, DSA #1, DSA #2, AOS #1, and AOS #2	VOCs									
	87-61-6	1,2,3-TRICHLOROBENZENE	mg/kg	4.18	2.92	8.3	2.92E+00	mg/kg	% UCL	99% KM (Chebyshev) UCL
	120-82-1	1,2,4-TRICHLOROBENZENE	mg/kg	7.87	3.26	53	3.26E+00	mg/kg	% UCL	95% KM (t) UCL
	95-50-1	1,2-DICHLOROBENZENE	mg/kg	9.98	6.46	210	6.46E+00	mg/kg	% UCL	95% KM (t) UCL
	106-46-7	1,4-DICHLOROBENZENE	mg/kg	14.08	23.35	350	2.34E+01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	mg/kg	0.34	0.41	4.2	4.14E-01	mg/kg	% UCL	97.5% KM (Chebyshev) UCL
	99-87-6	P-ISOPROPYLTOLUENE	mg/kg	0.0037	0.0025	0.009	2.52E-03	mg/kg	% UCL	95% KM (t) UCL
	OTHER									
	112-40-3	DODECANE	mg/kg	845	813.5	1100	8.14E+02	mg/kg	% UCL	95% KM (t) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Chebyshev (Mean, Sd) UCL = $(1-\alpha)100\%$ UCL of the mean based upon the chebyshev theorem (using the sample mean and sample standard deviation - non-parametric).

Chebyshev (MVUE) UCL = $(1-\alpha)100\%$ UCL of the Mean of a Lognormal Population Based Upon the Chebyshev Theorem (Using the MVUE of the Mean and its Standard Error - parametric).

Gamma UCL = Computation of UCL of the mean of a Gamma, $G(k,\theta)$ distribution (parametric).

H-UCL = $(1-\alpha)100\%$ UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

TABLE 3.8
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 8 - SITE WIDE ALL GROUND WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Water
Exposure Medium: Ground Water (All Depths)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration				
							Value	Units	Statistic ^c	Rationale	
Site Wide Ground Water	METALS										
	7429-90-5	ALUMINUM	mg/L	10.21	24.63	291	2.46E+01	mg/L	% UCL	97.5% KM (Chebyshev) UCL	
	7440-36-0	ANTIMONY	mg/L	0.0029	0.0022	0.0058	2.19E-03	mg/L	% UCL	95% KM (t) UCL	
	7440-38-2	ARSENIC	mg/L	0.020	0.0094	0.102	9.39E-03	mg/L	% UCL	95% KM (t) UCL	
	7440-39-3	BARIUM	mg/L	0.719	1.485	20.3	1.49E+00	mg/L	% UCL	95% KM (Chebyshev) UCL	
	7440-41-7	BERYLLIUM	mg/L	0.0011	0.00080	0.0073	8.03E-04	mg/L	% UCL	95% KM (BCA) UCL	
	7440-43-9	CADMIUM	mg/L	0.0075	0.0020	0.027	1.98E-03	mg/L	% UCL	95% KM (t) UCL	
	7440-47-3	CHROMIUM	mg/L	0.063	0.070	0.86	7.03E-02	mg/L	% UCL	95% KM (Chebyshev) UCL	
	7440-48-4	COBALT	mg/L	0.041	0.012	0.13	1.16E-02	mg/L	% UCL	95% KM (t) UCL	
	7440-50-8	COPPER	mg/L	0.090	0.093	1.23	9.29E-02	mg/L	% UCL	95% KM (Chebyshev) UCL	
	57-12-5	CYANIDE	mg/L	0.054	0.030	0.53	3.01E-02	mg/L	% UCL	95% KM (t) UCL	
	7439-89-6	IRON	mg/L	16.4	39.4	446	3.94E+01	mg/L	% UCL	97.5% KM (Chebyshev) UCL	
	7439-92-1	LEAD	mg/L	0.0673	0.064	1.7	6.73E-02	mg/L	Avg	USEPA Guidance for Lead Exposure	
	7439-96-5	MANGANESE	mg/L	1.054	1.83	16.1	1.83E+00	mg/L	% UCL	97.5% KM (Chebyshev) UCL	
	7439-97-6	MERCURY	mg/L	0.00265	0.00213	0.0308	2.13E-03	mg/L	% UCL	95% KM (Chebyshev) UCL	
	7440-02-0	NICKEL	mg/L	0.045	0.049	0.39	4.87E-02	mg/L	% UCL	95% KM (Chebyshev) UCL	
	7782-49-2	SELENIUM	mg/L	0.0059	0.0036	0.02	3.63E-03	mg/L	% UCL	95% KM (t) UCL	
	7440-22-4	SILVER	mg/L	0.0057	0.0021	0.025	2.14E-03	mg/L	% UCL	95% KM (t) UCL	
	7440-28-0	THALLIUM	mg/L	0.031	0.0069	0.088	6.89E-03	mg/L	% UCL	95% KM (t) UCL	
	7440-62-2	VANADIUM	mg/L	0.041	0.044	0.57	4.40E-02	mg/L	% UCL	95% KM (Chebyshev) UCL	
	7440-66-6	ZINC	mg/L	0.13	0.097	1.9	9.73E-02	mg/L	% UCL	95% KM (BCA) UCL	
	PCBs										
			HIGHLY CHLORINATED PCBs ^a	ug/L	0.07	NA	0.07	7.00E-02	ug/L	Max	Insufficient Data*
			TOTAL PCBs ^b	ug/L	0.07	NA	0.07	7.00E-02	ug/L	Max	Insufficient Data*
	PESTICIDES										
		72-54-8	4,4'-DDD	ug/L	0.51	0.089	2.2	8.87E-02	ug/L	% UCL	95% KM (t) UCL
		50-29-3	4,4'-DDT	ug/L	6.75	1.096	20	1.10E+00	ug/L	% UCL	97.5% KM (Chebyshev) UCL
		309-00-2	ALDRIN	ug/L	0.11	NA	0.17	3.41E-02	ug/L	% UCL	95% KM (t) UCL
		319-84-6	ALPHA-BHC	ug/L	0.19	NA	0.19	1.90E-01	ug/L	Max	Insufficient Data*
		33213-65-9	ENDOSULFAN II	ug/L	0.13	0.063	0.2	6.31E-02	ug/L	% UCL	95% KM (t) UCL
		1031-07-8	ENDOSULFAN SULFATE	ug/L	0.097	0.021	0.18	2.10E-02	ug/L	% UCL	95% KM (Chebyshev) UCL
		1024-57-3	HEPTACHLOR EPOXIDE	ug/L	0.01	NA	0.01	1.00E-02	ug/L	Max	Insufficient Data*

TABLE 3.8
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 8 - SITE WIDE ALL GROUND WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Water
Exposure Medium: Ground Water (All Depths)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site Wide Ground Water	SVOCs									
	92-52-4	1,1'-BIPHENYL	ug/L	22.68	12.28	83	1.23E+01	ug/L	% UCL	95% KM (t) UCL
	120-83-2	2,4-DICHLOROPHENOL	ug/L	30.43	9.805	75	9.81E+00	ug/L	% UCL	95% KM (t) UCL
	105-67-9	2,4-DIMETHYLPHENOL	ug/L	4225	4021	38000	4.02E+03	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	91-57-6	2-METHYLNAPHTHALENE	ug/L	391.7	619.2	9800	6.19E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	95-48-7	2-METHYLPHENOL	ug/L	2111	977.7	15000	9.78E+02	ug/L	% UCL	95% KM (t) UCL
	88-75-5	2-NITROPHENOL	ug/L	3.87	5.64	6	5.64E+00	ug/L	% UCL	95% KM (t) UCL
	34METPH	3&4-METHYLPHENOL	ug/L	2679	4331	24000	4.33E+03	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	59-50-7	4-CHLORO-3-METHYLPHENOL	ug/L	1	NA	1	1.00E+00	ug/L	Max	Insufficient Data*
	106-44-5	4-METHYLPHENOL	ug/L	3439	8332	30000	8.33E+03	ug/L	% UCL	99% KM (Chebyshev) UCL
	100-02-7	4-NITROPHENOL	ug/L	5.84	9.69	18	9.69E+00	ug/L	% UCL	95% KM (t) UCL
	83-32-9	ACENAPHTHENE	ug/L	87.39	102	2200	1.02E+02	ug/L	% UCL	95% KM (Chebyshev) UCL
	208-96-8	ACENAPHTHYLENE	ug/L	146.9	167.6	2700	1.68E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	120-12-7	ANTHRACENE	ug/L	111.3	110.2	2000	1.10E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	1912-24-9	ATRAZINE	ug/L	53	NA	53	5.30E+01	ug/L	Max	Insufficient Data*
	56-55-3	BENZ(A)ANTHRACENE	ug/L	85.35	54.52	700	5.45E+01	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	ug/L	45.31	19.97	310	2.00E+01	ug/L	% UCL	95% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	ug/L	47.63	21.36	360	2.14E+01	ug/L	% UCL	95% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	ug/L	13.02	5.00	80	5.00E+00	ug/L	% UCL	95% KM (BCA) UCL
	207-08-9	BENZO(K)FLUORANTHENE	ug/L	45.35	17.99	340	1.80E+01	ug/L	% UCL	95% KM (Chebyshev) UCL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	ug/L	15.21	10.26	110	1.03E+01	ug/L	% UCL	95% KM (BCA) UCL
	86-74-8	CARBAZOLE	ug/L	115.5	102.4	930	1.02E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	ug/L	66.01	35.29	590	3.53E+01	ug/L	% UCL	95% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	ug/L	11.53	2.841	32	2.84E+00	ug/L	% UCL	95% KM (t) UCL
	132-64-9	DIBENZOFURAN	ug/L	142.3	191.9	3400	1.92E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	206-44-0	FLUORANTHENE	ug/L	119.7	162.9	3200	1.63E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	86-73-7	FLUORENE	ug/L	139.3	173.8	4200	1.74E+02	ug/L	% UCL	95% KM (Chebyshev) UCL
	87-68-3	HEXACHLOROBUTADIENE	ug/L	1	NA	1	1.00E+00	ug/L	Max	Insufficient Data*
	193-39-5	INDENO(1,2,3-CD)PYRENE	ug/L	21.61	8.46	110	8.46E+00	ug/L	% UCL	95% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	ug/L	2866	3975	35000	3.98E+03	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	98-95-3	NITROBENZENE	ug/L	2.6	NA	2.6	2.60E+00	ug/L	Max	Insufficient Data*
	85-01-8	PHENANTHRENE	ug/L	230.7	426.1	8300	4.26E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	108-95-2	PHENOL	ug/L	1154	1925	23000	1.93E+03	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	129-00-0	PYRENE	ug/L	82.76	100.9	1900	1.01E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL

TABLE 3.8
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 8 - SITE WIDE ALL GROUND WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Medium: Water
Exposure Medium: Ground Water (All Depths)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
Site Wide Ground Water	VOCs									
	87-61-6	1,2,3-TRICHLOROBENZENE	ug/L	10	12.08	19	1.21E+01	ug/L	% UCL	99% KM (Chebyshev) UCL
	120-82-1	1,2,4-TRICHLOROBENZENE	ug/L	105	13.54	468	1.35E+01	ug/L	% UCL	95% KM (t) UCL
	95-63-6	1,2,4-TRIMETHYLBENZENE	ug/L	171.4	326.2	900	3.26E+02	ug/L	% UCL	95% KM (Chebyshev) UCL
	95-50-1	1,2-DICHLOROBENZENE	ug/L	728.9	530.7	7560	5.31E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	108-67-8	1,3,5-TRIMETHYLBENZENE	ug/L	74.14	214.2	320	2.14E+02	ug/L	% UCL	99% KM (Chebyshev) UCL
	541-73-1	1,3-DICHLOROBENZENE	ug/L	16.08	5.451	62	5.45E+00	ug/L	% UCL	95% KM (t) UCL
	106-46-7	1,4-DICHLOROBENZENE	ug/L	578.4	468.2	8700	4.68E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	591-78-6	2-HEXANONE	ug/L	1.86	1.95	6.28	1.95E+00	ug/L	% UCL	95% KM (t) UCL
	67-64-1	ACETONE	ug/L	72.41	77.98	560	7.80E+01	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	71-43-2	BENZENE	ug/L	2537	5831	126000	5.83E+03	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	75-27-4	BROMODICHLOROMETHANE	ug/L	1.8	3	3	3.00E+00	ug/L	% UCL	95% KM (BCA) UCL
	75-15-0	CARBON DISULFIDE	ug/L	11.85	12.49	200	1.25E+01	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	108-90-7	CHLOROBENZENE	ug/L	265.6	181.1	3080	1.81E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	75-00-3	CHLOROETHANE	ug/L	9.584	4.58	32.6	4.58E+00	ug/L	% UCL	95% KM (BCA) UCL
	67-66-3	CHLOROFORM	ug/L	27.64	11.75	240	1.18E+01	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	100-41-4	ETHYLBENZENE	ug/L	171.7	146.6	1000	1.47E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	98-82-8	ISOPROPYLBENZENE	ug/L	9.569	3.95	68	3.95E+00	ug/L	% UCL	95% KM (t) UCL
	75-09-2	METHYLENE CHLORIDE	ug/L	10.2	0.74	25	7.44E-01	ug/L	% UCL	95% KM (t) UCL
	99-87-6	P-ISOPROPYLTOLUENE	ug/L	3.48	3.31	20	3.31E+00	ug/L	% UCL	95% KM (BCA) UCL
	135-98-8	SEC-BUTYLBENZENE	ug/L	23.68	11.88	120	1.19E+01	ug/L	% UCL	95% KM (t) UCL
	100-42-5	STYRENE	ug/L	721.9	819.6	17000	8.20E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	127-18-4	TETRACHLOROETHENE	ug/L	0.49	0.30	1.7	2.96E-01	ug/L	% UCL	95% KM (t) UCL
	108-88-3	TOLUENE	ug/L	998.7	1270	6500	1.27E+03	ug/L	% UCL	97.5% KM (Chebyshev) UCL
	75-01-4	VINYL CHLORIDE	ug/L	2.262	1.10	4.1	1.10E+00	ug/L	% UCL	95% KM (t) UCL
	1330-20-7	XYLENES, TOTAL	ug/L	549.8	891.9	4800	8.92E+02	ug/L	% UCL	97.5% KM (Chebyshev) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1248, -1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

c = Max - maximum detected concentration; %UCL - % upper confidence limit. KM - Kaplan-Meier method.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

KM (BCA) UCL = UCL based upon Kaplan-Meier Estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

TABLE 3.9a
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 9 - SYW-12 - SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil (0 - 2 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^c	Rationale
SYW-12 - Surface Soil	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	20.74	31.33	162.4	3.13E+01	ng/kg	% UCL	95% Approximate Gamma UCL
	METALS									
	7429-90-5	ALUMINUM	mg/kg	4802	5280	14000	5.28E+03	mg/kg	% UCL	95% Student's-t UCL
	7440-38-2	ARSENIC	mg/kg	5.62	6.27	20	6.27E+00	mg/kg	% UCL	95% KM (BCA) UCL
	7440-43-9	CADMIUM	mg/kg	12.48	17.43	52	1.74E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	102.2	124.1	410	1.24E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-50-8	COPPER	mg/kg	100.7	118.2	370	1.18E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-89-6	IRON	mg/kg	12307	13392	31000	1.34E+04	mg/kg	% UCL	95% Student's-t UCL
	7439-96-5	MANGANESE	mg/kg	319	334.1	630	3.34E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-97-6	MERCURY	mg/kg	1.50	1.87	8.6	1.87E+00	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-62-2	VANADIUM	mg/kg	13	14.65	53	1.47E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	PCBs									
		HIGHLY CHLORINATED PCBs ^a	mg/kg	0.75	0.94	3.47	9.36E-01	mg/kg	% UCL	95% Approximate Gamma UCL
		TOTAL PCBs ^b	mg/kg	0.75	0.94	3.47	9.36E-01	mg/kg	% UCL	95% Approximate Gamma UCL
	SVOCs									
	208-96-8	ACENAPHTHYLENE	mg/kg	1.28	2.03	15	2.03E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	4.47	9.31	91	9.31E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	3.79	6.61	49	6.61E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	5.45	9.55	67	9.55E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	1.51	2.33	15	2.33E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	1.88	3.26	24	3.26E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	4.74	9.51	89	9.51E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	0.5	0.59	5.3	5.86E-01	mg/kg	% UCL	95% KM (BCA) UCL
	132-64-9	DIBENZOFURAN	mg/kg	0.80	1.68	20	1.68E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	1.07	1.80	13	1.80E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	5.15	14.71	200	1.47E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	VOCs									
	71-43-2	BENZENE	mg/kg	0.0020	0.00098	0.0043	9.75E-04	mg/kg	% UCL	95% KM (t) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

c = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

Approximate Gamma UCL = Computation of UCL of the mean of a Gamma, G(k,θ) distribution (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.9b
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 9 - SYW-12 - SUBSURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
SYW-12 - Subsurface Soil	DIOXIN/FURAN									
	1746-01-6	2,3,7,8-TCDD Equivalent	ng/kg	20.74	31.33	162.4	3.13E+01	ng/kg	% UCL	95% Approximate Gamma UCL
	METALS									
	7429-90-5	ALUMINUM	mg/kg	4773	5276	14000	5.28E+03	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-38-2	ARSENIC	mg/kg	5.56	6.008	20	6.01E+00	mg/kg	% UCL	95% KM (BCA) UCL
	7440-43-9	CADMIUM	mg/kg	12.35	17.82	100	1.78E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL
	7440-47-3	CHROMIUM	mg/kg	97.5	148.1	470	1.48E+02	mg/kg	% UCL	95% H-UCL
	7440-50-8	COPPER	mg/kg	100.6	117.2	450	1.17E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-89-6	IRON	mg/kg	12181	13155	31000	1.32E+04	mg/kg	% UCL	95% Student's-t UCL
	7439-92-1	LEAD	mg/kg	138.7	161.1	410	1.39E+02	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/kg	311.8	326	630	3.26E+02	mg/kg	% UCL	95% Approximate Gamma UCL
	7439-97-6	MERCURY	mg/kg	1.44	1.78	8.6	1.78E+00	mg/kg	% UCL	95% Approximate Gamma UCL
	7440-62-2	VANADIUM	mg/kg	12.55	14	53	1.40E+01	mg/kg	% UCL	95% Approximate Gamma UCL
	PCBs									
		LESS CHLORINATED PCBs ^a	mg/kg	0.029	NA	0.029	2.88E-02	mg/kg	Max	*Insufficient Data
		HIGHLY CHLORINATED PCBs ^b	mg/kg	0.75	0.93	3.47	9.30E-01	mg/kg	% UCL	95% Approximate Gamma UCL
		TOTAL PCBs ^c	mg/kg	0.75	0.93	3.47	9.29E-01	mg/kg	% UCL	95% Approximate Gamma UCL
	SVOCs									
	208-96-8	ACENAPHTHYLENE	mg/kg	1.52	2.37	16	2.37E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	56-55-3	BENZ(A)ANTHRACENE	mg/kg	4.66	8.84	91	8.84E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	50-32-8	BENZO(A)PYRENE	mg/kg	4.18	6.81	49	6.81E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	205-99-2	BENZO(B)FLUORANTHENE	mg/kg	5.74	9.35	67	9.35E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	mg/kg	1.66	2.41	15	2.41E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	207-08-9	BENZO(K)FLUORANTHENE	mg/kg	1.94	3.14	24	3.14E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	218-01-9	CHRYSENE	mg/kg	4.94	9.09	89	9.09E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	53-70-3	DIBENZ(A,H)ANTHRACENE	mg/kg	0.55	0.63	5.3	6.34E-01	mg/kg	% UCL	95% KM (BCA) UCL
	132-64-9	DIBENZOFURAN	mg/kg	0.76	1.50	20	1.50E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	193-39-5	INDENO(1,2,3-CD)PYRENE	mg/kg	1.19	1.85	13	1.85E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	91-20-3	NAPHTHALENE	mg/kg	1.23	1.77	11	1.77E+00	mg/kg	% UCL	95% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	mg/kg	6.03	14.43	200	1.44E+01	mg/kg	% UCL	95% KM (Chebyshev) UCL

TABLE 3.9b
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 9 - SYW-12 - SUBSURFACE SOIL
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Subsurface Soil (0 - 10 ft bgs)

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^d	Rationale
	VOCs									
	71-43-2	BENZENE	mg/kg	0.0020	0.0018	0.0043	1.78E-03	mg/kg	% UCL	95% KM (t) UCL

Footnotes:

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Aroclor-1016,-1221,-1232,-1242 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

b = Aroclor-1248,-1254, -1260 samples were combined for analysis by ProUCL 4.0 and determination of an EPC.

c = All aroclor samples were combined for analysis by ProUCL 4.0 and the determination of an EPC.

d = Max - maximum detected concentration; %UCL - % upper confidence limit.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95% UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

Approximate Gamma UCL = Computation of UCL of the mean of a Gamma, G(k,θ) distribution (parametric).

H-UCL = (1-α)100% UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier Estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

Student's-t UCL = Computation method based upon student's t-distribution (parametric).

TABLE 3.9c
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 9 - SYW-12 - SHALLOW GROUND WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water
(0 - 10 ft bgs)**

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
SYW-12 - Shallow Ground Water	METALS									
	7429-90-5	ALUMINUM	mg/L	7.30	42.11	57	4.21E+01	mg/L	% UCL	99% KM (Chebyshev) UCL
	7440-36-0	ANTIMONY	mg/L	0.0038	0.0073	0.0058	5.80E-03	mg/L	Max	Insufficient Data*
	7440-38-2	ARSENIC	mg/L	0.020	0.019	0.059	1.91E-02	mg/L	% UCL	95% KM (BCA) UCL
	7440-39-3	BARIUM	mg/L	0.575	0.84	1.7	8.44E-01	mg/L	% UCL	95% Approximate Gamma UCL
	7440-43-9	CADMIUM	mg/L	0.012	0.011	0.027	1.07E-02	mg/L	% UCL	95% KM (t) UCL
	7440-47-3	CHROMIUM	mg/L	0.082	0.17	0.33	1.65E-01	mg/L	% UCL	95% KM (Chebyshev) UCL
	7440-50-8	COPPER	mg/L	0.13	0.33	0.74	3.26E-01	mg/L	% UCL	95% KM (Chebyshev) UCL
	7439-89-6	IRON	mg/L	27.45	49.32	120	4.93E+01	mg/L	% UCL	95% H-UCL
	7439-92-1	LEAD	mg/L	0.27	0.68	1.7	2.68E-01	mg/kg	Avg	USEPA Guidance for Lead Exposure
	7439-96-5	MANGANESE	mg/L	0.84	1.17	3.3	1.17E+00	mg/L	% UCL	95% H-UCL
	7439-97-6	MERCURY	mg/L	0.0018	0.0022	0.0087	2.20E-03	mg/L	% UCL	95% KM (BCA) UCL
	7440-02-0	NICKEL	mg/L	0.036	0.065	0.2	6.52E-02	mg/L	% UCL	95% Approximate Gamma UCL
	7782-49-2	SELENIUM	mg/L	0.011	0.010	0.022	1.03E-02	mg/L	% UCL	95% KM (t) UCL
	7440-28-0	THALLIUM	mg/L	0.023	NV	0.023	2.30E-02	mg/L	Max	Insufficient Data*
	7440-62-2	VANADIUM	mg/L	0.022	0.072	0.14	7.21E-02	mg/L	% UCL	97.5% KM (Chebyshev) UCL
	7440-66-6	ZINC	mg/L	0.367	0.47	1.9	4.70E-01	mg/L	% UCL	95% KM (BCA) UCL
	SVOC									
	100-02-7	4-NITROPHENOL	ug/l	1.1	NA	1.1	1.10E+00	ug/l	Max	Insufficient Data*
	83-32-9	ACENAPHTHENE	ug/l	9.23	10.13	41	1.01E+01	ug/l	% UCL	95% KM (t) UCL
	208-96-8	ACENAPHTHYLENE	ug/l	4.48	4.40	17	4.40E+00	ug/l	% UCL	95% KM (t) UCL
	1912-24-9	ATRAZINE	ug/L	53	NA	53	5.30E+01	ug/L	Max	Insufficient Data*
	56-55-3	BENZ(A)ANTHRACENE	ug/L	4.42	4.95	13	4.95E+00	ug/L	% UCL	95% KM (t) UCL
	50-32-8	BENZO(A)PYRENE	ug/l	5.03	5.59	18	5.59E+00	ug/l	% UCL	95% KM (t) UCL
	205-99-2	BENZO(B)FLUORANTHENE	ug/l	5.81	6.58	20	6.58E+00	ug/l	% UCL	95% KM (t) UCL
	191-24-2	BENZO(G,H,I)PERYLENE	ug/l	2.9	4.84	7.3	4.84E+00	ug/l	% UCL	95% KM (t) UCL
	207-08-9	BENZO(K)FLUORANTHENE	ug/l	2.93	5.30	6.9	5.30E+00	ug/l	% UCL	95% KM (t) UCL
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	ug/l	2.77	5.49	5.9	5.49E+00	ug/l	% UCL	95% KM (t) UCL
	86-74-8	CARBAZOLE	ug/l	2.35	4.35	3.5	3.50E+00	ug/l	Max	Max
	218-01-9	CHRYSENE	ug/l	4.31	4.88	14	4.88E+00	ug/l	% UCL	95% KM (t) UCL
	91-20-3	NAPHTHALENE	ug/l	21.53	107.5	170	1.08E+02	ug/l	% UCL	99% KM (Chebyshev) UCL
	85-01-8	PHENANTHRENE	ug/l	6.58	7.04	17	7.04E+00	ug/l	% UCL	95% KM (t) UCL
	129-00-0	PYRENE	ug/l	5.41	6.71	22	6.71E+00	ug/l	% UCL	95% KM (t) UCL

TABLE 3.9c
EXPOSURE POINT CONCENTRATION SUMMARY - EXPOSURE UNIT 9 - SYW-12 - SHALLOW GROUND WATER
REASONABLE MAXIMUM EXPOSURE/CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Medium: Water
Exposure Medium: Shallow Ground Water (0 - 10 ft bgs)**

Exposure Point	CAS Number	Chemical of Potential Concern	Units	Average Detected Concentration	Potential UCL to Use	Maximum Detected Concentration	Exposure Point Concentration			
							Value	Units	Statistic ^a	Rationale
	VOC									
	106-46-7	1,4-DICHLOROBENZENE	ug/l	0.19	0.32	0.34	3.22E-01	ug/l	% UCL	95% KM (t) UCL
	71-43-2	BENZENE	ug/l	0.9	NA	0.9	9.00E-01	ug/l	Max	Insufficient Data*

Footnotes:

** Sample start depth less than or equal to 10 ft bgs.

UCL based on USEPA ProUCL (V. 4.0) recommendation, using Regression on Order Statistics (ROS) for evaluating data with non-detect (ND) samples.

a = Max - maximum detected concentration; %UCL - % upper confidence limit.

NA = UCL could not be calculated because of low sample number or low detection frequency.

It is possible that in certain instances, the calculated 95% UCL is smaller than the mean detected concentration. This reflects a low detection frequency and non-detect samples largely outnumbering detected samples, causing the 95%

UCL recommended by ProUCL v4.0 to be smaller than the mean detected concentration, since it reflects the large number of non-detect samples.

* = ProUCL does not provide 95% UCLs when there is insufficient data, as defined by fewer than 3 samples or fewer than 2 unique detected samples. In these cases, the EPC is obtained by using the maximum detected concentration.

Approximate Gamma UCL = Computation of UCL of the mean of a Gamma, G(k,θ) distribution (parametric).

H-UCL = (1-α)100% UCL of the mean based upon H-statistic (H-UCL) (parametric).

KM (BCA) UCL = UCL based upon Kaplan-Meier Estimates using the bias corrected accelerated percentile bootstrap method. (non-parametric).

KM (Chebyshev) UCL = UCL based upon Kaplan-Meier estimates using the Chebyshev inequality (non-parametric).

KM (t) UCL = UCL based upon Kaplan-Meier estimates using student's t-distribution critical value (non-parametric).

RAGS Table 4 RME Series

TABLE 4.1a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5400	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	3	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	1.2	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	1.37E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Trespasser	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Trespasser	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Adult Trespasser	Adult > 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	1.37E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1a RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutagenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future	
Exposure Areas:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area*	
Medium:	Soil	
Exposure Medium:	Surface Soil	(0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (12-<16) x FI x EF x CF x 1/AT [for child aged 12-<16 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (12-<16)	Age Adjusted Ingestion Rate of Soil (12-<16 yrs)	mg-yr/day-kg	7.7	Calculated	
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
		Older Child 16 to < 18 years	EU-1	IR-S-Adj (16-<18)	Age Adjusted Ingestion Rate of Soil (16-<18 yrs)	mg-yr/day-kg	3.2	Calculated	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (16-<18) x FI x EF x CF x 1/AT [for child aged 16-<18 years]
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (12-<16) x ABS x CF x EF x 1/AT [for child aged 12-<16 years]
				SSAF-Adj (12-<16)	Age Adjusted Soil to Skin Adherence Factor (12-<16 yrs)	mg-yr/day-kg	1144	Calculated	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				SSAF-Adj (16-<18)	Age Adjusted Soil to Skin Adherence Factor (16-<18 yrs)	mg-yr/day-kg	533	Calculated	
		Older Child 16 to < 18 years	EU-1	CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (16-<18) x ABS x CF x EF x 1/AT [for child aged 16-<18 years]
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (12-<16) x ET x EF x 1/AT [for child aged 12-<16 years]
				IN-Adj (12-<16)	Age Adjusted Inhalation Rate (12-<16 yrs)	m ³ -yr/hr-kg	0.044	Calculated	
				ET	Exposure Time	hr/day	4	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	
				IN-Adj (16-<18)	Age Adjusted Inhalation Rate (16-<18 yrs)	m ³ -yr/hr-kg	0.019	Calculated	
				ET	Exposure Time	hr/day	4	Best Professional Judgement	
		Older Child 16 to < 18 years	EU-1	EF	Exposure Frequency	days/year	42	Best Professional Judgement	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (16-<18) x ET x EF x 1/AT [for child aged 16-<18 years]
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

United States Environmental Protection Agency (USEPA). 1996. Soil Screening Guidance: User's Guide. USEPA/540/F-95/041.

United States Environmental Protection Agency (USEPA). 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24.

TABLE 4.1b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004; Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004; Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Referenece	Intake Equation/Model Name
Inhalation	Utility Worker	Adult > 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs:
				InR	Inhalation Rate	m ³ /hour	1.5	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m3) =
				PEF	Particulate Emission Factor	m ³ /kg	1.37E+09	See Appendix F	CS / PEF
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	For VOCs:
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	Chemical Concentration in Air (CA, mg/m3) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CS / VF
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1c RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Construction Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Construction Worker	Adult > 18 years	EU-1	AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1c RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Construction Worker	Adult > 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	3.2	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	8.72E+05	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1d RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Utility Worker	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_p \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_p \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_p \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Sample start depth less than or equal to 10 ft bgs.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1e RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Construction Worker	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	<p>DAD (mg/kg-day) =</p> $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Sample start depth less than or equal to 10 ft bgs.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1f RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5400	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	3	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Ingestion	Adult Trespasser	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1f RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Adult Trespasser	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Ingestion	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1f RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) =
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.9	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = In a few instances, sediment samples with start depths of 0 ft and end depths ranging from >1 to 3 ft were also incorporated in the evaluation of surface sediment.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1f RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area*
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (12-<16) x EF x CF x 1/AT [for child aged 12-<16 years]
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (12-<16)	Age Adjusted Ingestion Rate of Soil (12-<16 yrs)	mg-yr/day-kg	7.7	Calculated	
				FI	Fraction Ingested from Sediment	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	USEPA 1989, Exhibits 6-11 through 6-16
				AT-C	Averaging Time - Cancer	days	25550		
				CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
		Older Child 16 to < 18 years	EU-1	IR-S-Adj (16-<18)	Age Adjusted Ingestion Rate of Soil (12-<16 yrs)	mg-yr/day-kg	3.2	Calculated	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (16-<18) x EF x CF x 1/AT [for child aged 16-<18 years]
				FI	Fraction Ingested from Sediment	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (12-<16) x ABS x CF x EF x 1/AT [for child aged 12-<16 years]
				SSAF-Adj (12-<16)	Age Adjusted Sediment to Skin Adherence Factor (12-<16 yrs)	mg-yr/day-kg	1144	Calculated	
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	USEPA 1989, Exhibits 6-11 through 6-16
				AT-C	Averaging Time - Cancer	days	25550		
				CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	
				SSAF-Adj (16-<18)	Age Adjusted Sediment to Skin Adherence Factor (16-<18 yrs)	mg-yr/day-kg	533	Calculated	
		Older Child 16 to < 18 years	EU-1	CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (16-<18) x ABS x CF x EF x 1/AT [for child aged 16-<18 years]
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

TABLE 4.1g RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SEDIMENT & SUBSURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface & Subsurface Sediment (0 - 10 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) =
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				IR	Ingestion Rate of Sediment	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) =
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.9	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Where construction or utility workers have may contact with the sediment of Harbor Brook, a depth interval of 0 - 10 ft bgs is applied. This reflects the potential for contact with deeper sediments for bridge reconstruction, which is anticipated and unique to the Harbor Brook exposure area. In a few instances, sediment samples with start depths of 0 ft and end depths ranging from >1 to 3 ft were also incorporated in the evaluation of surface sediment.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1h RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE SEDIMENT & SUBSURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface & Subsurface Sediment (0 - 10 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Construction Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Construction Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.9	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Where construction or utility workers have may contact with the sediment of Harbor Brook, a depth interval of 0 - 10 ft bgs is applied. This reflects the potential for contact with deeper sediments for bridge reconstruction, which is anticipated and unique to the Harbor Brook exposure area. In a few instances, sediment samples with start depths of 0 ft and end depths ranging from >1 to 3 ft were also incorporated in the evaluation of surface sediment.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1i RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ $\text{Where DA}_{\text{event}} \text{ (Organics)} =$ $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ $\text{Where DA}_{\text{event}} \text{ (Inorganics)} =$ $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	5400	NYSDEC 2002, Onondaga Lake HHRA	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	4	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
	Adult Trespasser	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ $\text{Where DA}_{\text{event}} \text{ (Organics)} =$ $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ $\text{Where DA}_{\text{event}} \text{ (Inorganics)} =$ $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	4	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1i RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Utility Worker	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K_P	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t_{event}	Event Duration	hr/event	8	Best Professional Judgment	
				τ_{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t^*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1i RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area*
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CW	Chemical Concentration in Water	µg/L	See Table 3	See RAGS Table 3 Series	<p>Dermal Absorbed Dose (DAD mg/kg-day) = $DA_{event} \times EV \times EF \times CF \times ESA-Adj \times 1/AT$ Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg-L/µg-mL	0.00001	Unit Conversion	
				ESA-Adj (12-<16)	Exposed Surface Area Available for Dermal Contact to Water (12-<16)	[cm ² -yr]/kg	381	Calculated	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	USEPA, 2004; Exhibit 3-2	
				FA	Fraction Absorbed	unitless		USEPA 2004, Exhibits B-3 & B-4	
				K _p	Permeability Constant	cm/hr		USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	4	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event		USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr		USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless		USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
	Older Child Trespasser	Older Child 16 to < 18 years	EU-1	CW	Chemical Concentration in Water	µg/L	See Table 3	See RAGS Table 3 Series	<p>Dermal Absorbed Dose (DAD mg/kg-day) = $DA_{event} \times EV \times EF \times CF \times ESA-Adj \times 1/AT$ Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg-L/µg-mL	0.00001	Unit Conversion	
				ESA-Adj (12-<16)	Exposed Surface Area Available for Dermal Contact to Water (12-<16)	[cm ² -yr]/kg	177.7	Calculated	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	USEPA, 2004; Exhibit 3-2	
				FA	Fraction Absorbed	unitless		USEPA 2004, Exhibits B-3 & B-4	
				K _p	Permeability Constant	cm/hr		USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	4	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event		USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr		USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless		USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

United States Environmental Protection Agency (USEPA). 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1j RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Construction Worker	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K_P	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t_{event}	Event Duration	hr/event	8	Best Professional Judgment	
				τ_{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t^*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1k RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 1, FISH TISSUE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Fish Tissue
Exposure Medium:	Onondaga Lake Fish Tissue

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	$\text{Chronic Daily Intake (CDI)} \text{ (mg/kg-day)} = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} \text{ (mg/kg-day)} = C \times [1-CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion USEPA 1997; Table 10-46, avg male and female	
				IR	Ingestion Rate of Fish Tissue	g fish/day	16.7		
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	NA	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
	Adult Trespasser	Adult > 18 years	EU-1	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	$\text{Chronic Daily Intake (CDI)} \text{ (mg/kg-day)} = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} \text{ (mg/kg-day)} = C \times [1-CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion USEPA 1997; Page 10-26	
				IR	Ingestion Rate of Fish Tissue	g fish/day	25		
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	NA	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

1 = Used to adjust exposure point concentration (EPC) for PCBs and PCDD/PCDFs ingested for central tendency (CT) only. NA indicates not applicable to the RME scenario.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 2 Food Ingestion Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.2 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 2, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, and DSA #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Surveillance Worker	Adult > 18 years	EU-2	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	37	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Surveillance Worker	Adult > 18 years	EU-2	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2480	USEPA 2004; Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.07	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency**	days/year	37	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.2 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 2, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, and DSA #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Surveillance Worker	Adult > 18 years	EU-2	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	1	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	3.44E+09	USEPA, 2002.	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	37	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Value is based on surveillance once per week, factoring in two weeks of vacation annually and reduction of 25% due to snow cover (rounded up from 24.69%). The number of days of work that exposure is reduced are rounded to nearest whole day, see HHRA text for derivation.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.3a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 3, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Interstate 690 Drainage Ditch
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Drainage Ditch Worker	Adult > 18 years	EU-3	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	330	USEPA 2002; Exhibit 1-2, NYSDEC 2002, Onondaga Lake HHRA	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	10	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Drainage Ditch Worker	Adult > 18 years	EU-3	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.9	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	10	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.3a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 3, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Interstate 690 Drainage Ditch
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Drainage Ditch Worker	Adult > 18 years	EU-3	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For VOCs:
				InR	Inhalation Rate	m ³ /hour	1.5	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m3) =
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	CS / VF
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	10	Best Professional Judgment	Chronic Daily Intake (CDI in mg/kg-day) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CA x InR x ET x EF x ED x 1/BW x 1/AT
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989; Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.3b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 3, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Interstate 690 Drainage Ditch
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Drainage Ditch Worker	Adult > 18 years	EU-3	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$DAD \text{ (mg/kg-day)} =$ $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	10	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.4 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 4, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Railroad Worker	Adult > 18 years	EU-4	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Railroad Worker	Adult > 18 years	EU-4	AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.4 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 4, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Railroad Worker	Adult > 18 years	EU-4	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	2.5	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	8.22E+08	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	2	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Value is based on 250 work days per year reduced by 25% due to snow cover (rounded up from 24.69%). The number of days of work that exposure is reduced are rounded to nearest whole day, see HHRA text for derivation.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.5 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 5, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current
Exposure Areas*:	Penn-Can Property
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Commercial and Industrial Worker	Adult > 18 years	EU-5	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Commercial and Industrial Worker	Adult > 18 years	EU-5	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.5 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 5, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current
Exposure Areas*:	Penn-Can Property
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Commercial and Industrial Worker	Adult > 18 years	EU-5	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs:
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m3) =
				PEF	Particulate Emission Factor	m ³ /kg	5.89E+08	See Appendix F	CS / PEF
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	For VOCs:
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	Chemical Concentration in Air (CA, mg/m3) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CS / VF
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

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TABLE 4.6a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Recreator	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Recreator	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA; USEPA 2004; Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Adult Recreator	Adult > 18 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	<p>For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m³) = CS / PEF</p> <p>For VOCs: Chemical Concentration in Air (CA, mg/m³) = CS / VF</p> <p>Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT</p>
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Ingestion	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	<p>Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT</p>
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	200	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	3	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Recreator	Younger Child 0 to < 6 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = $CA \times InR \times ET \times EF \times ED \times 1/BW \times 1/AT$
				InR	Inhalation Rate	m ³ /hour	1.2	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Resident	> 18 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Resident	> 18 years	EU-6	AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004; Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.07	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Adult Resident	Adult > 18 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.8	USEPA 1997, Table 5-11	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	16	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Ingestion	Child Resident	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	200	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Resident	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Child Resident	Younger Child 0 to < 6 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	0.42	USEPA 1997, Table 5-11	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	24	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

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TABLE 4.6a RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (0-<2) x FI x EF x CF x 1/AT [for child aged 0-<2 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	39.7	Calculated	
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	USEPA 1989, Exhibits 6-11 through 6-16
				AT-C	Averaging Time - Cancer	days	25550		
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
		Younger Child 2 to < 6 years	EU-6	IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	49.8	Calculated	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (2-<6) x FI x EF x CF x 1/AT [for child aged 2-<6 years]
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	USEPA, 2004; Exhibit 3-2	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (0-<2) x ABS x CF x EF x 1/AT [for child aged 0-<2 years]
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	1548	Calculated	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	USEPA 1989, Exhibits 6-11 through 6-16
				AT-C	Averaging Time - Cancer	days	25,550		
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				SSAF-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	2159	Calculated	
		Younger Child 2 to < 6 years	EU-6	CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (2-<6) x ABS x CF x EF x 1/AT [for child aged 2-<6 years]
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Recreator	Younger Child 0 to < 2 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (0-<2) x ET x EF x 1/AT [for child aged 0-<2 years]
				IN-Adj (0-<2)	Age Adjusted Inhalation Rate (0-<2 yrs)	m ³ -yr/hr-kg	0.046	Calculated	
				ET	Exposure Time	hr/day	4	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	
				IN-Adj (2-<6)	Age Adjusted Inhalation Rate (2-<6 yrs)	m ³ -yr/hr-kg	0.081	Calculated	
				ET	Exposure Time	hr/day	4	Best Professional Judgement	
		Younger Child 2 to < 6 years	EU-6	EF	Exposure Frequency	days/year	42	Best Professional Judgement	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (2-<6) x ET x EF x 1/AT [for child aged 2-<6 years]
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Resident	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (0-<2) x FI x EF x CF x 1/AT [for child aged 0-<2 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	39.7	Calculated	
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (2-<6) x FI x EF x CF x 1/AT [for child aged 2-<6 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	49.8	Calculated	
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
Dermal	Child Resident	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (0-<2) x ABS x CF x EF x 1/AT [for child aged 0-<2 years]
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	103	Calculated	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (2-<6) x ABS x CF x EF x 1/AT [for child aged 2-<6 years]
				SSAF-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	144	Calculated	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
Inhalation	Child Resident	Younger Child 0 to < 2 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (0-<2) x ET x EF x 1/AT [for child aged 0-<2 years]
				IN-Adj (0-<2)	Age Adjusted Inhalation Rate (0-<2 yrs)	m ³ -yr/hr-kg	0.046	Calculated	
				ET	Exposure Time	hr/day	24	Best Professional Judgement	
				EF	Exposure Frequency	days/year	350	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				Younger Child 2 to < 6 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	
		IN-Adj (2-<6)	Age Adjusted Inhalation Rate (2-<6 yrs)			m ³ -yr/hr-kg	0.081	Calculated	
		ET	Exposure Time			hr/day	24	Best Professional Judgement	
		EF	Exposure Frequency			days/year	350	Best Professional Judgement	
					AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

United States Environmental Protection Agency (USEPA). 1996. Soil Screening Guidance: User's Guide. USEPA/540/F-95/041.

United States Environmental Protection Agency (USEPA). 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Recreator	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Recreator	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004; Exhibit C-1	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	200	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	3	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6b RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Soil
Exposure Medium:	Surface Sediment (0 - 1 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (0-<2) x FI x EF x CF x 1/AT [for child aged 0-<2 years]
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	39.7	Calculated	
				FI	Fraction Ingested from Sediment	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (2-<6) x FI x EF x CF x 1/AT [for child aged 2-<6 years]
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	49.8	Calculated	
Dermal	Child Recreator	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (0-<2) x ABS x CF x EF x 1/AT [for child aged 0-<2 years]
				SSAF-Adj (0-<2)	Age Adjusted Sediment to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	1548	Calculated	
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (2-<6) x ABS x CF x EF x 1/AT [for child aged 2-<6 years]
				SSAF-Adj (2-<6)	Age Adjusted Sediment to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	2159	Calculated	
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

United States Environmental Protection Agency (USEPA). 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6c RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Adult Recreator	Adult > 18 years	EU-6	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} =$ $\text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004; Exhibit C-1	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	4	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
Dermal	Adult Recreator	Adult > 18 years	EU-6	AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6c RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-6	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$DAD \text{ (mg/kg-day)} =$ $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	NYSDEC 2002, Onondaga Lake HHRA	
				SA	Skin Surface Area	cm ²	2800	USEPA 2004; Exhibit C-1	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA 2004, Exhibits B-3 & B-4	
				BW	Body Weight	kg	15	USEPA 1991; Attachment B	
				FA	Fraction Absorbed	unitless	Chemical Specific	NYSDEC 2002, Onondaga Lake HHRA	
				t _{event}	Event Duration	hr/event	4	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	NYSDEC 2002, Onondaga Lake HHRA	
				t*	Time to Reach Steady State	hr	Chemical Specific	NYSDEC 2002, Onondaga Lake HHRA	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	NYSDEC 2002, Onondaga Lake HHRA	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1997; Table 7-11	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1997; Table 7-11	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6c RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Child Recreator	Younger Child 0 to < 2 years	EU-6	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	<div>Dermal Absorbed Dose (DAD mg/kg-day) = $DA_{event} \times EV \times EF \times CF \times ESA-Adj \times 1/AT$ Where DA_{event} (Organics) = <div>If $t_{event} \leq t^*$, then $DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ If $t_{event} > t^*$, then $DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ Where DA_{event} (Inorganics) = $DA_{event} = K_P \times C_W \times t_{event}$</div></div>
				CF	Unit Conversion Factor for Water	mg-L/μg-mL	0.000001	Unit Conversion	
				ESA-Adj (0-<2)	Exposed Surface Area Available for Dermal Contact to Water (0-<2)	[cm2-yr]/kg	516	Calculated	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	USEPA, 2004; Exhibit 3-2	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				Kp	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	4	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Younger Child 2 to < 6 years	EU-6	EU-6	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	<div>Dermal Absorbed Dose (DAD mg/kg-day) = $DA_{event} \times EV \times EF \times CF \times ESA-Adj \times 1/AT$ Where DA_{event} (Organics) = <div>If $t_{event} \leq t^*$, then $DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ If $t_{event} > t^*$, then $DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ Where DA_{event} (Inorganics) = $DA_{event} = K_P \times C_W \times t_{event}$</div></div>
				CF	Unit Conversion Factor for Water	mg-L/μg-mL	0.000001	Unit Conversion	
				ESA-Adj (2-<6)	Exposed Surface Area Available for Dermal Contact to Water (2-<6)	[cm2-yr]/kg	720	Calculated	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	42	USEPA, 2004; Exhibit 3-2	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				Kp	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	4	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

TABLE 4.6d RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 6, FISH TISSUE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1*
Medium:	Fish Tissue
Exposure Medium:	Onondage Lake Fish Tissue

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Adult Recreator	Adult > 18 years	EU-6	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	$\text{Chronic Daily Intake (CDI)} \text{ (mg/kg-day)} = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} \text{ (mg/kg-day)} = C \times [1-CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion	
				IR	Ingestion Rate of Fish Tissue	g fish/day	25	USEPA 1997; Page 10-26	
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	NA	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
	Child Recreator	Younger Child 0 to < 6 years	EU-6	AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	$\text{Chronic Daily Intake (CDI)} \text{ (mg/kg-day)} = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} \text{ (mg/kg-day)} = C \times [1-CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
				C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion	
				IR	Ingestion Rate of Fish Tissue	g fish/day	8.3	USEPA 1997; Table 10-46	
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	NA	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	$\text{Chronic Daily Intake (CDI)} \text{ (mg/kg-day)} = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} \text{ (mg/kg-day)} = C \times [1-CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Attachment B	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

1 = Used to adjust exposure point concentration (EPC) for PCBs and PCDD/PCDFs ingested for central tendency (CT) only. NA indicates not applicable to the RME scenario.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 2 Food Ingestion Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.7 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 7, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Penn-Can Property, Lakeshore Area, East Flume, DSA #A, DSA #2, AOS #1, and AOS #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Commercial and Industrial Worker	Adult > 18 years	EU-7	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Commercial and Industrial Worker	Adult > 18 years	EU-7	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.7 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 7, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Penn-Can Property, Lakeshore Area, East Flume, DSA #A, DSA #2, AOS #1, and AOS #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Commercial and Industrial Worker	Adult > 18 years	EU-7	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	1.74E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

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USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.8 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 8, POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Resident	Adult > 18 years	EU-8	CW	Chemical Concentration in Potable Water	ug/L	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CW \times CF \times IR \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				IR	Ingestion Rate of Water	L/day	2	USEPA 1989; Exhibit 6-11	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Child 0 to < 6 years	EU-8	CW	Chemical Concentration in Potable Water	ug/L	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) = $CW \times CF \times IR \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				IR	Ingestion Rate of Water	L/day	1	USEPA 1989; Exhibit 6-11	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
	Commercial and Industrial Worker	Adult > 18 years	EU-8	CS	Chemical Concentration in Potable Water	ug/L	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) = $CW \times CF \times IR \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				IR	Ingestion Rate of Water	L/day	2	USEPA 1989; Exhibit 6-11	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.8 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 8, POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Resident	Adult > 18 years	EU-8 Shower/Bathing	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	<p>DAD (mg/kg-day) =</p> $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> <p>If $t_{event} \leq t^*$, then $DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$</p> <p>If $t_{event} > t^*$, then $DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right)}{1+B} \right]$</p> <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _P	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	18000	USEPA, 2004; Exhibit 3-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	0.58	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Resident	Child 0 to < 6 years	EU-8 Shower/Bathing	AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	<p>DAD (mg/kg-day) =</p> $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> <p>If $t_{event} \leq t^*$, then $DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$</p> <p>If $t_{event} > t^*$, then $DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right)}{1+B} \right]$</p> <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _P	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	6600	USEPA, 2004; Exhibit 3-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	1	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.8 RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 8, POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Resident	Adult > 18 years	EU-8 Shower/Bathing	CA	Chemical Concentration in Air	mg/m ³	Calculated	Schaum et al. 1994	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.8	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	0.58	Schaum et al. 1994	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Child (Ages 0 - 6)	EU-8 Shower/Bathing	CA	Chemical Concentration in Air	mg/m ³	Calculated	Schaum et al. 1994	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.42	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	1	Schaum et al. 1994	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

Schaum, J., K. Hoang, R. Kinerson, J. Moya, and R.G.M. Wang. 1994. Estimating Dermal and Inhalation Exposure to volatile Chemicals in Domestic Water. USEPA Region II.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.8 RME Supplement
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 8: POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Resident	Child 0 to < 2 years	EU-8	CW	Chemical Concentration in Water	µg/L	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) = CW x IR-W-Adj (0-<2) x EF x CF x 1/AT [for child aged 0-<2 years]
				IRW-Adj (0-<2)	Ingestion Rate of Water, Age-adjusted (0-<2 yrs)	L-yr/day-kg	0.22	Calculated	
				CF	Unit Conversion Factor for Water	mg/µg	0.001	Unit Conversion	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
		Child 2 to < 6 years	EU-8	AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	CDI (mg/kg-day) = CW x IR-W-Adj (2-<6) x EF x CF x 1/AT [for child aged 2-<6 years]
				CW	Chemical Concentration in Water	µg/L	See Table 3	RAGS Table 3 Series	
				IRW-Adj (2-<6)	Ingestion Rate of Water, Age-adjusted (2-<6 yrs)	L-yr/day-kg	0.37	Calculated	
				CF	Unit Conversion Factor for Water	mg/µg	0.001	Unit Conversion	
Dermal	Resident	Child 0 to < 2 years	EU-8 Bath	EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	Dermal Absorbed Dose (DAD mg/kg-day) = DA _{event} x EV x EF x CF x TSA-Adj x 1/AT Where DA _{event} (Organics) = $\text{If } t_{\text{event}} \leq t^*, \text{ then } DA_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } DA_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ Where DA _{event} (Inorganics) = $DA_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				TSA-Adj (0-<2)	Exposed Surface Area Available for Dermal Contact to Water (0-<2)	[cm ² -yr]/kg	1172	Calculated	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	1	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	
				CF	Unit Conversion Factor for Water	mg-L/µg-mL	0.000001	Unit Conversion	
								Calculated	
								USEPA 2004, Exhibits B-3 & B-4	

TABLE 4.8 RME Supplement
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 8: POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Resident	Child 2 to < 6 years	EU-8 Bath	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	<p>Dermal Absorbed Dose (DAD mg/kg-day) = $DA_{event} \times EV \times EF \times CF \times TSA-Adj \times 1/AT$ Where DA_{event} (Organics) =</p> <div> <p>If $t_{event} \leq t^*$, then $DA_{event} = 2FA \times K_p \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$</p> <p>If $t_{event} > t^*$, then $DA_{event} = FA \times K_p \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$</p> <p>Where DA_{event} (Inorganics) =</p> <div> $DA_{event} = K_p \times C_W \times t_{event}$ </div> </div>
				CF	Unit Conversion Factor for Water	mg-L/ μ g-mL	0.000001	Unit Conversion	
				TSA-Adj (2-<6)	Exposed Surface Area Available for Dermal Contact to Water (2-<6)	[cm ² -yr]/kg	1685	Calculated	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	1	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Resident	Child 0 to < 2 years	EU-8 Shower/Bathing	CA	Chemical Concentration in Air	mg/m ³	Table TBD	Schaum et al. 1994	Chronic Daily Intake (CDI in mg/kg-day) = $CA \times IN-Adj (0-<2) \times ET \times EF \times 1/AT$ [for child aged 0-<2 years]
				IN-Adj (0-<2)	Inhalation Rate	m ³ -yr/hr-kg	0.046	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	1	Schaum et al. 1994	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Child 2 to < 6 years	EU-8 Shower/Bathing	CA	Chemical Concentration in Air	mg/m ³	Table TBD	Schaum et al. 1994	Chronic Daily Intake (CDI in mg/kg-day) = $CA \times IN-Adj (0-<2) \times ET \times EF \times 1/AT$ [for child aged 2-<6 years]
				IN-Adj (2-<6)	Inhalation Rate	m ³ -yr/hr-kg	0.081	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	1	Schaum et al. 1994	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

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United States Environmental Protection Agency (USEPA). 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Recreator	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Recreator	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA; USEPA 2004; Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
Inhalation	Adult Recreator	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	200	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA Per NYSDEC Comment; Letter dated 3/12/2008	
				AF	Soil to Skin Adherence Factor	mg/cm ²	3	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Recreator	Younger Child 0 to < 6 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI in mg/kg-day) = $CA \times InR \times ET \times EF \times ED \times 1/BW \times 1/AT$
				InR	Inhalation Rate	m ³ /hour	1.2	USEPA 1997, Table 5-23	
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Railroad Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Railroad Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9a RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Railroad Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	2.5	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	2	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Value is based on 250 work days per year reduced by 25% due to snow cover (rounded up from 24.69%). The number of days of work that exposure is reduced are rounded to nearest whole day, see HHRA text for derivation.

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USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9a RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutagenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 2 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (0-<2) x FI x EF x CF x 1/AT [for child aged 0-<2 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	39.7	Calculated	
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (2-<6) x FI x EF x CF x 1/AT [for child aged 2-<6 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	49.8	Calculated	
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 0 to < 2 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSFAF-Adj (0-<2) x ABS x CF x EF x 1/AT [for child aged 0-<2 years]
				SSFAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	1548	Calculated	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSFAF-Adj (2-<6) x ABS x CF x EF x 1/AT [for child aged 2-<6 years]
				SSFAF-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	2159	Calculated	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Recreator	Younger Child 0 to < 2 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (0-<2) x ET x EF x 1/AT [for child aged 0-<2 years]
				IN-Adj (0-<2)	Age Adjusted Inhalation Rate (0-<2 yrs)	m ³ -yr/hr-kg	0.046	Calculated	
				ET	Exposure Time	hr/day	4	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (2-<6) x ET x EF x 1/AT [for child aged 2-<6 years]
				IN-Adj (2-<6)	Age Adjusted Inhalation Rate (2-<6 yrs)	m ³ -yr/hr-kg	0.081	Calculated	
				ET	Exposure Time	hr/day	4	Best Professional Judgement	
				EF	Exposure Frequency	days/year	42	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

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United States Environmental Protection Agency (USEPA). 1996. Soil Screening Guidance: User's Guide. USEPA/540/F-95/041.

TABLE 4.9b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Resident	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Resident	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004; Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.07	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Adult Resident	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.8	USEPA 1997, Table 5-11	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	16	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Resident	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	200	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Resident	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	3	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Resident	Younger Child 0 to < 6 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.42	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	24	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Commercial and Industrial Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Commercial and Industrial Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9b RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Commercial and Industrial Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9b RME Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future	
Exposure Areas:	State Wetland SYW-12	
Medium:	Soil	
Exposure Medium:	Surface Soil	(0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Resident	Younger Child 0 to < 2 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (0-<2) x FI x EF x CF x 1/AT [for child aged 0-<2 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	39.7	Calculated	
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	350	Best Professional Judgement	USEPA 1989, Exhibits 6-11 through 6-16
				AT-C	Averaging Time - Cancer	days	25550		
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
		Younger Child 2 to < 6 years	EU-9	IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	49.8	Calculated	Chronic Daily Intake (CDI mg/kg-day) = CS x IR-S-Adj (2-<6) x FI x EF x CF x 1/AT [for child aged 2-<6 years]
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	
				EF	Exposure Frequency	days/year	350	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Resident	Younger Child 0 to < 2 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (0-<2) x ABS x CF x EF x 1/AT [for child aged 0-<2 years]
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	103	Calculated	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	350	Best Professional Judgement	USEPA 1989, Exhibits 6-11 through 6-16
				AT-C	Averaging Time - Cancer	days	25550		
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				SSAF-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	144	Calculated	
		Younger Child 2 to < 6 years	EU-9	CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	Dermal Absorbed Dose (DAD mg/kg-day) = CS x SSAF-Adj (2-<6) x ABS x CF x EF x 1/AT [for child aged 2-<6 years]
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				EF	Exposure Frequency	days/year	350	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Resident	Younger Child 0 to < 2 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (0-<2) x ET x EF x 1/AT [for child aged 0-<2 years]
				IN-Adj (0-<2)	Age Adjusted Inhalation Rate (0-<2 yrs)	m ³ -yr/hr-kg	0.046	Calculated	
				ET	Exposure Time	hr/day	24	Best Professional Judgement	
				EF	Exposure Frequency	days/year	350	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI mg/kg-day) = CA x IN-Adj (2-<6) x ET x EF x 1/AT [for child aged 2-<6 years]
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	
				IN-Adj (2-<6)	Age Adjusted Inhalation Rate (2-<6 yrs)	m ³ -yr/hr-kg	0.081	Calculated	
				ET	Exposure Time	hr/day	24	Best Professional Judgement	
				EF	Exposure Frequency	days/year	350	Best Professional Judgement	USEPA 1989, Exhibits 6-11 through 6-16
				AT-C	Averaging Time - Cancer	days	25550		

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

United States Environmental Protection Agency (USEPA). 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24.

United States Environmental Protection Agency (USEPA). 1996. Soil Screening Guidance: User's Guide. USEPA/540/F-95/041.

TABLE 4.9c RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Utility Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Utility Worker	Adult > 18 years	EU-9	AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9c RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Utility Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs:
				InR	Inhalation Rate	m ³ /hour	1.5	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m ³) =
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	CS / PEF
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	For VOCs:
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	Chemical Concentration in Air (CA, mg/m ³) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CS / VF
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9d RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Construction Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA, 2004; Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Construction Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.3	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	250	USEPA, 2004; Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9d RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name (1)
Inhalation	Construction Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	3.2	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	8.89E+05	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9e RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas:	State Wetland SYW-12
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Utility Worker	Adult > 18 years	EU-9	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	DAD (mg/kg-day) =
				CF	Unit Conversion Factor for Water	mg/μg	0.000001	Unit Conversion	$DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Where DA_{event} (Organics) =
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	$\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	$\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Where DA_{event} (Inorganics) = $DA_{event} = K_P \times C_W \times t_{event}$
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9f RME
VALUES USED FOR DAILY INTAKE CALCULATIONS
REASONABLE MAXIMUM EXPOSURE - EXPOSURE UNIT 9, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	RME Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Construction Worker	Adult > 18 years	EU-9	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$DAD \text{ (mg/kg-day)} =$ $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg/μg	0.000001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.10 RME - Supplement B.
AGE DEPENDENT ADJUSTMENT FACTOR - EXPOSURE PARAMETERS (RME)
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

AGE (year)	ED (years)	BW [1] (kg)	INTAKE RATE			DERMAL				AGE-ADJUSTED EXPOSURE PARAMETERS							
			WATER IR-W [2] (L/day)	SOIL IR-S [3] (mg/day)	AIR IN [4] (m3/hr)	RESIDENT SSAF [7] (mg/cm2-day)	RECREATOR / TRESPASSER SSAF [7] (mg/cm2-day)	EXPOSED SA-E [8] (cm2)	TOTAL SA-T [9] (cm2)	AGE GROUP	IR-S-Adj (mg-yr/day-kg)	IR-W-Adj (liter-yr/day-kg)	IN-Adj (m3-yr/hr-kg)	SSAF-Adj (mg-yr/day-kg) [5]	SSAF-Adj (mg-yr/day-kg) [6]	ESA-Adj ([cm2-yr]/kg)	TSA-Adj ([cm2-yr]/kg)
0	1	9.1	0.76	200	0.188	0.2	3	2,625	5,910	0-<2 yrs	39.7	0.22	0.046	103	1548	516	1,172
1	1	11.3	1.5	200	0.283	0.2	3	2,571	5,910	2-<6 yrs	49.8	0.37	0.081	144	2159	720	1,685
2	1	13.3	1.5	200	0.283	0.2	3	2,434	5,910								
3	1	15.3	1.5	200	0.346	0.2	3	2,893	6,565								
4	1	17.4	1.5	200	0.346	0.2	3	3,175	7,185								
5	1	19.7	1.5	200	0.346	0.2	3	3,255	7,860								
6	1	22.6	1.5	100	0.417	0.2	3	2,949	8,545								
7	1	24.9	1.5	100	0.417	0.2	3	3,182	9,265								
8	1	28.1	1.5	100	0.417	0.2	3	3,434	10,000								
9	1	31.5	1.5	100	0.563	0.2	3	3,657	10,650								
10	1	36.3	1.5	100	0.563	0.2	3	3,819	11,750								
11	1	41.1	2	100	0.563	0.2	3	4,111	12,650	12-<16yrs	7.7	0.15	0.044	39	1144	381	1,159
12	1	45.3	2	100	0.563	0.2	3	4,453	13,700								
13	1	50.4	2	100	0.563	0.07	3	4,916	14,750								
14	1	56	2	100	0.563	0.07	3	5,205	15,800								
15	1	58.1	2	100	0.604	0.07	3	5,386	16,350								
16	1	62.6	2	100	0.604	0.07	3	5,534	16,800	16-<18 yrs	3.2	0.06	0.019	12.4	533.0	177.7	540
17	1	63.2	2	100	0.604	0.07	3	5,641	17,150								

Equations:

$$\begin{aligned} \text{IR-S-Adj (mg-yr/day-kg)} &= \sum (\text{ED} * \text{IR-S}) / \text{BW} \\ \text{IR-W-Adj (liter-yr/day-kg)} &= \sum (\text{ED} * \text{IR-W}) / \text{BW} \\ \text{SSAF-Adj (mg-yr/day-kg)} &= \sum (\text{ED} * \text{EV} * \text{SSAF} * \text{SA}) / \text{BW} \\ \text{IN-Adj (m3-yr/hour-kg)} &= \sum (\text{ED} * \text{IN}) / \text{BW} \\ \text{ESA-Adj ([cm2-yr]/kg)} &= \sum (\text{ED} * \text{SA-E}) / \text{BW} \\ \text{TSA-Adj ([cm2-yr]/kg)} &= \sum (\text{ED} * \text{SA-T}) / \text{BW} \end{aligned}$$

Footnotes:

- [1] EPA 1997. Exposure Factors Handbook. Tables 7-2 (adults) and 7-3 (children), mean. Values are mean of male and female. Source: National Center of Health Statistics (NCHS) 1987.
- [2] EPA 1997. Exposure Factors Handbook. Table 3-30 - Summary of Recommended Drinking Water Intake Rates. 95th Percentile (90th Percentile was used when 95th Percentile is not listed).
- [3] EPA 1991. Standard Default Exposure Factors. Default for resident child and adult.
- [4] EPA 1997. Exposure Factors Handbook. Table 5-23 - Summary of Recommended Values for Inhalation. Mean of male and female. Values were given as (m³ / day) and were then divided by 24 hours to give an hourly rate.
- [5] SSAF-Adj to be used with the Child Resident exposed to surface soils. Derived from the Soil SSAF.
- [6] SSAF-Adj to be used with the Older Child Trespasser and Child Recreator exposed to surface soils and sediment. Derived from the Sediment SSAF.
- [7] EPA 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Recommended default adherence factor for a child resident (0.2) and adult resident (0.07). For older children, the geometric mean weighted adherence factor for children playing in wet soil of was used for children 6 - 12, as a central tendency estimate of a high end soil contact activity (see Exhibit 3-3).
- [8] EPA 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Calculated from Exhibit C-1 - Body Part-Specific Surface Area Calculations (Children). Data from Exposure Factors Handbook, Tables 6-6, 6-7 and 6-8. Surface area of head, forearms, hands, lower legs and feet (for child <6 years); feet excluded from surface area calculation for >6 years. Surface area for >18 is recommended default for adult resident (EPA 2004).
- [9] EPA 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Exhibit C-1 - Body Part-Specific Surface Area Calculations (Children). Data from Exposure Factors Handbook, Tables 6-6 and 6-7. Total Body Surface Area (50th % tile).

RAGS Table 4 CT Series

TABLE 4.1a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5400	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = $CA \times InR \times ET \times EF \times ED \times 1/BW \times 1/AT$
				InR	Inhalation Rate	m ³ /hour	1.2	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	1.37E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Trespasser	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Trespasser	Adult > 18 years	EU-1	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.15	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Adult Trespasser	Adult > 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	1.37E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1a CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutagenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future		
Exposure Areas:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area*		
Medium:	Soil		
Exposure Medium:	Surface Soil	(0 - 2 ft)	

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	CT Value	Units	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (12-<16)	Age Adjusted Ingestion Rate of Soil (12-<16 yrs)	mg-yr/day-kg	3.8	calculated	CS x IR-S-Adj (12-<16) x EF x CF x 1/AT
				FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	[for child aged 12-<16 years]
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (16-<18)	Age Adjusted Ingestion Rate of Soil (16-<18 yrs)	mg-yr/day-kg	1.6	calculated	CS x IR-S-Adj (16-<18) x EF x CF x 1/AT
		Older Child 16 to < 18 years	EU-1	FI	Fraction Ingested from Soil	unitless	1.0	Best Professional Judgement	[for child aged 16-<18 years]
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				SSAF-Adj (12-<16)	Age Adjusted Soil to Skin Adherence Factor (12-<16 yrs)	mg-yr/day-kg	6.8	calculated	CS x SSAF-Adj (12-<16) x DABS x CF x EF x 1/AT
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 12-<16 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				SSAF-Adj (16-<18)	Age Adjusted Soil to Skin Adherence Factor (16-<18 yrs)	mg-yr/day-kg	1.81	calculated	CS x SSAF-Adj (16-<18) x DABS x CF x EF x 1/AT
		Older Child 16 to < 18 years	EU-1	DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 16-<18 years]
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	CA x IN-Adj (12-<16) x ET x EF x 1/AT
				IN-Adj-(12-<16)	Age Adjusted Inhalation Rate (12-<16 yrs)	m ³ -yr/day-kg	1.1	calculated	[for child aged 12-<16 years]
				PEF	Particulate Emission Factor	m ³ /kg	calculated	USEPA, 2002 (1)	
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	EPA, 1996	CA (mg/m ³) = CS (1/PEF + 1/VF)
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
		Older Child 16 to < 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	CA x IN-Adj (16-<18) x ET x EF x 1/AT
				IN-Adj-(16-<18)	Age Adjusted Inhalation Rate (0-<16-<18 yrs)	m ³ -yr/day-kg	0.42	calculated	[for child aged 16-<18 years]
				PEF	Particulate Emission Factor	m ³ /kg	calculated	USEPA, 2002 (1)	
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	EPA, 1996	CA (mg/m ³) = CS (1/PEF + 1/VF)
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

TABLE 4.1a CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future	
Exposure Areas:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area*	
Medium:	Soil	
Exposure Medium:	Surface Soil	(0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	CT Value	Units	Rationale/Reference	Intake Equation/Model Name
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USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Utility Worker	Adult > 18 years	EU-1	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Utility Worker	Adult > 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	1.5	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	1.37E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1c CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Construction Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Construction Worker	Adult > 18 years	EU-1	AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.1	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1c CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name (1)
Inhalation	Construction Worker	Adult > 18 years	EU-1	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	3.2	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	8.72E+05	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1d CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Utility Worker	> 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1e CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Construction Worker	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	DAD (mg/kg-day) =
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	$DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Where DA_{event} (Organics) =
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	$\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	$\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Where DA_{event} (Inorganics) =
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	$DA_{event} = K_P \times C_W \times t_{event}$
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1f CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	100	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Sediment	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day		NYSDEC 2002, Onondaga Lake HHRA	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.2	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1f CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Trespasser	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Sediment	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Trespasser	Adult > 18 years	EU-1	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.15	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1f CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Utility Worker	Adult > 18 years	EU-1	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1f CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area*
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (12-<16)	Age Adjusted Ingestion Rate of Soil (12-<16 yrs)	mg-yr/day-kg	3.8	calculated	CS x IR-S-Adj (12-<16) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	Best Professional Judgement
				CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (16-<18)	Age Adjusted Ingestion Rate of Soil (16-<18 yrs)	mg-yr/day-kg	1.59	calculated	CS x IR-S-Adj (16-<18) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	[for child aged 16-<18 years]
				CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				SSAF-Adj (12-<16)	Age Adjusted Soil to Skin Adherence Factor (12-<16 yrs)	mg-yr/day-kg	6.8	calculated	CS x SSAF-Adj (12-<16) x DABS x CF x EF x 1/AT
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 12-<16 years]
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Older Child 16 to < 18 years	EU-1	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				SSAF-Adj (16-<18)	Age Adjusted Soil to Skin Adherence Factor (16-<18 yrs)	mg-yr/day-kg	1.81	calculated	CS x SSAF-Adj (16-<18) x DABS x CF x EF x 1/AT
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 16-<18 years]
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

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TABLE 4.1g CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SEDIMENT & SUBSURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface & Subsurface Sediment (0 - 10 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Utility Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Where construction or utility workers have may contact with the sediment of Harbor Brook, a depth interval of 0 - 10 ft bgs is applied. This reflects the potential for contact with deeper sediments for bridge reconstruction, which is anticipated and unique to the Harbor Brook exposure area. In a few instances, sediment samples with start depths of 0 ft and end depths ranging from >1 to 3 ft were also incorporated in the evaluation of surface sediment.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1h CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE SEDIMENT & SUBSURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Sediment
Exposure Medium:	Surface & Subsurface Sediment (0 - 10 ft bgs)**

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Construction Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Construction Worker	Adult > 18 years	EU-1	CS	Chemical Concentration in Subsurface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.1	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Where construction or utility workers have may contact with the sediment of Harbor Brook, a depth interval of 0 - 10 ft bgs is applied. This reflects the potential for contact with deeper sediments for bridge reconstruction, which is anticipated and unique to the Harbor Brook exposure area. In a few instances, sediment samples with start depths of 0 ft and end depths ranging from >1 to 3 ft were also incorporated in the evaluation of surface sediment.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1i CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	5400	NYSDEC 2002, Onondaga Lake HHRA	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	2	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
	Adult Trespasser	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	5700	USEPA 2004, Exhibit C-1; NYSDEC 2002, Onondaga Lake HHRA	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	2	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.1i CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Utility Worker	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K_p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t_{event}	Event Duration	hr/event	8	Best Professional Judgment	
				τ_{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t^*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1i CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 1, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area*
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Older Child Trespasser	Older Child 12 to < 16 years	EU-1	DA-Adj	Dermally Absorbed Dose per Event	mg/cm ² -event	calculated	calculated	CDI (mg/kg-day) = DAevent x SA-Adj (12-<16) x EV x EF x 1/AT [for child aged 12-<16 years]
				SA-Adj (12-<16)	Skin Surface Area, Age-adjusted (12-<16 yrs)	cm ² -year/kg	1158.6	calculated	
				EV	Event Frequency	events/day	0.33	EPA, 2004	
				t _{event}	Event Time (0-<6 yrs)	hr/event	1	EPA, 2004	
				EF	Exposure Frequency	days/year	125	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
	Older Child Trespasser	Older Child 16 to < 18 years	EU-1	DA-Adj	Dermally Absorbed Dose per Event	mg/cm ² -event	calculated	calculated	CDI (mg/kg-day) = DAevent x SA-Adj (16-<18) x EV x EF x 1/AT [for child aged 16-<18 years]
				SA-Adj (16-<18)	Skin Surface Area, Age-adjusted (16-<18 yrs)	cm ² -year/kg	572.35	calculated	
				EV	Event Frequency	events/day	0.33	EPA, 2004	
				t _{event}	Event Time (0-<6 yrs)	hr/event	1	EPA, 2004	
				EF	Exposure Frequency	days/year	125	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1j CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1 - SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Construction Worker	Adult > 18 years	EU-1	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} =$ $\text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	250	Best Professional Judgment	
				ED	Exposure Duration	years	1	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.1k CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 1, FISH TISSUE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Site Wide: Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1, AOS #2, Penn-Can Property, and Railroad Area
Medium:	Fish Tissue
Exposure Medium:	Onondaga Lake Fish Tissue

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Older Child Trespasser	Older Child 12 to < 18 years	EU-1	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	$\text{Chronic Daily Intake (CDI)} (\text{mg/kg-day}) = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} (\text{mg/kg-day}) = C \times [1-CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion	
				IR	Ingestion Rate of Fish Tissue	g fish/day	5.3	USEPA 1997; Page 10-26	
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	0.33	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	56	USEPA 1997; Table 7-3	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Ingestion	Adult Trespasser	Adult > 18 years	EU-1	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	$\text{Chronic Daily Intake (CDI)} (\text{mg/kg-day}) = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} (\text{mg/kg-day}) = C \times [1-CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion	
				IR	Ingestion Rate of Fish Tissue	g fish/day	8	USEPA 1997; Page 10-26	
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	0.33	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

1 = Used to adjust exposure point concentration (EPC) for PCBs and PCDD/PCDFs ingested for central tendency (CT) only. NA indicates Not Applicable to the RME scenario.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 2 Food Ingestion Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.2 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 2, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, and DSA #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Surveillance Worker	Adult > 18 years	EU-2	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	37	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Surveillance Worker	Adult > 18 years	EU-2	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	1930	USEPA 2004, Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.01	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency**	days/year	37	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.2 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 2, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, and DSA #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Surveillance Worker	Adult > 18 years	EU-2	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs:
				InR	Inhalation Rate	m ³ /hour	1	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m3) =
				PEF	Particulate Emission Factor	m ³ /kg	3.44E+09	See Appendix F	CS / PEF
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	For VOCs:
				EF	Exposure Frequency**	days/year	37	Best Professional Judgment	Chemical Concentration in Air (CA, mg/m3) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CS / VF
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Value is based on surveillance once per week, factoring in two weeks of vacation annually and reduction of 25% due to snow cover (rounded up from 24.69%). The number of days of work that exposure is reduced are rounded to nearest whole day, see HHRA text for derivation.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.3a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 3, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Interstate 690 Drainage Ditch
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Drainage Ditch Worker	Adult > 18 years	EU-3	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	330	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Sediment	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Drainage Ditch Worker	Adult > 18 years	EU-3	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004, Exhibit 3-5	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.3a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 3, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Interstate 690 Drainage Ditch
Medium:	Sediment
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Drainage Ditch Worker	Adult > 18 years	EU-3	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For VOCs:
				InR	Inhalation Rate	m ³ /hour	1.5	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m3) =
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	CS / VF
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	10	Best Professional Judgment	Chronic Daily Intake (CDI in mg/kg-day) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CA x InR x ET x EF x ED x 1/BW x 1/AT
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.3b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 3, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Interstate 690 Drainage Ditch
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Drainage Ditch Worker	Adult > 18 years	EU-3	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} = \text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2004, Exhibit C-1	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
Dermal	Drainage Ditch Worker	Adult > 18 years	EU-3	AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.4 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 4, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Railroad Worker	Adult > 18 years	EU-4	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	164	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Railroad Worker	Adult > 18 years	EU-4	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.07	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency**	days/year	164	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.4 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 4, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	Railroad Area
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Railroad Worker	Adult > 18 years	EU-4	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	2.5	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	8.22E+08	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	2	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Value is based on 219 work days per year reduced by 25% due to snow cover (rounded up from 24.69%). The number of days of work that exposure is reduced are rounded to nearest whole day, see HHRA text for derivation.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.5 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 5, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current
Exposure Areas*:	Penn-Can Property
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Commercial and Industrial Worker	Adult > 18 years	EU-5	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1991; Section 6.0 Summary Table	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	219	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Commercial and Industrial Worker	Adult > 18 years	EU-5	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.1	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	219	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.5 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 5, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current
Exposure Areas*:	Penn-Can Property
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Commercial and Industrial Worker	Adult > 18 years	EU-5	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs:
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m3) =
				PEF	Particulate Emission Factor	m ³ /kg	5.89E+08	See Appendix F	CS / PEF
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	For VOCs:
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	Chemical Concentration in Air (CA, mg/m3) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CS / VF
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Recreator	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Recreator	Adult > 18 years	EU-6	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004, Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.15	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Adult Recreator	Adult > 18 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.8	USEPA 1997, Table 5-11	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	16	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Ingestion	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Recreator	Younger Child 0 to < 6 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Metals & VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = $CA \times InR \times ET \times EF \times ED \times 1/BW \times 1/AT$
				InR	Inhalation Rate	m ³ /hour	1	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	2	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Resident	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Resident	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004, Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.01	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Adult Resident	Adult > 18 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.8	USEPA 1997, Table 5-11	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	16	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
Ingestion	Child Resident	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Resident	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.04	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Child Resident	Younger Child 0 to < 6 years	EU-6	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	0.42	USEPA 1997, Table 5-11	
				InR	Inhalation Rate	m ³ /hour	1	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	3.97E+09	See Appendix F	For VOCs: Chemical Concentration in Air (CA, mg/m ³) = CS / VF
				ET	Exposure Time	hours/day	24	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6a CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated Best Professional Judgement Unit Conversion USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg-day) = CS \times IR-S-Adj (0-<2) \times EF \times CF \times 1/AT$ [for child aged 0-<2 years]
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	19.8		
				EF	Exposure Frequency	days/year	32		
				CF	Conversion Factor 1	kg/mg	0.000001		
				AT-C	Averaging Time - Cancer	days	25,550		
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated Best Professional Judgement Unit Conversion USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg-day) = CS \times IR-S-Adj (12-<16) \times EF \times CF \times 1/AT$ [for child aged 2-<6 years]
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	24.9		
				EF	Exposure Frequency	days/year	32		
				CF	Conversion Factor 1	kg/mg	0.000001		
				AT-C	Averaging Time - Cancer	days	25,550		
Dermal	Child Recreator	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated USEPA, 2004; Exhibit 3-4 Unit Conversion Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg-day) = CS \times SSAF-Adj (0-<2) \times DABS \times CF \times EF \times 1/AT$ [for child aged 0-<2 years]
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	21		
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific		
				CF	Conversion Factor	kg/mg	0.000001		
				EF	Exposure Frequency	days/year	32		
				AT-C	Averaging Time - Cancer	days	25,550		
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated USEPA, 2004; Exhibit 3-4 Unit Conversion Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg-day) = CS \times SSAF-Adj (2-<6) \times DABS \times CF \times EF \times 1/AT$ [for child aged 2-<6 years]
				SSAF-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	29		
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific		
				CF	Conversion Factor	kg/mg	0.000001		
				EF	Exposure Frequency	days/year	32		
				AT-C	Averaging Time - Cancer	days	25,550		
Inhalation	Child Recreator	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated USEPA, 2002 (1) EPA, 1996 Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg-day) = CA \times IN-Adj (0-<2) \times ET \times EF \times 1/AT$ [for child aged 0-<2 years]
				CA	Chemical Concentration in Air	mg/m ³	See Table 3		
				IN-Adj (0-<2)	Age Adjusted Inhalation Rate (0-<2 yrs)	m ³ -yr/day-kg	1.1		
				PEF	Particulate Emission Factor	m ³ /kg	calculated		
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CA (mg/m^3) = CS (1/PEF + 1/VF)$
				EF	Exposure Frequency	days/year	32		
				AT-C	Averaging Time - Cancer	days	25,550		
				CS	Chemical Concentration in Soil	mg/kg	See Table 3		
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series calculated USEPA, 2002 (1) EPA, 1996 Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg-day) = CA \times IN-Adj (2-<6) \times ET \times EF \times 1/AT$ [for child aged 2-<6 years]
				IN-Adj (2-<6)	Age Adjusted Inhalation Rate (2-<6 yrs)	m ³ -yr/day-kg	2		
				PEF	Particulate Emission Factor	m ³ /kg	calculated		
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated		
				EF	Exposure Frequency	days/year	32	Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CA (mg/m^3) = CS (1/PEF + 1/VF)$
				AT-C	Averaging Time - Cancer	days	25,550		

TABLE 4.6a CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Resident	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated Best Professional Judgement Unit Conversion USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg\text{-}day) = CS \times IR\text{-}S\text{-}Adj (0\text{-}<2) \times EF \times CF \times 1/AT$ [for child aged 0-<2 years]
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	19.8		
				EF	Exposure Frequency	days/year	32		
				CF	Conversion Factor 1	kg/mg	0.000001		
				AT-C	Averaging Time - Cancer	days	25,550		
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated Best Professional Judgement Unit Conversion USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg\text{-}day) = CS \times IR\text{-}S\text{-}Adj (2\text{-}<6) \times EF \times CF \times 1/AT$ [for child aged 2-<6 years]
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	24.9		
				EF	Exposure Frequency	days/year	32		
				CF	Conversion Factor 1	kg/mg	0.000001		
				AT-C	Averaging Time - Cancer	days	25,550		
Dermal	Child Resident	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated USEPA, 2004; Exhibit 3-4 Unit Conversion Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg\text{-}day) = CS \times SSAF\text{-}Adj (0\text{-}<2) \times DABS \times CF \times EF \times 1/AT$ [for child aged 0-<2 years]
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	21		
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific		
				CF	Conversion Factor	kg/mg	0.000001		
				EF	Exposure Frequency	days/year	32		
				AT-C	Averaging Time - Cancer	days	25,550		
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated USEPA, 2004; Exhibit 3-4 Unit Conversion Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg\text{-}day) = CS \times SSAF\text{-}Adj (2\text{-}<6) \times DABS \times CF \times EF \times 1/AT$ [for child aged 2-<6 years]
				SSAF-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	29		
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific		
				CF	Conversion Factor	kg/mg	0.000001		
				EF	Exposure Frequency	days/year	32		
				AT-C	Averaging Time - Cancer	days	25,550		
Inhalation	Child Resident	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated USEPA, 2002 (1) EPA, 1996 Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg\text{-}day) = CA \times IN\text{-}Adj (0\text{-}<2) \times ET \times EF \times 1/AT$ [for child aged 0-<2 years]
				CA	Chemical Concentration in Air	mg/m ³	See Table 3		
				IN-Adj (0-<2)	Age Adjusted Inhalation Rate (0-<2 yrs)	m ³ -yr/day-kg	1.1		
				PEF	Particulate Emission Factor	m ³ /kg	calculated		
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CA (mg/m^3) = CS (1/PEF + 1/VF)$
				EF	Exposure Frequency	days/year	32		
				AT-C	Averaging Time - Cancer	days	25,550		
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series calculated USEPA, 2002 (1) EPA, 1996 Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CDI (mg/kg\text{-}day) = CA \times IN\text{-}Adj (2\text{-}<6) \times ET \times EF \times 1/AT$ [for child aged 2-<6 years]
				CA	Chemical Concentration in Air	mg/m ³	See Table 3		
				IN-Adj (2-<6)	Age Adjusted Inhalation Rate (2-<6 yrs)	m ³ -yr/day-kg	2		
				PEF	Particulate Emission Factor	m ³ /kg	calculated		
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	Best Professional Judgement USEPA 1989, Exhibits 6-11 through 6-16	$CA (mg/m^3) = CS (1/PEF + 1/VF)$
				EF	Exposure Frequency	days/year	32		
				AT-C	Averaging Time - Cancer	days	25,550		

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Recreator	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Sediment	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Recreator	Adult > 18 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004, Exhibit C-1	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.15	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Soil
Exposure Medium:	Surface Sediment (0 - 1 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Sediment	mg/day	100	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Sediment	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-6	CS	Chemical Concentration in Surface Sediment	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Sediment	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Sediment to Skin Adherence Factor	mg/cm ²	0.2	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

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NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

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USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

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TABLE 4.6b CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE SEDIMENT
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Soil
Exposure Medium:	Surface Sediment (0 - 1 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	19.8	calculated	CS x IR-S-Adj (0-<2) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	[for child aged 0-<2 years]
				CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2-<6 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	24.9	calculated	CS x IR-S-Adj (2-<6) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	[for child aged 2-<6 years]
				CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 0-<2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	21	calculated	CS x SSAF-Adj (0-<2) x DABS x CF x EF x 1/AT
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 0-<2 years]
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2-<6 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	29	calculated	CS x SSAF-Adj (2-<6) x DABS x CF x EF x 1/AT
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 2-<6 years]
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6c CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Adult Recreator	Adult > 18 years	EU-6	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} =$ $\text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				K _p	PeCTability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004, Exhibit C-1	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	2	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
Dermal	Adult Recreator	Adult > 18 years	EU-6	AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.6c CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-6	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$DAD \text{ (mg/kg-day)} =$ $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \frac{\left(\frac{1+3B+3B^2}{(1+B)^2} \right)}{\right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = K_P \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	2800	NYSDEC 2002, Onondaga Lake HHRA	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	2	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound absorbed to the skin versus relative to the KP	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6c CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 6, SURFACE WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Water
Exposure Medium:	Surface Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Child Recreator	Younger Child 0-<2 years	EU-6	DA-Adj	Dermally Absorbed Dose per Event	mg/cm ² -event	calculated	calculated	CDI (mg/kg-day) = DAevent x SA-Adj (0-<2) x EV x EF x 1/AT [for child aged 0-<2 years]
				SA-Adj (0-<2)	Skin Surface Area, Age-adjusted (0-<2 yrs)	cm ² -year/kg	1,172	calculated	
				EV	Event Frequency	events/day	0.33	EPA, 2004	
				t _{event}	Event Time (0-<6 yrs)	hr/event	1	EPA, 2004	
				EF	Exposure Frequency	days/year	125	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2-<6 years	EU-6	DA-Adj	Dermally Absorbed Dose per Event	mg/cm ² -event	calculated	calculated	CDI (mg/kg-day) = DAevent x SA-Adj (2-<6) x EV x EF x 1/AT [for child aged 2-<6 years]
				SA-Adj (2-<6)	Skin Surface Area, Age-adjusted (2-<6 yrs)	cm ² -year/kg	1,685	calculated	
				EV	Event Frequency	events/day	0.33	EPA, 2004	
				t _{event}	Event Time (0-<6 yrs)	hr/event	1	EPA, 2004	
				EF	Exposure Frequency	days/year	125	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

NYSDEC. 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6d CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 6, FISH TISSUE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, and AOS #1
Medium:	Fish Tissue
Exposure Medium:	Onondaga Lake Fish Tissue

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Adult Recreator	Adult > 18 years	EU-6	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	$\text{Chronic Daily Intake (CDI)} \text{ (mg/kg-day)} = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} \text{ (mg/kg-day)} = C \times [1 - CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion	
				IR	Ingestion Rate of Fish Tissue	g fish/day	8	USEPA 1997; Page 10-26	
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	0.33	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991, Section 6.0 Summary Table	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
	Child Recreator	Younger Child 0 to < 6 years	EU-6	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series	$\text{Chronic Daily Intake (CDI)} \text{ (mg/kg-day)} = C \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$ $\text{Chronic Daily Intake for PCBs and PCDD/PCDFs (CDI)} \text{ (mg/kg-day)} = C \times [1 - CL] \times CF \times IR \times FI \times EF \times ED / (BW \times AT)$
				CF	Unit Conversion Factor for Fish Tissue	kg/g	0.001	Unit Conversion	
				IR	Ingestion Rate of Fish Tissue	g fish/day	2.7	USEPA 1997; Page 10-26	
				CL	Cooking Loss (PCBs and PCDD/PCDFs only) ¹	unitless	0.33	USEPA 1997; Section 10.9	
				EF	Exposure Frequency	days/year	365	USEPA 1997; Page 10-26	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Attachment B	
				FI	Fraction Ingested of Fish Tissue	unitless	1	Best Professional Judgment	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

1 = Used to adjust exposure point concentration (EPC) for PCBs and PCDD/PCDFs ingested for central tendency (CT) only. NA indicates Not Applicable to the RME scenario.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 2 Food Ingestion Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.6d CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 6, FISH TISSUE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Harbor Brook, Lakeshore Area, East Flume, DSA #1, DSA #2, AOS #1*
Medium:	Fish Tissue
Exposure Medium:	Onondage Lake Fish Tissue

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 2 years	EU-6	C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series calculated USEPA 1997; Page 10-26 Unit Conversion USEPA 1989, Exhibits 6-11 through 6-16	CDI (mg/kg-day) = CW x IR-FT-Adj (0-<2) x EF x CF x 1/AT [for child aged 0-<2 years]
				IR-FT-Adj (0-<2)	Ingestion Rate of Fish, Age-adjusted (0-<2 yrs)	liter-year/kg-day	0.79		
				EF	Exposure Frequency	days/year	365		
				CF	Unit Conversion Factor for Fish Tissue	mg/µg	0.001		
	Child Recreator	Younger Child 2 to < 6 years	EU-6	AT-C	Averaging Time - Cancer	days	25550		
				C	Chemical Concentration in Fish	mg/kg (wet)	See Table 3	See RAGS Table 3 Series calculated USEPA 1997; Page 10-26 Unit Conversion USEPA 1989, Exhibits 6-11 through 6-16	CDI (mg/kg-day) = CW x IR-FT-Adj (2-<6) x EF x CF x 1/AT [for child aged 2-<6 years]
				IR-FT-Adj (2-<6)	Ingestion Rate of Fish, Age-adjusted (2-<6 yrs)	liter-year/kg-day	1.00		
				EF	Exposure Frequency	days/year	365		
				CF	Unit Conversion Factor for Fish Tissue	mg/µg	0.001		
				AT-C	Averaging Time - Cancer	days	25550		

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

United States Environmental Protection Agency (USEPA). 1997. Exposure Factors Handbook - Volume 2 Food Ingestion Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

TABLE 4.7 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 7, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Penn-Can Property, Lakeshore Area, East Flume, DSA #A, DSA #2, AOS #1, and AOS #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Commercial and Industrial Worker	Adult > 18 years	EU-7	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1991; Section 6.0 Summary Table	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	219	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Commercial and Industrial Worker	Adult > 18 years	EU-7	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.1	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	219	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.7 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 7, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Penn-Can Property, Lakeshore Area, East Flume, DSA #A, DSA #2, AOS #1, and AOS #2
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Commercial and Industrial Worker	Adult > 18 years	EU-7	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	1.74E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1991. Risk Assessment Guidance for Superfund Volume 1, Human Health Supplemental Guidance Standard Default Exposure Factors. OSWER Directive 9285.6-03. March 25, 1991.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.8 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 8, POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Resident	Adult > 18 years	EU-8	CW	Chemical Concentration in Potable Water	ug/L	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CW x CF x IR x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				IR	Ingestion Rate of Water	L/day	2	USEPA 1989; Exhibit 6-11	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
		Child 0 to < 6 years	EU-8	AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	CDI (mg/kg-day) = CW x CF x IR x EF x ED x 1/BW x 1/AT
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				CW	Chemical Concentration in Potable Water	ug/L	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				IR	Ingestion Rate of Water	L/day	1	USEPA 1989; Exhibit 6-11	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
	Commercial and Industrial Worker	Adult > 18 years	EU-8	CW	Chemical Concentration in Potable Water	ug/L	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) = CW x CF x IR x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	
				IR	Ingestion Rate of Water	L/day	2	USEPA 1989; Exhibit 6-11	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.8 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 8, POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Resident	Adult > 18 years	EU-8 Shower/Bathing	CW	Chemical Concentration in Water	mg/L	Appendix D	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} =$ $\text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _P	PeCTability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	18000	USEPA, 2004; Exhibit 3-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	0.25	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Resident	Child 0 to < 6 years	EU-8 Shower/Bathing	AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
				CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$\text{DAD (mg/kg-day)} =$ $\text{DA}_{\text{event}} \times \text{CF} \times \text{EV} \times \text{ED} \times \text{EF} \times \text{SA} \times 1/\text{BW} \times 1/\text{AT}$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{\text{event}} \leq t^*, \text{ then } \text{DA}_{\text{event}} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{\text{event}} \times t_{\text{event}}}{\pi}}$ $\text{If } t_{\text{event}} > t^*, \text{ then } \text{DA}_{\text{event}} = FA \times K_P \times C_W \left[\frac{t_{\text{event}}}{1+B} + 2\tau_{\text{event}} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $\text{DA}_{\text{event}} = K_P \times C_W \times t_{\text{event}}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _P	PeCTability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	6600	USEPA, 2004; Exhibit 3-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	0.33	USEPA, 2004; Exhibit 3-2	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.8 CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 8, POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Resident	Adult > 18 years	EU-8 Shower/Bathing	CA	Chemical Concentration in Air	mg/m ³	Calculated	Schaum et al. 1994	Chronic Daily Intake (CDI in mg/kg-day) = $CA \times \ln R \times ET \times EF \times ED \times 1/BW \times 1/AT$
				lnR	Inhalation Rate	m ³ /hour	0.8	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	0.58	Schaum et al. 1994	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-N	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
		Child (Ages 0 - 6)	EU-8 Shower/Bathing	CA	Chemical Concentration in Air	mg/m ³	Calculated	Schaum et al. 1994	Chronic Daily Intake (CDI in mg/kg-day) = $CA \times \ln R \times ET \times EF \times ED \times 1/BW \times 1/AT$
				lnR	Inhalation Rate	m ³ /hour	0.42	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	1	Schaum et al. 1994	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-N	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

Schaum, J., K. Hoang, R. Kinerson, J. Moya, and R.G.M. Wang. 1994. Estimating Dermal and Inhalation Exposure to volatile Chemicals in Domestic Water. USEPA Region II.

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TABLE 4.8 CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 8: POTABLE GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	Site Wide Ground Water (All Depths)
Medium:	Ground Water
Exposure Medium:	Potable Water

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Resident	Child (Ages 0-2)	EU-8	CW	Chemical Concentration in Water	µg/l	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-W-Adj (0-<2)	Ingestion Rate of Water, Age-adjusted (0-<2 yrs)	liter-year/kg-day	0.22	calculated	CW x IR-W-Adj (0-<2) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	[for child aged 0-<2 years]
				CF	Unit Conversion Factor for Water	mg/µg	0.001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Child (Ages 2-6)	EU-8	CW	Chemical Concentration in Water	µg/l	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-W-Adj (2-<6)	Ingestion Rate of Water, Age-adjusted (2-<6 yrs)	liter-year/kg-day	0.37	calculated	CW x IR-W-Adj (2-<6) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	[for child aged 2-<6 years]
				CF	Unit Conversion Factor for Water	mg/µg	0.001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Resident	Child (Ages 0-2)	EU-8 Bath	DA-Adj	Dermally Absorbed Dose per Event	mg/cm ² -event	calculated	calculated	CDI (mg/kg-day) =
				SA-Adj (0-<2)	Skin Surface Area, Age-adjusted (0-<2 yrs)	cm ² -year/kg	1,172	calculated	DAevent x SA-Adj (0-<2) x EV x EF x 1/AT
				EV	Event Frequency	events/day	0.33	EPA, 2004	[for child aged 0-<2 years]
				t _{event}	Event Time (0-<6 yrs)	hr/event	1	EPA, 2004	
				EF	Exposure Frequency	days/year	125	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Child (Ages 2-<6)	EU-8 Bath	DA-Adj	Dermally Absorbed Dose per Event	mg/cm ² -event	calculated	calculated	CDI (mg/kg-day) =
				SA-Adj (2-<6)	Skin Surface Area, Age-adjusted (2-<6 yrs)	cm ² -year/kg	1,685	calculated	DAevent x SA-Adj (2-<6) x EV x EF x 1/AT
				EV	Event Frequency	events/day	0.33	EPA, 2004	[for child aged 2-<6 years]
				t _{event}	Event Time (0-<6 yrs)	hr/event	1	EPA, 2004	
				EF	Exposure Frequency	days/year	125	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

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TABLE 4.9a CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) = CS x IR-S-Adj (0-<2) x EF x CF x 1/AT [for child aged 0-<2 years]
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	19.8	calculated	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	
Ingestion	Child Recreator	Younger Child 2 to < 6 years	EU-6	AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	CDI (mg/kg-day) = CS x IR-S-Adj (2-<6) x EF x CF x 1/AT [for child aged 2-<6 years]
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	24.9	calculated	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
Dermal	Child Recreator	Younger Child 0 to < 2 years	EU-6	CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	CDI (mg/kg-day) = CS x SSFA-Adj (0-<2) x DABS x CF x EF x 1/AT [for child aged 0-<2 years]
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				SSFA-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	21.0	calculated	
Dermal	Child Recreator	Younger Child 2 to < 6 years	EU-6	DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	CDI (mg/kg-day) = CS x SSFA-Adj (2-<6) x DABS x CF x EF x 1/AT [for child aged 2-<6 years]
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) = CS x SSFA-Adj (2-<6) x DABS x CF x EF x 1/AT [for child aged 2-<6 years]
				SSFA-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	29.0	calculated	
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
Dermal	Child Recreator	Younger Child 2 to < 6 years	EU-6	EF	Exposure Frequency	days/year	32	Best Professional Judgement	CDI (mg/kg-day) = CS x SSFA-Adj (2-<6) x DABS x CF x EF x 1/AT [for child aged 2-<6 years]
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				SSFA-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	29.0	calculated	

TABLE 4.9a CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Child Recreator	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	$CDI \text{ (mg/kg-day)} =$ $CA \times IN-Adj \text{ (0-<2)} \times ET \times EF \times 1/AT$ [for child aged 0-<2 years] $CA \text{ (mg/m}^3\text{)} = CS \text{ (1/PEF + 1/VF)}$
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	
				IN-Adj-(0-<2)	Age Adjusted Inhalation Rate (0-<2 yrs)	m ³ -yr/day-kg	1.1	calculated	
				PEF	Particulate Emission Factor	m ³ /kg	calculated	USEPA, 2002 (1)	
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	EPA, 1996	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	$CDI \text{ (mg/kg-day)} =$ $CA \times IN-Adj \text{ (2-<6)} \times ET \times EF \times 1/AT$ [for child aged 2-<6 years] $CA \text{ (mg/m}^3\text{)} = CS \text{ (1/PEF + 1/VF)}$
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	
				IN-Adj-(2-<6)	Age Adjusted Inhalation Rate (2-<6 yrs)	m ³ -yr/day-kg	2.0	calculated	
				PEF	Particulate Emission Factor	m ³ /kg	calculated	USEPA, 2002 (1)	
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	EPA, 1996	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

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TABLE 4.9a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Recreator	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Recreator	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004, Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.15	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Adult Recreator	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Recreator	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	0.5	Best Professional Judgment	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Recreator	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	Per NYSDEC Comment; Letter dated 3/12/2008	
				EF	Exposure Frequency	days/year	32	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Recreator	Younger Child 0 to < 6 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	1.2	USEPA 1997, Table 5-23	
				ET	Exposure Time	hours/day	4	Best Professional Judgment	
				EF	Exposure Frequency	days/year	42	Best Professional Judgment	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Railroad Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	164	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Railroad Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.07	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency**	days/year	164	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9a CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Railroad Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	2.5	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	2	Best Professional Judgment	
				EF	Exposure Frequency**	days/year	188	Best Professional Judgment	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

** = Value is based on 219 work days per year reduced by 25% due to snow cover (rounded up from 24.69%). The number of days of work that exposure is reduced are rounded to nearest whole day, see HHRA text for derivation.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

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USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Adult Resident	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Adult Resident	Adult > 18 years	EU-9	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	5700	NYSDEC 2002, Onondaga Lake HHRA, USEPA 2004, Exhibit C-1	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.01	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-2	
Inhalation	Adult Resident	Adult > 18 years	EU-9	BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
				CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	
				InR	Inhalation Rate	m ³ /hour	0.8	USEPA 1997, Table 5-11	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	16	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	30	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	70	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	10950	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Resident	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 1997; Table 4-23	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Child Resident	Younger Child 0 to < 6 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	2800	NYSDEC 2002, Onondaga Lake HHRA	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.04	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	
Inhalation	Child Resident	Younger Child 0 to < 6 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	0.42	USEPA 1997, Table 5-11	
				ET	Exposure Time	hours/day	24	Best Professional Judgment	
				EF	Exposure Frequency	days/year	350	USEPA, 2004; Exhibit 3-2	
				ED	Exposure Duration	years	6	USEPA, 2004; Exhibit 3-2	
				BW	Body Weight	kg	15	USEPA 1991; Section 6.0 Summary Table	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	2190	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Commercial and Industrial Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = $CS \times CF \times IR \times FI \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	50	USEPA 1991; Section 6.0 Summary Table	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	219	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Commercial and Industrial Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Surface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Dermally Absorbed Dose (DAD, mg/kg-day) = $CS \times CF \times SA \times ABS \times AF \times EF \times ED \times 1/BW \times 1/AT$
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.1	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	219	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9b CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Commercial and Industrial Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	1.6	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA 1991; Section 3.0	
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

New York State Department of Environmental Conservation (NYSDEC). 2002. Onondaga Lake Human Health Risk Assessment. Division of Environmental Remediation. Albany, New York.

NYSDEC. 2008. Comments on Wastebed B/Harbor Brook Site HHRA RAGS Tables 1 through 6 prepared by O'Brien & Gere for Honeywell, dated February 20, 2008. Letter dated March 12, 2008.

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TABLE 4.9b CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Child Resident	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (0-<2)	Age Adjusted Ingestion Rate of Soil (0-<2 yrs)	mg-yr/day-kg	19.8	calculated	CS x IR-S-Adj (0-<2) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	[for child aged 0-<2 years]
				CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				IR-S-Adj (2-<6)	Age Adjusted Ingestion Rate of Soil (2-<6 yrs)	mg-yr/day-kg	24.9	calculated	CS x IR-S-Adj (2-<6) x EF x CF x 1/AT
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	[for child aged 2-<6 years]
				CF	Conversion Factor 1	kg/mg	0.000001	Unit Conversion	
Dermal	Child Resident	Younger Child 0 to < 2 years	EU-6	AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
				SSAF-Adj (0-<2)	Age Adjusted Soil to Skin Adherence Factor (0-<2 yrs)	mg-yr/day-kg	21.0	calculated	CS x SSAF-Adj (0-<2) x DABS x CF x EF x 1/AT
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 0-<2 years]
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
				CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	CDI (mg/kg-day) =
		Younger Child 2 to < 6 years	EU-6	SSAF-Adj (2-<6)	Age Adjusted Soil to Skin Adherence Factor (2-<6 yrs)	mg-yr/day-kg	29.0	calculated	CS x SSAF-Adj (2-<6) x DABS x CF x EF x 1/AT
				DABS	Dermal Absorption Factor Solids	unitless	Chemical-Specific	USEPA, 2004; Exhibit 3-4	[for child aged 2-<6 years]
				CF	Conversion Factor	kg/mg	0.000001	Unit Conversion	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9b CT Supplement A
VALUES USED FOR DAILY INTAKE CALCULATIONS (mutigenic mode of action)
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil (0 - 2 ft)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Child Resident	Younger Child 0 to < 2 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	$CDI (mg/kg-day) = CA \times IN-Adj (0-2) \times ET \times EF \times 1/AT$ [for child aged 0-2 years] $CA (mg/m^3) = CS (1/PEF + 1/VF)$
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	
				IN-Adj-(0-2)	Age Adjusted Inhalation Rate (0-2 yrs)	m ³ -yr/day-kg	1.1	calculated	
				PEF	Particulate Emission Factor	m ³ /kg	calculated	USEPA, 2002 (1)	
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	EPA, 1996	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	
		Younger Child 2 to < 6 years	EU-6	CS	Chemical Concentration in Soil	mg/kg	See Table 3	RAGS Table 3 Series	$CDI (mg/kg-day) = CA \times IN-Adj (2-6) \times ET \times EF \times 1/AT$ [for child aged 2-6 years] $CA (mg/m^3) = CS (1/PEF + 1/VF)$
				CA	Chemical Concentration in Air	mg/m ³	See Table 3	RAGS Table 3 Series	
				IN-Adj-(2-6)	Age Adjusted Inhalation Rate (2-6 yrs)	m ³ -yr/day-kg	2.0	calculated	
				PEF	Particulate Emission Factor	m ³ /kg	calculated	USEPA, 2002 (1)	
				VF	Volatilization Factor for volatile constituents	m ³ /kg	calculated	EPA, 1996	
				EF	Exposure Frequency	days/year	32	Best Professional Judgement	
				AT-C	Averaging Time - Cancer	days	25,550	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

United States Environmental Protection Agency (USEPA). 1996. Soil Screening Guidance: User's Guide. USEPA/540/F-95/041.

United States Environmental Protection Agency (USEPA). 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24.

TABLE 4.9c CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Ingestion	Utility Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	100	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Utility Worker	Adult > 18 years	EU-9	AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.2	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004; Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9c CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Inhalation	Utility Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs:
				InR	Inhalation Rate	m ³ /hour	1.5	USEPA 1997, Table 5-23	Chemical Concentration in Air (CA, mg/m3) =
				PEF	Particulate Emission Factor	m ³ /kg	2.31E+09	See Appendix F	CS / PEF
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	For VOCs:
				EF	Exposure Frequency	days/year	20	Best Professional Judgment	Chemical Concentration in Air (CA, mg/m3) =
				ED	Exposure Duration	years	25	USEPA, 2004, Exhibit 3-5	CS / VF
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	Chronic Daily Intake (CDI in mg/kg-day) =
				AT-NC	Averaging Time - Non-Cancer	days	9125	USEPA 1989, Exhibits 6-11 through 6-16	CA x InR x ET x EF x ED x 1/BW x 1/AT

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9d CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name (1)
Ingestion	Construction Worker	Adult > 18 years	EU-9	CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	Chronic Daily Intake (CDI, mg/kg-day) = CS x CF x IR x FI x EF x ED x 1/BW x 1/AT
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				IR	Ingestion Rate of Soil	mg/day	330	USEPA 2002; Exhibit 1-2	
				FI	Fraction Ingested from Soil	unitless	1	Best Professional Judgment	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
Dermal	Construction Worker	Adult > 18 years	EU-9	AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	Dermally Absorbed Dose (DAD, mg/kg-day) = CS x CF x SA x ABS x AF x EF x ED x 1/BW x 1/AT
				CS	Chemical Concentration in Subsurface Soil	mg/kg	See Table 3	RAGS Table 3 Series	
				CF	Unit Conversion Factor for Soil	kg/mg	0.000001	Unit Conversion	
				ABS	Dermal Absorption Factor	unitless	Chemical Specific	USEPA, 2004; Exhibit 3-4	
				SA	Skin Surface Area for Dermal Absorption	cm ² /day	3300	USEPA 2002; Exhibit 1-2	
				AF	Soil to Skin Adherence Factor	mg/cm ²	0.1	USEPA, 2004; Exhibit 3-3	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

TABLE 4.9d CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SURFACE SOIL & SUBSURFACE SOIL
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Soil
Exposure Medium:	Surface Soil & Subsurface Soil (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name (1)
Inhalation	Construction Worker	Adult > 18 years	EU-9	CA	Chemical Concentration in Air	mg/m ³	Calculated	RAGS Table 3 Series	For Dioxin, Metals, Pesticides, & SVOCs: Chemical Concentration in Air (CA, mg/m3) = CS / PEF For VOCs: Chemical Concentration in Air (CA, mg/m3) = CS / VF Chronic Daily Intake (CDI in mg/kg-day) = CA x InR x ET x EF x ED x 1/BW x 1/AT
				InR	Inhalation Rate	m ³ /hour	3.2	USEPA 1997, Table 5-23	
				PEF	Particulate Emission Factor	m ³ /kg	8.89E+05	See Appendix F	
				VF	Volatilization Factor	m ³ /kg	Chemical Specific	See Appendix E	
				ET	Exposure Time	hours/day	8	Best Professional Judgment	
				EF	Exposure Frequency	days/year	250	USEPA, 2004, Exhibit 3-5	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-NC	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9e CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current / Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Utility Worker	Adult > 18 years	EU-9	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	$DAD \text{ (mg/kg-day)} =$ $DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$ <p>Where DA_{event} (Organics) =</p> $\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times KP \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$ $\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times KP \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$ <p>Where DA_{event} (Inorganics) =</p> $DA_{event} = KP \times C_W \times t_{event}$
				CF	Unit Conversion Factor for Water	mg/μg	0.001	Unit Conversion	
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	5	Best Professional Judgment	
				ED	Exposure Duration	years	9	USEPA, 2004, Exhibit 3-5	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	3285	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.9f CT
VALUES USED FOR DAILY INTAKE CALCULATIONS
CENTRAL TENDENCY - EXPOSURE UNIT 9, SHALLOW GROUND WATER
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Exposure Areas*:	State Wetland SYW-12
Medium:	Water
Exposure Medium:	Shallow Ground Water (0 - 10 ft bgs)

Exposure Route	Receptor Population	Receptor Age	Exposure Point	Parameter Code	Parameter Definition	Units	CT Value	Rationale/Reference	Intake Equation/Model Name
Dermal	Construction Worker	Adult > 18 years	EU-9	CW	Chemical Concentration in Water	mg/L	See Table 3	See RAGS Table 3 Series	DAD (mg/kg-day) =
				CF	Unit Conversion Factor for Water	mg/ug	0.001	Unit Conversion	$DA_{event} \times CF \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$
				K _p	Permeability Constant	cm/hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Where DA_{event} (Organics) =
				SA	Skin Surface Area	cm ²	3300	USEPA 2002; Exhibit 1-2	$\text{If } t_{event} \leq t^*, \text{ then } DA_{event} = 2FA \times K_P \times C_W \sqrt{\frac{6\tau_{event} \times t_{event}}{\pi}}$
				EV	Event Frequency	event/day	1	USEPA 2004, Exhibit A-9	
				EF	Exposure Frequency	days/year	125	Best Professional Judgment	
				ED	Exposure Duration	years	1	Best Professional Judgment	
				BW	Body Weight	kg	70	USEPA 1997; Table 7-11	$\text{If } t_{event} > t^*, \text{ then } DA_{event} = FA \times K_P \times C_W \left[\frac{t_{event}}{1+B} + 2\tau_{event} \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right]$
				FA	Fraction Absorbed	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t _{event}	Event Duration	hr/event	8	Best Professional Judgment	
				t _{event}	Lag Time Per Event	hr/event	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	
				t*	Time to Reach Steady State	hr	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	Where DA_{event} (Inorganics) =
				B	Dimensionless ratio of the KP of a compound through the stratum corneum relative to its KP across the viable epidermis (ve)	unitless	Chemical Specific	USEPA 2004, Exhibits B-3 & B-4	$DA_{event} = K_P \times C_W \times t_{event}$
				AT-C	Averaging Time - Cancer	days	25550	USEPA 1989, Exhibits 6-11 through 6-16	
				AT-N	Averaging Time - Non-Cancer	days	365	USEPA 1989, Exhibits 6-11 through 6-16	

Footnotes:

* = Media evaluated in this table not necessarily present in each exposure area within the exposure unit.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. EPA/540/1089/002.

USEPA. 1997. Exposure Factors Handbook - Volume 1 General Factors. Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa. August 1997.

USEPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response. OSWER Directive 9355.4-24.

USEPA. 2004. Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005. July 2004.

TABLE 4.10 CT - Supplement B.
AGE DEPENDENT ADJUSTMENT FACTOR - EXPOSURE PARAMETERS (CT)
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

AGE (year)	ED (years)	BW [1] (kg)	INTAKE RATE			DERMAL				AGE-ADJUSTED EXPOSURE PARAMETERS							
			WATER IR-W [2] (L/day)	SOIL IR-S [3] (mg/day)	AIR IN [4] (m3/hr)	RESIDENT SSAF [7] (mg/cm2-day)	RECREATOR / TRESPASSER SSAF [7] (mg/cm2-day)	EXPOSED SA-E [8] (cm2)	TOTAL SA-T [9] (cm2)	AGE GROUP	IR-S-Adj (mg-yr/day-kg)	IR-W-Adj (liter-yr/day-kg)	IN-Adj (m3-yr/hr-kg)	SSAF-Adj (mg-yr/day-kg) [5]	SSAF-Adj (mg-yr/day-kg) [6]	ESA-Adj ([cm2-yr]/kg)	TSA-Adj ([cm2-yr]/kg)
0	1	9.1	0.76	100	0.188	0.04	0.2	2,625	5,910	0-<2 yrs	19.8	0.22	0.046	21	103	516	1,172
1	1	11.3	1.5	100	0.283	0.04	0.2	2,571	5,910	2-<6 yrs	24.9	0.37	0.081	29	144	720	1,685
2	1	13.3	1.5	100	0.283	0.04	0.2	2,434	5,910								
3	1	15.3	1.5	100	0.346	0.04	0.2	2,893	6,565								
4	1	17.4	1.5	100	0.346	0.04	0.2	3,175	7,185								
5	1	19.7	1.5	100	0.346	0.04	0.2	3,255	7,860								
6	1	22.6	1.5	50	0.417	0.04	0.2	2,949	8,545								
7	1	24.9	1.5	50	0.417	0.04	0.2	3,182	9,265	12-<16yrs	3.8	0.15	0.044	7	76	381	1,159
8	1	28.1	1.5	50	0.417	0.04	0.2	3,434	10,000								
9	1	31.5	1.5	50	0.563	0.04	0.2	3,657	10,650								
10	1	36.3	1.5	50	0.563	0.04	0.2	3,819	11,750								
11	1	41.1	2	50	0.563	0.04	0.2	4,111	12,650								
12	1	45.3	2	50	0.563	0.04	0.2	4,453	13,700								
13	1	50.4	2	50	0.563	0.01	0.2	4,916	14,750	16-<18 yrs	1.6	0.06	0.019	1.8	35.5	177.7	540
14	1	56	2	50	0.563	0.01	0.2	5,205	15,800								
15	1	58.1	2	50	0.604	0.01	0.2	5,386	16,350								
16	1	62.6	2	50	0.604	0.01	0.2	5,534	16,800								
17	1	63.2	2	50	0.604	0.01	0.2	5,641	17,150								

Equations:

$$\begin{aligned} \text{IR-S-Adj (mg-yr/day-kg)} &= \sum (\text{ED} * \text{IR-S}) / \text{BW} \\ \text{IR-W-Adj (liter-yr/day-kg)} &= \sum (\text{ED} * \text{IR-W}) / \text{BW} \\ \text{SSAF-Adj (mg-yr/day-kg)} &= \sum (\text{ED} * \text{EV} * \text{SSAF} * \text{SA}) / \text{BW} \\ \text{IN-Adj (m3-yr/hour-kg)} &= \sum (\text{ED} * \text{IN}) / \text{BW} \\ \text{ESA-Adj ([cm2-yr]/kg)} &= \sum (\text{ED} * \text{SA-E}) / \text{BW} \\ \text{TSA-Adj ([cm2-yr]/kg)} &= \sum (\text{ED} * \text{SA-T}) / \text{BW} \end{aligned}$$

Footnotes:

- [1] EPA 1997. Exposure Factors Handbook. Tables 7-2 (adults) and 7-3 (children), mean. Values are mean of male and female. Source: National Center of Health Statistics (NCHS) 1987.
- [2] EPA 1997. Exposure Factors Handbook. Table 3-30 - Summary of Recommended Drinking Water Intake Rates. 95th Percentile (90th Percentile was used when 95th Percentile is not listed).
- [3] EPA 1991. Standard Default Exposure Factors. Default for resident child and adult.
- [4] EPA 1997. Exposure Factors Handbook. Table 5-23 - Summary of Recommended Values for Inhalation. Mean of male and female. Values were given as (m³ / day) and were then divided by 24 hours to give an hourly rate.
- [5] SSAF-Adj to be used with the Child Resident exposed to surface soils. Derived from the Soil SSAF.
- [6] SSAF-Adj to be used with the Older Child Trespasser and Child Recreator exposed to surface soils and sediment. Derived from the Sediment SSAF.
- [7] EPA 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Recommended default adherence factor for a child resident (0.2) and adult resident (0.07). For older children, the geometric mean weighted adherence factor for children playing in wet soil of was used for children 6 - 12, as a central tendency estimate of a high end soil contact activity (see Exhibit 3-3).
- [8] EPA 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Calculated from Exhibit C-1 - Body Part-Specific Surface Area Calculations (Children). Data from Exposure Factors Handbook, Tables 6-6, 6-7 and 6-8. Surface area of head, forearms, hands, lower legs and feet (for child <6 years): feet excluded from surface area calculation for >6 years. Surface area for >18 is recommended default for adult resident (EPA 2004).
- [9] EPA 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Exhibit C-1 - Body Part-Specific Surface Area Calculations (Children). Data from Exposure Factors Handbook, Tables 6-6 and 6-7. Total Body Surface Area (50th % tile).

RAGS Table 5 Series

TABLE 5.1
NON-CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD		Oral Absorption Efficiency for Dermal (unitless) (1)	Absorbed RfD for Dermal (2)		Primary Target Organ(s)/Critical Effect(s) (3)	Combined Uncertainty/Modifying Factors (Uncertainty) (Modifying)		RfD:Target Organ(s)	
		Value	Units		Value	Units				Source(s)	Date(s) (MM/DD/YYYY)
DIOXINS											
2,3,7,8-TCDD Equivalent	Chronic	1.0E-09	mg/kg-day	7.0E-01	1.0E-09	mg/kg-day	Developmental effects	90	1	ATSDR (STSC)	12/01/1998
METALS											
ALUMINUM	Chronic	1.0E+00	mg/kg-day	1.00E+00	1.00E+00	mg/kg-day	Neutotoxicology	100	1	PPRTV	10/23/2006
ANTIMONY	Chronic	4.0E-04	mg/kg-day	1.5E-01	6.0E-05	mg/kg-day	Longevity (M); Blood glucose (E); Cholesterol (E)	1000	1	IRIS	02/01/2008
ARSENIC	Chronic	3.0E-04	mg/kg-day	9.5E-01	3.0E-04	mg/kg-day	Hyperpigmentation (In); Vascular (V); PNS (N)	3	1	IRIS	02/01/2008
BARIUM	Chronic	2.0E-01	mg/kg-day	7.0E-02	1.4E-02	mg/kg-day	Humans - none observed (O); Rats - Kidney (R)	3	1	IRIS	02/01/2008
BERYLLIUM	Chronic	2.0E-03	mg/kg-day	7.0E-03	1.4E-05	mg/kg-day	Small intestinal lesions	300	1	IRIS	02/01/2008
CADMIUM	Chronic	1.0E-03	mg/kg-day	2.5E-02	2.5E-05	mg/kg-day	Renal (R); Significant Proteinuria	10	1	IRIS	02/01/2008
CHROMIUM ^a	Chronic	3.0E-03	mg/kg-day	2.5E-02	7.5E-05	mg/kg-day	None Reported (O)	300	3	IRIS (chromium VI as surrogate)	02/01/2008
COBALT	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
COPPER	Chronic	4.0E-02	mg/kg-day	1.0E+00	4.0E-02	mg/kg-day	Gastrointestinal effects	1	1	HEAST (STSC)	06/19/1997
CYANIDE	Chronic	2.0E-02	mg/kg-day	1.0E+00	2.0E-02	mg/kg-day	Weight loss, thyroid effects, myelin degeneration	100	5	IRIS	02/01/2008
IRON	Chronic	7.0E-01	mg/kg-day	1.0E+00	7.0E-01	mg/kg-day	Gastrointestinal effects	2	1	PPRTV	09/11/2006
LEAD	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
MANGANESE	Chronic	1.4E-01	mg/kg-day	4.0E-02	5.6E-03	mg/kg-day	CNS (N)	1	1	IRIS	02/01/2008
MERCURY	Chronic	3.0E-04	mg/kg-day	7.0E-02	2.1E-05	mg/kg-day	Autoimmune effects	1000	1	IRIS (mercuric chloride)	05/01/1995
METHYLMERCURY	Chronic	1.0E-04	mg/kg-day	1.0E+00	1.0E-04	mg/kg-day	Developmental neuropsychological impairment (N)	10	1	IRIS	07/07/2001
NICKEL	Chronic	2.0E-02	mg/kg-day	4.0E-02	8.0E-04	mg/kg-day	Decreased body and organ weight (W)	300	1	IRIS	02/01/2008
SELENIUM	Chronic	5.0E-03	mg/kg-day	8.0E-01	5.0E-03	mg/kg-day	Clinical selenosis	3	1	IRIS	02/01/2008
SILVER	Chronic	5.0E-03	mg/kg-day	4.0E-02	2.0E-04	mg/kg-day	Argyria (In)	3	1	IRIS	02/01/2008
THALLIUM	Chronic	8.0E-05	mg/kg-day	1.0E+00	8.0E-05	mg/kg-day	Hematological effects	3000	1	IRIS (thallium chloride)	02/01/2008
VANADIUM	Chronic	9.0E-03	mg/kg-day	2.6E-02	2.3E-04	mg/kg-day	Decreased hair cystine	100	1	IRIS (Vanadium pentoxide as surrogate)	02/01/2008
ZINC	Chronic	3.0E-01	mg/kg-day	1.0E+00	3.0E-01	mg/kg-day	Decreased ESOD (B)	3	1	IRIS	02/01/2008
PCBs											
LESS CHLORINATED ^b	Chronic	7.0E-05	mg/kg-day	9.6E-01	7.0E-05	mg/kg-day	Reduced birth weights (W)	100	1	IRIS	02/01/2008
HIGHLY CHLORINATED ^c	Chronic	2.0E-05	mg/kg-day	9.6E-01	2.0E-05	mg/kg-day	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	300	1	IRIS	02/01/2008
TOTAL PCBs ^d	Chronic	2.0E-05	mg/kg-day	9.6E-01	2.0E-05	mg/kg-day	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	300	1	IRIS	02/01/2008
PESTICIDES											
4,4'-DDD	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4,4'-DDT	Chronic	5.0E-04	mg/kg-day	9.0E-01	5.0E-04	mg/kg-day	Liver lesions (H)	100	1	IRIS	02/01/2008
ALDRIN	Chronic	3.0E-05	mg/kg-day	1.0E+00	3.0E-05	mg/kg-day	Liver toxicity (H)	1000	1	IRIS	02/01/2008
ALPHA-BHC	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ATRAZINE	Chronic	3.5E-02	mg/kg-day	1.0E+00	3.5E-02	mg/kg-day	Decreased body weight gain (W)	100	1	IRIS	02/01/2008
CHLORDANE	Chronic	5.00E-04	mg/kg-day	1.0E+00	5.00E-04	mg/kg-day	Neurotoxicity and hematotoxicity.	300	1	IRIS	04/28/2008
DELTA-BHC	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
DIELDRIN	Chronic	5.0E-05	mg/kg-day	1.0E+00	5.0E-05	mg/kg-day	Hepatic (H)	100	1	IRIS	02/01/2008

TABLE 5.1
NON-CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD		Oral Absorption Efficiency for Dermal (unitless) (1)	Absorbed RfD for Dermal (2)		Primary Target Organ(s)/Critical Effect(s) (3)	Combined Uncertainty/Modifying Factors (Uncertainty) (Modifying)		RfD:Target Organ(s)	
		Value	Units		Value	Units				Source(s)	Date(s) (MM/DD/YYYY)
ENDOSULFAN I	Chronic	6.0E-03	mg/kg-day	1.0E+00	6.0E-03	mg/kg-day	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	100	1	IRIS (Endosulfan as surrogate)	02/01/2008
ENDOSULFAN II	Chronic	6.0E-03	mg/kg-day	1.0E+00	6.0E-03	mg/kg-day	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	100	1	IRIS (Endosulfan as surrogate)	02/01/2008
ENDOSULFAN SULFATE	Chronic	6.0E-03	mg/kg-day	1.0E+00	6.0E-03	mg/kg-day	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	100	1	IRIS (Endosulfan as surrogate)	02/01/2008
ENDRIN ALDEHYDE	Chronic	3.0E-04	mg/kg-day	1.0E+00	3.0E-04	mg/kg-day	Mild histological lesions in liver (H), occasional convulsions	100	1	IRIS (Endrin as surrogate)	02/01/2008
ENDRIN KETONE	Chronic	3.0E-04	mg/kg-day	1.0E+00	3.0E-04	mg/kg-day	Mild histological lesions in liver (H), occasional convulsions	100	1	IRIS (Endrin as surrogate)	02/01/2008
HEPTACHLOR EPOXIDE	Chronic	1.3E-05	mg/kg-day	1.0E+00	1.3E-05	mg/kg-day	Increased liver-to-body weight ratio in males and females (H)	1000	1	IRIS	02/01/2008
TOXAPHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
SVOCs											
1,1'-BIPHENYL	Chronic	5.0E-02	mg/kg-day	1.0E+00	5.0E-02	mg/kg-day	Kidney Damage (R)	100	10	IRIS	02/01/2008
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,2'-OXYBIS(1-CHLOROPROPANE)	Chronic	4.0E-02	mg/kg-day	1.0E+00	4.0E-02	mg/kg-day	Decrease in hemoglobin (B) and possible erythrocyte destruction	1000	1	IRIS	02/01/2008
2,4,6-TRICHLOROPHENOL	Chronic	1.0E-03	mg/kg-day	1.0E+00	1.0E-03	mg/kg-day	No adverse effects observed (O)	3000	1	PPRTV	02/21/2007
2,4-DICHLOROPHENOL	Chronic	3.0E-03	mg/kg-day	1.0E+00	3.0E-03	mg/kg-day	Decreased delayed hypersensitivity response (O)	100	1	IRIS	02/01/2008
2,4-DIMETHYLPHENOL	Chronic	2.0E-02	mg/kg-day	1.0E+00	2.0E-02	mg/kg-day	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	3000	1	IRIS	02/01/2008
2,4-DINITROPHENOL	Chronic	2.0E-03	mg/kg-day	1.0E+00	2.0E-03	mg/kg-day	Cataract formation	1000	1	IRIS	02/01/2008
2,4-DINITROTOLUENE	Chronic	2.0E-03	mg/kg-day	1.0E+00	2.0E-03	mg/kg-day	Neurotoxicity, Heinz bodies and biliary tract hyperplasia	100	1	IRIS	02/01/2008
2,6-DINITROTOLUENE	Chronic	1.00E-03	mg/kg-day	1.00E+00	1.00E-03	mg/kg-day	Central nervous system and respiratory depression, ataxia	3000	1	PPRTV	12/13/2004
2-CHLOROPHENOL	Chronic	5.0E-03	mg/kg-day	1.0E+00	5.0E-03	mg/kg-day	Reproductive efforts	1000	1	IRIS	02/01/2008
2-METHYLNAPHTHALENE	Chronic	4.0E-03	mg/kg-day	1.0E+00	4.0E-03	mg/kg-day	Pulmonary alveolar proteinosis	1000	1	IRIS	02/01/2008
2-METHYLPHENOL	Chronic	5.0E-02	mg/kg-day	1.0E+00	5.0E-02	mg/kg-day	Decreased body weights and neurotoxicity	1000	1	IRIS	02/01/2008
2-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3&4-METHYLPHENOL	Chronic	5.0E-02	mg/kg-day	1.0E+00	5.0E-02	mg/kg-day	Decreased body weight and neurotoxicity	1000	1	IRIS (3-methylphenol used)	02/01/2008
3,3'-DICHLOROBENZIDINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4,6-DINITRO-2-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-BROMOPHENYL PHENYL ETHER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-CHLORO-3-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-CHLOROPHENYL PHENYL ETHER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ACENAPHTHENE	Chronic	6.0E-02	mg/kg-day	1.0E+00	6.0E-02	mg/kg-day	Hepatotoxicity (H)	3000	1	IRIS	02/01/2008

TABLE 5.1
NON-CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD		Oral Absorption Efficiency for Dermal (unitless) (1)	Absorbed RfD for Dermal (2)		Primary Target Organ(s)/Critical Effect(s) (3)	Combined Uncertainty/Modifying Factors (Uncertainty) (Modifying)		RfD:Target Organ(s)	
		Value	Units		Value	Units				Source(s)	Date(s) (MM/DD/YYYY)
ACENAPHTHYLENE*	Chronic	3.0E-02	mg/kg-day	1.0E+00	3.0E-02	mg/kg-day	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3000	1	IRIS (Pyrene used as surrogate)	02/01/2008
ANTHRACENE	Chronic	3.0E-01	mg/kg-day	1.0E+00	3.0E-01	mg/kg-day	No observed effects (O)	3000	1	IRIS	02/01/2008
BENZ(A)ANTHRACENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZO(A)PYRENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZO(B)FLUORANTHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZO(G,H,I)PERYLENE*	Chronic	3.0E-02	mg/kg-day	1.0E+00	3.0E-02	mg/kg-day	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3000	1	IRIS (Pyrene used as surrogate)	02/01/2008
BENZO(K)FLUORANTHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BIS(2-CHLOROETHOXY)METHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BIS(2-CHLOROETHYL)ETHER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BIS(2-ETHYLHEXYL)PHTHALATE	Chronic	2.0E-02	mg/kg-day	1.0E+00	2.0E-02	mg/kg-day	Increased relative liver weight (H)	1000	1	IRIS	02/01/2008
CARBAZOLE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CHRYSENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
DIBENZ(A,H)ANTHRACENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
DIBENZOFURAN	Chronic	1.0E-03	mg/kg-day	1.0E+00	1.0E-03	mg/kg-day	Reduced length and organ weight. Excess abdominal fat (O).	10000	1	PPRTV	06/11/2007
FLUORANTHENE	Chronic	4.0E-02	mg/kg-day	1.0E+00	4.0E-02	mg/kg-day	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3000	1	IRIS	02/01/2008
FLUORENE	Chronic	4.0E-02	mg/kg-day	1.0E+00	4.0E-02	mg/kg-day	Decreased RBC (B), packed cell volumen and hemoglobin (B)	3000	1	IRIS	02/01/2008
HEXACHLOROBENZENE	Chronic	8.0E-04	mg/kg-day	1.0E+00	8.0E-04	mg/kg-day	Hepatic (H)	100	1	IRIS	02/01/2008
HEXACHLOROBUTADIENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
HEXACHLOROETHANE	Chronic	1.0E-03	mg/kg-day	1.0E+00	1.0E-03	mg/kg-day	Atrophy and degeneration of the renal tubules (R)	1000	1	IRIS	02/01/2008
IINDENO(1,2,3-CD)PYRENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
NAPHTHALENE	Chronic	2.0E-02	mg/kg-day	8.9E-01	2.0E-02	mg/kg-day	Decreased body weight (W)	3000	1	IRIS	02/01/2008
N-HEXADACANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
NITROBENZENE	Chronic	5.0E-04	mg/kg-day	1.0E+00	5.0E-04	mg/kg-day	Hematologic (B), adrenal, renal (R) and hepatic (H) lesions	10000	1	IRIS	02/01/2008
N-NITROSO-DI-N-PROPYLAMINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PENTACHLOROPHENOL	Chronic	3.0E-02	mg/kg-day	7.6E-01	3.0E-02	mg/kg-day	Liver (H) and kidney (R) pathology	100	1	IRIS	02/01/2008
PHENANTHRENE*	Chronic	3.0E-02	mg/kg-day	1.0E+00	3.0E-02	mg/kg-day	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3000	1	IRIS (Pyrene used as surrogate)	02/01/2008
PHENOL	Chronic	3.0E-01	mg/kg-day	1.0E+00	3.0E-01	mg/kg-day	Decreased maternal weight gain (W)	300	1	IRIS	02/01/2008
PYRENE	Chronic	3.0E-02	mg/kg-day	1.0E+00	3.0E-02	mg/kg-day	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3000	1	IRIS	02/01/2008
VOCs											
1,1,2,2-TETRACHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,1,2-TRICHLOROETHANE	Chronic	4.0E-03	mg/kg-day	1.0E+00	4.0E-03	mg/kg-day	Clinical serum chemistry	1000	1	IRIS	02/01/2008
1,2,3-TRICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2,4-TRICHLOROBENZENE	Chronic	1.0E-02	mg/kg-day	1.0E+00	1.0E-02	mg/kg-day	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1000	1	IRIS	02/01/2008
1,2,4-TRIMETHYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2-DICHLOROBENZENE	Chronic	9.0E-02	mg/kg-day	1.0E+00	9.0E-02	mg/kg-day	No adverse effects observed (O)	1000	1	IRIS	02/01/2008
1,2-DICHLOROETHANE	Chronic	2.0E-02	mg/kg-day	NA	NA	NA	Cardiac arrhythmia, bronchitis, central nervous system depression, and injury to the liver, kidneys, and gastrointestinal tract	3000	1	PPRTV	10/31/2002
1,2-DICHLOROPROPANE	Chronic	9.0E-02	mg/kg-day	1.0E+00	9.0E-02	mg/kg-day	Liver (H)	1000	1	ATSDR (STSC)	12/01/1989
1,3,5-TRICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,3,5-TRIMETHYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,3-DICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	Chronic	7.0E-02	mg/kg-day	1.0E+00	7.0E-02	mg/kg-day	Liver	100	1	ATSDR (STSC)	07/01/2006

TABLE 5.1
NON-CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD		Oral Absorption Efficiency for Dermal (unitless) (1)	Absorbed RfD for Dermal (2)		Primary Target Organ(s)/Critical Effect(s) (3)	Combined Uncertainty/Modifying Factors		RfD:Target Organ(s)	
		Value	Units		Value	Units		(Uncertainty)	(Modifying)	Source(s)	Date(s) (MM/DD/YYYY)
2-HEXANONE	Chronic	2.0E-01	mg/kg-day	1.0E+00	2.0E-01	mg/kg-day	Myofibrillar atrophy of the quadriceps.	300	1	PPRTV	02/01/2008
ACETONE	Chronic	9.0E-01	mg/kg-day	1.0E+00	9.0E-01	mg/kg-day	Nephropathy	1000	1	IRIS	02/01/2008
BENZENE	Chronic	4.0E-03	mg/kg-day	1.0E+00	4.0E-03	mg/kg-day	Reduced lymphocyte count	300	1	IRIS	02/01/2008
BROMODICHLOROMETHANE	Chronic	2.0E-02	mg/kg-day	1.0E+00	2.0E-02	mg/kg-day	Renal cytomegaly (R)	1000	1	IRIS	02/01/2008
BROMOMETHANE	Chronic	1.4E-03	mg/kg-day	1.0E+00	1.4E-03	mg/kg-day	Epithelial hyperplasia of the forestomach	1000	1	IRIS	02/01/2008
CARBON DISULFIDE	Chronic	1.0E-01	mg/kg-day	1.0E+00	1.0E-01	mg/kg-day	Fetal toxicity/malformations	100	1	IRIS	02/01/2008
CARBON TETRACHLORIDE	Chronic	7.0E-04	mg/kg-day	1.0E+00	7.0E-04	mg/kg-day	Liver lesions (H)	1000	1	IRIS	02/01/2008
CHLOROBENZENE	Chronic	2.0E-02	mg/kg-day	1.0E+00	2.0E-02	mg/kg-day	Histopathologic changes in liver	1000	1	IRIS	02/01/2008
CHLORODIBROMOMETHANE	Chronic	2.0E-02	mg/kg-day	1.0E+00	2.0E-02	mg/kg-day	Hepatic lesions	1000	1	IRIS	02/01/2008
CHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CHLOROFORM	Chronic	1.0E-02	mg/kg-day	1.0E+00	1.0E-02	mg/kg-day	Moderate/marked fatty cyst formation in the liver and elevated SGPT	1000	1	IRIS	02/01/2008
CIS-1,3-DICHLOROPROPENE	Chronic	3.0E-02	mg/kg-day	1.0E+00	3.0E-02	mg/kg-day	Chronic irritation	100	1	IRIS	02/01/2008
DICHLOROBENZENES	Chronic	7.0E-02	mg/kg-day	1.0E+00	7.0E-02	mg/kg-day					
DODECANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ETHYLBENZENE	Chronic	1.0E-01	mg/kg-day	1.0E+00	1.0E-01	mg/kg-day	Liver (H) and kidney (R) toxicity	1000	1	IRIS	02/01/2008
ISOPROPYLBENZENE	Chronic	1.0E-01	mg/kg-day	1.0E+00	1.0E-01	mg/kg-day	Increased average kidney weight in female rats (R)	1000	1	IRIS	02/01/2008
METHYLENE CHLORIDE	Chronic	6.0E-02	mg/kg-day	1.0E+00	6.0E-02	mg/kg-day	Liver toxicity (H)	100	1	IRIS	02/01/2008
P-ISOPROPYLTOLUENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
SEC-BUTYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
STYRENE	Chronic	2.0E-01	mg/kg-day	1.0E+00	2.0E-01	mg/kg-day	Red blood cell (B) and liver effects (H)	1000	1	IRIS	02/01/2008
TETRACHLOROETHENE	Chronic	1.0E-02	mg/kg-day	1.0E+00	1.0E-02	mg/kg-day	Hepatotoxicity in mice (H), weight gain in rats	1000	1	IRIS	02/01/2008
TOLUENE	Chronic	8.0E-02	mg/kg-day	1.0E+00	8.0E-02	mg/kg-day	Increased kidney weight (R)	3000	1	IRIS	02/01/2008
TRANS-1,3-DICHLOROPROPENE	Chronic	3.0E-02	mg/kg-day	1.0E+00	3.0E-02	mg/kg-day	Chronic irritation	100	1	IRIS (cis-1,3-Dichloropropene as surrogate)	02/01/2008
TRICHLOROETHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
VINYL CHLORIDE	Chronic	3.0E-03	mg/kg-day	1.0E+00	3.0E-03	mg/kg-day	Liver cell polymorphism (H)	30	1	IRIS	02/01/2008
XYLENES, TOTAL	Chronic	2.0E-01	mg/kg-day	1.0E+00	2.0E-01	mg/kg-day	Decreased body weight (W), increased mortality (M)	1000	1	IRIS	02/01/2008

Notes:

(1) Oral Absorption Efficiency from Exhibit 4-1 of USEPA (2004) RAGS Part E. For constituents not listed in Exhibit 4-1, an absorption efficiency of 1 is assumed. For constituents with a range of absorption efficiencies in Exhibit 4-1, the highest value is reported.
(2) For Oral Absorption Efficiency for Dermal < 0.5, Absorbed RfD for Dermal = Oral RfD * Oral Absorption Efficiency for Dermal; otherwise, Absorbed RfD for Dermal = Oral RfD (USEPA 2004 RAGS Part E, Exhibit 4-1).

(3) Codes for Effects Endpoints: B - Hematological/Blood effect; E - Endocrine system effect; GI - Gastrointestinal system; H - Hepatic/Liver effect; I - Immune system effect; In - Integumentary/Skin effect; M - Mortality/Death/Longevity; N - Nervous system effect
O - Other effect (e.g., hyperactivity, none reported); OC - Ocular effect; R - Renal/Kidney effect; T - Teratogenic effect; V - Vascular system effect; W - Decreased body weight.

* = For non-carcinogenic PAHs, the proposed surrogate benzo(a)pyrene was applied to estimate Oral Reference Dose (see USEPA 1993) Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons, EPA/600/R-93/089).

a = Because chromium was not speciated, the RfC for chromium VI was utilized.

b = Less chlorinated PCBs includes Aroclors 1221, 1232, 1016, and 1242. RfD values for Aroclor-1016 (CAS# 126741120) utilized.

c = Highly Chlorinated PCBs includes Aroclors 1248, 1254, 1260 [and higher if reported]. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

d = Includes all detected Aroclors. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

NA - Not available

Sources:

Tier 1 - IRIS - United States Environmental Protection Agency (USEPA) Integrated Risk Information System (Available at: <http://www.epa.gov/iris>).

Tier 2 - PPRTV - USEPA Provisional Peer Reviewed Toxicity Values from the Office of Research and Development/National Center for Environmental Assessment/Superfund Health Risk Technical Support Center (STSC).

Tier 3 - Tox values approved by Superfund Technical Support Center. ATSDR - Agency for Toxic Substances and Disease Registry Minimal Risk Levels (MRLs, Available at: <http://www.atsdr.cdc.gov/mrls/index.html>);

CALEPA - California Environmental Protection Agency toxicity criteria database (Available at: <http://www.oehha.ca.gov/risk/chemicalDB/index.asp>); HEAST - USEPA Health Effects Assessment Summary Tables from the USEPA STSC;

NCEA - National Center for Environmental Assessment;USEPA (2003). Memo from Southerland. OSWER Directive 9285.7-75. USEPA (1993) Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons, EPA/600/R-93/089).

STSC - Indicates that the associated value was provided for this assessment by the Superfund Technical Support Center.

TABLE 5.2
NON-CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Inhalation RfC		Extrapolated RfD (1)		Primary Target Organ(s) (2)	Combined Uncertainty/Modifying Factors (Uncertainty) (Modifying)		RfC : Target Organ(s)	
		Value	Units	Value	Units		Source(s)	Date(s) (MM/DD/YYYY)		
DIOXINS										
2,3,7,8-TCDD Equivalent	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
METALS										
ALUMINUM	Chronic	5.0E-03	mg/m³	1.4E-03	mg/kg-day	Psychomotor and cognitive impairments	300	1	PPRTV	10/23/2006
ANTIMONY	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ARSENIC	Chronic	5.0E-05	mg/m3	1.4E-05	mg/kg-day	Development, cardiovascular, nervous system	NA	NA	CalEPA (STSC)	02/04/2008
BARIUM	Chronic	5.0E-04	mg/m3	1.4E-04	mg/kg-day	Renal toxicity	NA	NA	HEAST (STSC)	1995
BERYLLIUM	Chronic	2.0E-05	mg/m³	5.7E-06	mg/kg-day	Beryllium sensitization and progression to chronic beryllium disease	10	1	IRIS	02/04/2008
CADMIUM	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CHROMIUM ^a	Chronic	1.0E-04	mg/m³	2.9E-05	mg/kg-day	Respiratory (P)	300	1	IRIS (Chromium VI particulates as surrogate)	02/04/2008
COBALT	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
COPPER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CYANIDE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
IRON	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
LEAD	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
MANGANESE	Chronic	5.0E-05	mg/m³	1.4E-05	mg/kg-day	Neurobehavioral changes (N, O)	1000	1	IRIS	02/04/2008
MERCURY	Chronic	3.0E-04	mg/m³	8.6E-05	mg/kg-day	PNS (N); CNS (N)	30	1	IRIS	02/04/2008
METHYLMERCURY	Chronic	NA	NA	NA	NA	NA	NA	NA	NA	NA
NICKEL	Chronic	9.0E-05	mg/m³	2.6E-05	mg/kg-day	Respiratory (P)	3.00E+01	1	ATSDR (ATSC)	09/01/2005
SELENIUM	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
SILVER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
THALLIUM	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
VANADIUM	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ZINC	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PCBs										
LESS CHLORINATED ^b	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
HIGHLY CHLORINATED ^c	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
TOTAL PCBs ^d	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PESTICIDES										
4,4'-DDD	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4,4'-DDT	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ALDRIN	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ALPHA-BHC	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ATRAZINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CHLORDANE	Chronic	7.0E-04	mg/m3	2.0E-04	mg/m3	Neurotoxicity and hematotoxicity.	1000	1	IRIS	4/28/2008
DELTA-BHC	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
DIELDRIN	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ENDOSULFAN I	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ENDOSULFAN II	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

TABLE 5.2
NON-CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Inhalation RfC		Extrapolated RfD (1)		Primary Target Organ(s) (2)	Combined Uncertainty/Modifying Factors		RfC : Target Organ(s)	
		Value	Units	Value	Units		(Uncertainty)	(Modifying)	Source(s)	Date(s) (MM/DD/YYYY)
ENDOSULFAN SULFATE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ENDRIN ALDEHYDE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ENDRIN KETONE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
HEPTACHLOR EPOXIDE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
TOXAPHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
SVOCs										
1,1'-BIPHENYL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,2'-OXYBIS(1-CHLOROPROPANE)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,4,6-TRICHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,4-DICHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,4-DIMETHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,4-DINITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,4-DINITROTOLUENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2,6-DINITROTOLUENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-CHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3&4-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3,3'-DICHLOROBENZIDINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4,6-DINITRO-2-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-BROMOPHENYL PHENYL ETHER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-CHLORO-3-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-CHLOROPHENYL PHENYL ETHER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
4-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ACENAPHTHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ACENAPHTHYLENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ANTHRACENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZ(A)ANTHRACENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZO(A)PYRENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZO(B)FLUORANTHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZO(G,H,I)PERYLENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BENZO(K)FLUORANTHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BIS(2-CHLOROETHOXY)METHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BIS(2-CHLOROETHYL)ETHER	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BIS(2-ETHYLHEXYL)PHTHALATE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CARBAZOLE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

TABLE 5.2
NON-CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Inhalation RfC		Extrapolated RfD (1)		Primary Target Organ(s) (2)	Combined Uncertainty/Modifying Factors		RfC : Target Organ(s)	
		Value	Units	Value	Units		(Uncertainty)	(Modifying)	Source(s)	Date(s) (MM/DD/YYYY)
CHRYSENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
DIBENZ(A,H)ANTHRACENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
DIBENZOFURAN	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
FLUORANTHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
FLUORENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
HEXACHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
HEXACHLOROBUTADIENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
HEXACHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
INDENO(1,2,3-CD)PYRENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
NAPHTHALENE	Chronic	3.0E-03	mg/m ³	8.6E-04	mg/kg-day	Nasal/respiratory (P)	3000	1	IRIS	02/04/2008
N-HEXADACANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
NITROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
N-NITROSO-DI-N-PROPYLAMINE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PENTACHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PHENANTHRENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PHENOL	Chronic	2.0E-01	mg/m ³	5.7E-02	mg/kg-day	Alimentary, cardiovascular, kidney, nervous system	NA	NA	CalEPA (STSC)	02/04/2008
PYRENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
VOCs										
1,1,2,2-TETRACHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,1,2-TRICHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2,3-TRICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2,4-TRICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2,4-TRIMETHYLBENZENE	Chronic	7.0E-03	mg/m ³	2.0E-03	mg/kg-day	Hematological and Pulmonary	3000	1	PPRTV	06/11/2007
1,2-DICHLOROBENZENE	Chronic	0.14	mg/m ³	4.0E-02	mg/kg-day	NA	NA	NA	HEAST (STSC)	1997
1,2-DICHLOROETHANE	Chronic	2.4E+00	mg/m ³	6.9E-01	mg/kg-day	Hepatic effects	90	1	ATSDR (STSC)	09/01/2001
1,2-DICHLOROPROPANE	Chronic	4.0E-03	mg/m ³	1.1E-03	mg/kg-day	Nasal	300	1	IRIS	02/04/2008
1,3,5-TRICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,3,5-TRIMETHYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,3-DICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	Chronic	8.0E-01	mg/m ³	2.3E-01	mg/kg-day	Liver	100	1	IRIS	02/04/2008
2-HEXANONE	Chronic	2.0E-01	mg/m ³	5.7E-02	mg/kg-day	Peripheral neuropathy	1000	1	IRIS	04/28/2008
ACETONE	Chronic	3.1E+00	mg/m ³	8.6E+00	mg/kg-day	Neurological effects	100	1	ATSDR (STSC)	05/01/1994
BENZENE	Chronic	3.0E-02	mg/m ³	8.6E-03	mg/kg-day	Decreased lymphocyte count	300	1	IRIS	02/04/2008
BROMODICHLOROMETHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
BROMOMETHANE	Chronic	5.0E-03	mg/m ³	1.4E-03	mg/kg-day	Nasal lesions and membrane degeneration	100	1	IRIS	02/04/2008
CARBON DISULFIDE	Chronic	7.0E-01	mg/m ³	2.0E-01	mg/kg-day	Peripheral nervous system dysfunction	30	1	IRIS	02/04/2008
CARBON TETRACHLORIDE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CHLORODIBROMOMETHANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
CHLOROETHANE	Chronic	1.0E+01	mg/m ³	2.8E+00	mg/kg-day	Delayed fetal ossification	300	1	IRIS	02/04/2008

TABLE 5.2
NON-CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Inhalation RfC		Extrapolated RfD (1)		Primary Target Organ(s) (2)	Combined Uncertainty/Modifying Factors (Uncertainty) (Modifying)		RfC : Target Organ(s)	
		Value	Units	Value	Units				Source(s)	Date(s) (MM/DD/YYYY)
CHLOROFORM	Chronic	9.8E-02	mg/m ³	2.8E-02	mg/kg-day	Hepatic effects	100	1	ATSDR (STSC)	09/01/1997
CIS-1,3-DICHLOROPROPENE	Chronic	2.0E-02	mg/m ³	5.7E-03	mg/kg-day	Nasal epithelium hypertrophy/hyperplasia	30	1	IRIS	02/04/2008
DICHLOROBENZENES	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
DODECANE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ETHYLBENZENE	Chronic	1.0E+00	mg/m ³	2.9E-01	mg/kg-day	Developmental toxicity	300	1	IRIS	02/04/2008
ISOPROPYLBENZENE	Chronic	4.0E-01	mg/m ³	1.10E-01	mg/kg-day	Increased kidney and adrenal weights	1000	1	IRIS	02/04/2008
METHYLENE CHLORIDE	Chronic	1.04E+00	mg/m ³	3.0E-01	mg/kg-day	Hepatic effects	30	1	ATSDR (STSC)	2007
P-ISOPROPYLTOLUENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
SEC-BUTYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
STYRENE	Chronic	1.0E+01	mg/m ³	2.9E+00	mg/kg-day	Central nervous system effects	30	1	IRIS	02/04/2008
TETRACHLOROETHENE	Chronic	2.7E-01	mg/m ³	7.6E-02	mg/kg-day	Neurological effects	100	1	ATSDR (STSC)	9/1/2007
TOLUENE	Chronic	5.0E+00	mg/m ³	1.4E+00	mg/kg-day	Neurological effects	10	1	IRIS	02/04/2008
TRANS-1,3-DICHLOROPROPENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
TRICHLOROETHENE	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
VINYL CHLORIDE	Chronic	1.1E-01	mg/m ³	3.1E-02	mg/kg-day	Liver cell polymorphism	30	1	IRIS	02/04/2008
XYLENES, TOTAL	Chronic	1.0E-01	mg/m ³	2.9E-02	mg/kg-day	Impaired motor coordination (decreased rotarod performance)	300	1	IRIS	02/04/2008

Notes:

(1) Extrapolated RfD = Inhalation RfC / (70 kg / 20 m³); USEPA (1989) RAGS Part A.

(2) Codes for Effects Endpoints: B - Hematological/Blood effect; E - Endocrine system effect; GI - Gastrointestinal system; H - Hepatic/Liver effect; I - Immune system effect; In - Integumentary/Skin effect; M - Mortality/Death/Longevity; N - Nervous system effect; O - Other effect (e.g., hyperactivity, none reported); OC - Ocular effect; R - Renal/Kidney effect; T - Teratogenic effect; V - Vascular system effect; W - Decreased body weight.

a = Because chromium was not speciated, RfC and RfD values for chromium VI were utilized.

b = Less chlorinated PCBs includes Aroclors 1221, 1232, 1016, and 1242. RfD values for Aroclor-1016 (CAS# 126741120) utilized.

c = Highly Chlorinated PCBs includes Aroclors 1248, 1254, 1260 [and higher if reported]. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

d = Includes all detected Aroclors. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

NA - Not available.

Sources:

Tier 1 - IRIS - United States Environmental Protection Agency (USEPA) Integrated Risk Information System (Available at: <http://www.epa.gov/iris>).

Tier 2 - PPRTV - USEPA Provisional Peer Reviewed Toxicity Values from the Office of Research and Development/National Center for Environmental Assessment/Superfund Health Risk Technical Support Center (STSC).

Tier 3 - Tox values approved by Superfund Technical Support Center. ATSDR - Agency for Toxic Substances and Disease Registry Minimal Risk Levels (MRLs, Available at: <http://www.atsdr.cdc.gov/mrls/index.html>);

CALEPA - California Environmental Protection Agency toxicity criteria database (Available at: <http://www.oehha.ca.gov/risk/chemicalDB/index.asp>); HEAST - USEPA Health Effects Assessment Summary Tables from

the USEPA STSC; NCEA - National Center for Environmental Assessment; USEPA (2003). Memo from Southerland. OSWER Directive 9285.7-75. USEPA (1993) Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons, EPA/600/R-93/089).

STSC - Indicates that the associated value was provided for this assessment by the Superfund Technical Support Center.

RAGS Table 6 Series

TABLE 6.1
CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Oral Cancer Slope Factor		Oral Absorption Efficiency for Dermal (Unitless) (1)	Absorbed Cancer Slope Factor for Dermal (2)		Weight of Evidence/ Cancer Guideline Description (3)	Oral CSF	
	Value	Units		Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
DIOXINS								
2,3,7,8-TCDD Equivalent**	1.5E+05	(mg/kg-day) ⁻¹	7.0E-01	1.5E+05	(mg/kg-day) ⁻¹	B2	HEAST	1997
METALS								
ALUMINUM	NA	NA	NA	NA	NA	NA	NA	NA
ANTIMONY	NA	NA	NA	NA	NA	NA	NA	NA
ARSENIC	1.5E+00	(mg/kg-day) ⁻¹	9.5E-01	1.5E+00	(mg/kg-day) ⁻¹	A	IRIS	02/04/2008
BARIUM	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
BERYLLIUM	NA	NA	NA	NA	NA	B1	IRIS	02/04/2008
CADMIUM	NA	NA	NA	NA	NA	B1	IRIS	02/04/2008
CHROMIUM ^a	NA	NA	NA	NA	NA	A	IRIS (Chromium VI as surrogate)	02/04/2008
COBALT	NA	NA	NA	NA	NA	NA	NA	NA
COPPER	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
CYANIDE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
IRON	NA	NA	NA	NA	NA	NA	NA	NA
LEAD	NA	NA	NA	NA	NA	B2	IRIS	04/29/2008
MANGANESE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
MERCURY	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
METHYLMERCURY	NA	NA	NA	NA	NA	C	IRIS	02/04/2008
NICKEL	NA	NA	NA	NA	NA	NA	NA	NA
SELENIUM	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
SILVER	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
THALLIUM	NA	NA	NA	NA	NA	D	IRIS (thallium chloride)	02/04/2008
VANADIUM	NA	NA	NA	NA	NA	NA	NA	NA
ZINC	NA	NA	NA	NA	NA	NA	NA	NA
PCBs								
LESS CHLORINATED ^b	2.0E+00	(mg/kg-day) ⁻¹	9.6E-01	2.0E+00	(mg/kg-day) ⁻¹	No IRIS eval., used upper bound PCBs (B2)	IRIS	02/04/2008
HIGHLY CHLORINATED ^c	2.0E+00	(mg/kg-day) ⁻¹	9.6E-01	2.0E+00	(mg/kg-day) ⁻¹	No IRIS eval., used upper bound PCBs (B2)	IRIS	02/04/2008
TOTAL PCBs ^d	2.0E+00	(mg/kg-day) ⁻¹	9.6E-01	2.0E+00	(mg/kg-day) ⁻¹	No IRIS eval., used upper bound PCBs (B2)	IRIS	02/04/2008
PESTICIDES								
4,4'-DDD	2.4E-01	(mg/kg-day) ⁻¹	1.0E+00	2.4E-01	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
4,4'-DDT	3.4E-01	(mg/kg-day) ⁻¹	9.0E-01	3.4E-01	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
ALDRIN	1.7E+01	(mg/kg-day) ⁻¹	1.0E+00	1.7E+01	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
ALPHA-BHC	6.3E+00	(mg/kg-day) ⁻¹	1.0E+00	6.3E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
ATRAZINE	NA	NA	NA	NA	NA	NA	NA	NA
CHLORDANE	3.5E-01	(mg/kg-day) ⁻¹	1.0E+00	3.5E-01	(mg/kg-day) ⁻¹	B2	IRIS	04/29/2008
DELTA-BHC	NA	NA	NA	NA	NA	B2	IRIS	02/04/2008
DIELDRIN	1.6E+01	(mg/kg-day) ⁻¹	1.0E+00	1.6E+01	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
ENDOSULFAN I	NA	NA	NA	NA	NA	NA	NA	NA
ENDOSULFAN II	NA	NA	NA	NA	NA	NA	NA	NA
ENDOSULFAN SULFATE	NA	NA	NA	NA	NA	NA	NA	NA

TABLE 6.1
CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Oral Cancer Slope Factor		Oral Absorption Efficiency for Dermal (Unitless) (1)	Absorbed Cancer Slope Factor for Dermal (2)		Weight of Evidence/ Cancer Guideline Description (3)	Oral CSF	
	Value	Units		Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
ENDRIN ALDEHYDE	NA	NA	NA	NA	NA	NA	NA	NA
ENDRIN KETONE	NA	NA	NA	NA	NA	NA	NA	NA
HEPTACHLOR EPOXIDE	9.1E+00	(mg/kg-day) ⁻¹	1.0E+00	9.1E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
TOXAPHENE	1.1E+00	(mg/kg-day) ⁻¹	1.0E+00	1.1E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
SVOCs								
1,1'-BIPHENYL	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	NA	NA	NA
2,2'-OXYBIS(1-CHLOROPROPANE)	NA	NA	NA	NA	NA	NA	NA	NA
2,4,6-TRICHLOROPHENOL	1.1E-02	(mg/kg-day) ⁻¹	7.0E-01	1.1E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
2,4-DICHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
2,4-DIMETHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
2,4-DINITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
2,4-DINITROTOLUENE	6.8E-01	(mg/kg-day) ⁻¹	1.0E+00	6.8E-01	(mg/kg-day) ⁻¹	B2	IRIS (2,4-/2,6-Dinitrotoluene Mixture as surrogate)	02/04/2008
2,6-DINITROTOLUENE	6.8E-01	(mg/kg-day) ⁻¹	1.0E+00	6.8E-01	(mg/kg-day) ⁻¹	B2	IRIS (2,4-/2,6-Dinitrotoluene Mixture as surrogate)	02/04/2008
2-CHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
2-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	NA	NA	NA
2-METHYLPHENOL	NA	NA	NA	NA	NA	C	IRIS	02/04/2008
2-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA
2-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
3&4-METHYLPHENOL	NA	NA	NA	NA	NA	C	IRIS (3-methylphenol used as surrogate)	02/04/2008
3,3'-DICHLOROBENZIDINE	4.5E-01	(mg/kg-day) ⁻¹	1.0E+00	4.5E-01	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
3-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA
4,6-DINITRO-2-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
4-BROMOPHENYL PHENYL ETHER	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
4-CHLORO-3-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
4-CHLOROPHENYL PHENYL ETHER	NA	NA	NA	NA	NA	NA	NA	NA
4-METHYLPHENOL	NA	NA	NA	NA	NA	C	IRIS	02/04/2008
4-NITROANILINE	NA	NA	NA	NA	NA	NA	NA	NA
4-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA	NA
ACENAPHTHENE	NA	NA	NA	NA	NA	NA	NA	NA
ACENAPHTHYLENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
ANTHRACENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
BENZ(A)ANTHRACENE*	7.3E-01	(mg/kg-day) ⁻¹	8.9E-01	7.3E-01	(mg/kg-day) ⁻¹	B2	USEPA 1993 (STSC)	06/01/2003
BENZO(A)PYRENE*	7.3E+00	(mg/kg-day) ⁻¹	8.9E-01	7.3E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
BENZO(B)FLUORANTHENE*	7.3E-01	(mg/kg-day) ⁻¹	8.9E-01	7.3E-01	(mg/kg-day) ⁻¹	B2	USEPA 1993 (STSC)	06/01/2003
BENZO(G,H,I)PERYLENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
BENZO(K)FLUORANTHENE*	7.3E-02	(mg/kg-day) ⁻¹	8.9E-01	7.3E-02	(mg/kg-day) ⁻¹	B2	USEPA 1993 (STSC)	06/01/2003
BIS(2-CHLOROETHOXY)METHANE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
BIS(2-CHLOROETHYL)ETHER	1.1E+00	(mg/kg-day) ⁻¹	1.0E+00	1.1E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
BIS(2-ETHYLHEXYL)PHTHALATE	1.4E-02	(mg/kg-day) ⁻¹	1.0E+00	1.4E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008

TABLE 6.1
CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Oral Cancer Slope Factor		Oral Absorption Efficiency for Dermal (Unitless) (1)	Absorbed Cancer Slope Factor for Dermal (2)		Weight of Evidence/ Cancer Guideline Description (3)	Oral CSF	
	Value	Units		Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
CARBAZOLE	NA	NA	NA	NA	NA	NA	NA	NA
CHRYSENE*	7.3E-03	(mg/kg-day) ⁻¹	8.9E-01	7.3E-03	(mg/kg-day) ⁻¹	B2	USEPA 1993 (STSC)	06/01/2003
DIBENZ(A,H)ANTHRACENE*	7.3E+00	(mg/kg-day) ⁻¹	8.9E-01	7.3E+00	(mg/kg-day) ⁻¹	B2	USEPA 1993 (STSC)	06/01/2003
DIBENZOFURAN	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
FLUORANTHENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
FLUORENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
HEXACHLOROBENZENE	1.6E+00	(mg/kg-day) ⁻¹	1.0E+00	1.6E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
HEXACHLOROBUTADIENE	7.8E-02	(mg/kg-day) ⁻¹	1.0E+00	7.8E-02	(mg/kg-day) ⁻¹	C	IRIS	02/04/2008
HEXACHLOROETHANE	1.4E-02	(mg/kg-day) ⁻¹	1.0E+00	1.4E-02	(mg/kg-day) ⁻¹	C	IRIS	02/04/2008
INDENO(1,2,3-CD)PYRENE*	7.3E-01	(mg/kg-day) ⁻¹	8.9E-01	7.3E-01	(mg/kg-day) ⁻¹	B2	USEPA 1993 (STSC)	06/01/2003
NAPHTHALENE	NA	NA	NA	NA	NA	C	IRIS	02/04/2008
N-HEXADACANE	NA	NA	NA	NA	NA	NA	NA	NA
NITROBENZENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
N-NITROSO-DI-N-PROPYLAMINE	7.0E+00	(mg/kg-day) ⁻¹	1.0E+00	7.0E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
PENTACHLOROPHENOL	1.2E-01	(mg/kg-day) ⁻¹	7.6E-01	1.2E-01	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
PHENANTHRENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
PHENOL	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
PYRENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
VOC								
1,1,2,2-TETRACHLOROETHANE	2.0E-01	(mg/kg-day) ⁻¹	1.0E+00	2.0E-01	(mg/kg-day) ⁻¹	C	IRIS	02/04/2008
1,1,2-TRICHLOROETHANE	5.7E-02	(mg/kg-day) ⁻¹	1.0E+00	5.7E-02	(mg/kg-day) ⁻¹	C	IRIS	02/04/2008
1,2,3-TRICHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA
1,2,4-TRICHLOROETHANE	3.6E-03	(mg/kg-day) ⁻¹	1.0E+00	3.6E-03	(mg/kg-day) ⁻¹	D	CalEPA (STSC)	04/29/2008
1,2,4-TRIMETHYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA
1,2-DICHLOROETHANE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
1,2-DICHLOROETHANE	9.1E-02	(mg/kg-day) ⁻¹	1.0E+00	9.1E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
1,2-DICHLOROPROPANE	3.60E-02	(mg/kg-day) ⁻¹	1.0E+00	3.60E-02	(mg/kg-day) ⁻¹	NA	CalEPA (STSC)	04/29/2008
1,3,5-TRICHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA
1,3,5-TRIMETHYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA
1,3-DICHLOROETHANE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
1,4-DICHLOROETHANE	5.40E-03	(mg/kg-day) ⁻¹	1.00E+00	5.40E-03	(mg/kg-day) ⁻¹	B2	CalEPA (STSC)	04/29/2008
2-HEXANONE	NA	NA	NA	NA	NA	NA	NA	NA
ACETONE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
BENZENE	5.5E-02	(mg/kg-day) ⁻¹	1.0E+00	5.5E-02	(mg/kg-day) ⁻¹	A	IRIS	02/04/2008
BROMODICHLOROMETHANE	6.2E-02	(mg/kg-day) ⁻¹	1.0E+00	6.2E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
BROMOMETHANE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
CARBON DISULFIDE	NA	NA	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.3E-01	(mg/kg-day) ⁻¹	1.0E+00	1.3E-01	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
CHLOROBENZENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
CHLORODIBROMOMETHANE	8.4E-02	(mg/kg-day) ⁻¹	1.0E+00	8.4E-02	(mg/kg-day) ⁻¹	C	IRIS	02/04/2008
CHLOROETHANE	NA	NA	NA	NA	NA	NA	NA	NA
CHLOROFORM	NA	NA	NA	NA	NA	B2	IRIS	02/04/2008
CIS-1,3-DICHLOROPROPENE	5.0E-02	(mg/kg-day) ⁻¹	1.0E+00	5.0E-02	(mg/kg-day) ⁻¹	NA	NA	NA

TABLE 6.1
CANCER TOXICITY DATA -- ORAL/DERMAL
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Oral Cancer Slope Factor		Oral Absorption Efficiency for Dermal (Unitless) (1)	Absorbed Cancer Slope Factor for Dermal (2)		Weight of Evidence/ Cancer Guideline Description (3)	Oral CSF	
	Value	Units		Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
DICHLOROBENZENES	NA	NA	NA	NA	NA	NA	NA	NA
DODECANE	NA	NA	NA	NA	NA	NA	NA	NA
ETHYLBENZENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
ISOPROPYLBENZENE	NA	NA	NA	NA	NA	D	IRIS	02/04/2008
METHYLENE CHLORIDE	7.5E-03	(mg/kg-day) ⁻¹	1.0E+00	7.5E-03	(mg/kg-day) ⁻¹	B2	IRIS	02/04/2008
P-ISOPROPYLTOLUENE	NA	NA	NA	NA	NA	NA	NA	NA
SEC-BUTYLBENZENE	NA	NA	NA	NA	NA	NA	NA	NA
STYRENE	NA	NA	NA	NA	NA	NA	NA	NA
TETRACHLOROETHENE	5.4E-01	(mg/kg-day) ⁻¹	1.0E+00	5.4E-01	(mg/kg-day) ⁻¹	B2	USEPA 2003 (STSC)	06/01/2003
TOLUENE	NA	NA	NA	NA	NA	NA	NA	NA
TRANS-1,3-DICHLOROPROPENE	NA	NA	NA	NA	NA	NA	NA	NA
TRICHLOROETHENE	4.0E-01	(mg/kg-day) ⁻¹	1.0E+00	4.0E-01	(mg/kg-day) ⁻¹	A2	NCEA (STSC)	01/01/2001
VINYL CHLORIDE ^a	1.5E+00	(mg/kg-day) ⁻¹	1.0E+00	1.5E+00	(mg/kg-day) ⁻¹	A	IRIS	02/04/2008
VINYL CHLORIDE ^f	7.5E-01	(mg/kg-day) ⁻¹	1.0E+00	7.5E-01	(mg/kg-day) ⁻¹	A	IRIS	02/04/2008
XYLENES, TOTAL	NA	NA	NA	NA	NA	NA	NA	NA

Notes:

(1) Oral Absorption Efficiency from Exhibit 4-1 of USEPA (2004) RAGS Part E. For constituents not listed in Exhibit 4-1, an absorption efficiency of 1 is assumed. For constituents with a range of absorption efficiencies in Exhibit 4-1, the highest value is reported.

(2) For Oral Absorption Efficiency for Dermal < 0.5, Absorbed Cancer Slope Factor for Dermal = Oral Cancer Slope Factor / Oral Absorption Efficiency for Dermal; otherwise, Absorbed Cancer Slope Factor for Dermal = Oral Cancer Slope Factor (USEPA 2004 RAGS Part E, Exhibit 4-1).

(3) Codes for Weight of Evidence: A - Human Carcinogen; B - Probable Human Carcinogen; C - Possible Human Carcinogen; D - Not Classifiable as to Human Carcinogenicity; E - Evidence of Non-Carcinogenicity in Humans.

* = For carcinogenic PAHs, relative potency approach with respect to benzo(a)pyrene applied to estimate Oral Cancer Slope Factor (see Table L-5 and USEPA 1993 Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons, EPA/600/R-93/089).

a = Because chromium was not speciated, the CSF for chromium VI was utilized.

b = Less chlorinated includes Aroclors 1221, 1232, 1016, and 1242. RfD values for Aroclor-1016 (CAS# 126741120) utilized.

c = Highly Chlorinated includes Aroclors 1248, 1254, 1260 [and higher if reported]. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

d = Includes all detected Aroclors. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

e = Cancer slope factor/unit risk for continuous exposure to Vinyl Chloride from birth. To be used in calculation of risk to receptors <18 years of age only.

f = Cancer slope factor/unit risk for continuous exposure to Vinyl Chloride from adulthood. To be used in calculation of risk to receptors >18 years of age only.

NA - Not available

Sources:

Tier 1 - IRIS - United States Environmental Protection Agency (USEPA) Integrated Risk Information System (Available at: <http://www.epa.gov/iris>).

Tier 2 - PPRTV - USEPA Provisional Peer Reviewed Toxicity Values from the Office of Research and Development/National Center for Environmental Assessment/Superfund Health Risk Technical Support Center (STSC).

Tier 3 - Tox values approved by Superfund Technical Support Center. ATSDR - Agency for Toxic Substances and Disease Registry Minimal Risk Levels (MRLs, Available at: <http://www.atsdr.cdc.gov/mrls/index.html>); CALEPA - California Environmental Protection Agency toxicity criteria database (Available at: <http://www.oehha.ca.gov/risk/chemicalDB/index.asp>); HEAST - USEPA Health Effects Assessment Summary Tables from the USEPA STSC; NCEA - National Center for Environmental Assessment; USEPA (2003). Memo from Southerland. OSWER Directive 9285.7-75. USEPA (1993) Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons, EPA/600/R-93/089).

STSC - Indicates that the associated value was provided for this assessment by the Superfund Technical Support Center.

TABLE 6.2
CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Inhalation Unit Risk		Inhalation Cancer Slope Factor (1)		Weight of Evidence/ Cancer Guideline Description (2)	Unit Risk : Inhalation CSF	
	Value	Units	Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
DIOXIN							
2,3,7,8-TCDD Equivalent	NA	NA	NA	NA	NA	NA	NA
METALS							
ALUMINUM	NA	NA	NA	NA	NA	NA	NA
ANTIMONY	NA	NA	NA	NA	NA	NA	NA
ARSENIC	4.3E+00	(mg/m ³) ⁻¹	1.5E+01	(mg/kg-day) ⁻¹	A	IRIS	02/05/2008
BARIUM	NA	NA	NA	NA	D	IRIS	04/29/2008
BERYLLIUM	NA	NA	NA	NA	D	IRIS	02/05/2008
CADMIUM	1.8E+00	(mg/m ³) ⁻¹	6.3E+00	(mg/kg-day) ⁻¹	B1	IRIS	02/05/2008
CHROMIUM ^a	1.2E+01	(mg/m ³) ⁻¹	4.2E+01	(mg/kg-day) ⁻¹	A (Chromium VI used as surrogate)	IRIS	02/05/2008
COBALT	NA	NA	NA	NA	NA	NA	NA
COPPER	NA	NA	NA	NA	D	IRIS	02/05/2008
CYANIDE	NA	NA	NA	NA	D	IRIS	02/05/2008
IRON	NA	NA	NA	NA	NA	NA	NA
LEAD	NA	NA	NA	NA	B2 (IRIS): The agent is possibly carcinogenic to humans	IRIS	11/01/1993
MANGANESE	NA	NA	NA	NA	D	IRIS	02/05/2008
MERCURY	NA	NA	NA	NA	D	IRIS	02/05/2008
METHYLMERCURY	NA	NA	NA	NA	D	IRIS	02/05/2008
NICKEL	2.6E-01	(mg/m ³) ⁻¹	9.1E-01	(mg/kg-day) ⁻¹	A	CalEPA (STSC)	04/29/2008
SELENIUM	NA	NA	NA	NA	D	IRIS	02/05/2008
SILVER	NA	NA	NA	NA	D	IRIS	02/05/2008
THALLIUM	NA	NA	NA	NA	D (thallium chloride)	IRIS	02/05/2008
VANADIUM	NA	NA	NA	NA	NA	NA	NA
ZINC	NA	NA	NA	NA	NA	NA	NA
PCBs							
LESS CHLORINATED ^c	1.0E-01	(mg/m ³) ⁻¹	2.0E+00	(mg/kg-day) ⁻¹	No IRIS eval., used upper bound PCBs (B2)	IRIS	02/05/2008
HIGHLY CHLORINATED ^d	1.0E-01	(mg/m ³) ⁻¹	2.0E+00	(mg/kg-day) ⁻¹	No IRIS eval., used upper bound PCBs (B2)	IRIS	02/05/2008
TOTAL PCBs ^b	1.0E-01	(mg/m ³) ⁻¹	2.0E+00	(mg/kg-day) ⁻¹	No IRIS eval., used upper bound PCBs (B2)	IRIS	02/05/2008
PESTICIDES							
4,4'-DDD	NA	NA	NA	NA	B2	IRIS	02/05/2008
4,4'-DDT	9.7E-02	(mg/m ³) ⁻¹	3.4E-01	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
ALDRIN	4.9E+00	(mg/m ³) ⁻¹	1.7E+01	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
ALPHA-BHC	1.8E+00	(mg/m ³) ⁻¹	6.3E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
ATRAZINE	NA	NA	NA	NA	NA	NA	NA
CHLORDANE	1.0E-01	(mg/m ³) ⁻¹	3.5E-01	(mg/kg-day) ⁻¹	B2	IRIS	04/29/2008
DELTA-BHC	NA	NA	NA	NA	D	IRIS	02/05/2008
DIELDRIN	4.6E+00	(mg/m ³) ⁻¹	1.6E+01	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
ENDOSULFAN I	NA	NA	NA	NA	NA	NA	NA
ENDOSULFAN II	NA	NA	NA	NA	NA	NA	NA
ENDOSULFAN SULFATE	NA	NA	NA	NA	NA	NA	NA

TABLE 6.2
CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Inhalation Unit Risk		Inhalation Cancer Slope Factor (1)		Weight of Evidence/ Cancer Guideline Description (2)	Unit Risk : Inhalation CSF	
	Value	Units	Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
ENDRIN ALDEHYDE	NA	NA	NA	NA	NA	NA	NA
ENDRIN KETONE	NA	NA	NA	NA	NA	NA	NA
HEPTACHLOR EPOXIDE	2.6E+00	(mg/m ³) ⁻¹	9.1E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
TOXAPHENE	3.2E-01	(mg/m ³) ⁻¹	1.1E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
SVOC							
1,1'-BIPHENYL	NA	NA	NA	NA	D	IRIS	02/05/2008
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	NA	NA
2,2'-OXYBIS(1-CHLOROPROPANE)	NA	NA	NA	NA	NA	NA	NA
2,4,6-TRICHLOROPHENOL	3.1E-03	(mg/m ³) ⁻¹	1.1E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
2,4-DICHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA
2,4-DIMETHYLPHENOL	NA	NA	NA	NA	NA	NA	NA
2,4-DINITROPHENOL	NA	NA	NA	NA	NA	NA	NA
2,4-DINITROTOLUENE	NA	NA	NA	NA	NA	NA	NA
2,6-DINITROTOLUENE	NA	NA	NA	NA	B2	IRIS	02/05/2008
2-CHLOROPHENOL	NA	NA	NA	NA	NA	NA	NA
2-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	NA	NA
2-METHYLPHENOL	NA	NA	NA	NA	C	IRIS	02/05/2008
2-NITROANILINE	NA	NA	NA	NA	NA	NA	NA
2-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA
3&4-METHYLPHENOL	NA	NA	NA	NA	C	IRIS (3-methylphenol used as surrogate) CalEPA (STSC)	02/05/2008 04/29/2008
3,3'-DICHLOROBENZIDINE	3.0E-01	(mg/m ³) ⁻¹	1.1E+00	(mg/kg-day) ⁻¹	B2	NA	NA
3-NITROANILINE	NA	NA	NA	NA	NA	NA	NA
4,6-DINITRO-2-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA
4-BROMOPHENYL PHENYL ETHER	NA	NA	NA	NA	D	IRIS	02/05/2008
4-CHLORO-3-METHYLPHENOL	NA	NA	NA	NA	NA	NA	NA
4-CHLOROPHENYL PHENYL ETHER	NA	NA	NA	NA	NA	NA	NA
4-METHYLPHENOL	NA	NA	NA	NA	C	IRIS	02/05/2008
4-NITROANILINE	NA	NA	NA	NA	NA	NA	NA
4-NITROPHENOL	NA	NA	NA	NA	NA	NA	NA
ACENAPHTHENE	NA	NA	NA	NA	NA	NA	NA
ACENAPHTHYLENE	NA	NA	NA	NA	D	IRIS	02/05/2008
ANTHRACENE	NA	NA	NA	NA	D	IRIS	02/05/2008
BENZ(A)ANTHRACENE	NA	NA	NA	NA	B2	IRIS	03/01/1994
BENZO(A)PYRENE	NA	NA	NA	NA	B2	IRIS	07/01/1992
BENZO(B)FLUORANTHENE	NA	NA	NA	NA	B2	IRIS	03/01/1994
BENZO(G,H,I)PERYLENE	NA	NA	NA	NA	D	IRIS	02/05/2008
BENZO(K)FLUORANTHENE	NA	NA	NA	NA	B2	IRIS	03/01/1994
BIS(2-CHLOROETHOXY)METHANE	NA	NA	NA	NA	D	IRIS	02/05/2008
BIS(2-CHLOROETHYL)ETHER	3.3E-01	(mg/m ³) ⁻¹	1.2E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
BIS(2-ETHYLHEXYL)PHTHALATE	NA	NA	NA	NA	B2	IRIS	02/05/2008

TABLE 6.2
CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Inhalation Unit Risk		Inhalation Cancer Slope Factor (1)		Weight of Evidence/ Cancer Guideline Description (2)	Unit Risk : Inhalation CSF	
	Value	Units	Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
CARBAZOLE	NA	NA	NA	NA	NA	NA	NA
CHRYSENE	NA	NA	NA	NA	B2	NA	03/01/1994
DIBENZ(A,H)ANTHRACENE	NA	NA	NA	NA	B2	IRIS	03/01/1994
DIBENZOFURAN	NA	NA	NA	NA	D	IRIS	02/05/2008
FLUORANTHENE	NA	NA	NA	NA	D	IRIS	02/05/2008
FLUORENE	NA	NA	NA	NA	D	IRIS	02/05/2008
HEXACHLOROBENZENE	4.6E-01	(mg/m ³) ⁻¹	1.6E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
HEXACHLOROBUTADIENE	2.2E-02	(mg/m ³) ⁻¹	7.7E-02	(mg/kg-day) ⁻¹	C	IRIS	02/05/2008
HEXACHLOROETHANE	4.0E-03	(mg/m ³) ⁻¹	1.4E-02	(mg/kg-day) ⁻¹	C	IRIS	02/05/2008
INDENO(1,2,3-CD)PYRENE	NA	NA	NA	NA	B2	IRIS	03/01/1994
NAPHTHALENE	3.4E-02	(mg/m ³) ⁻¹	1.2E-01	(mg/kg-day) ⁻¹	C	CalEPA (STSC)	04/29/2008
N-HEXADACANE	NA	NA	NA	NA	NA	NA	NA
NITROBENZENE	NA	NA	NA	NA	D	IRIS	02/05/2008
N-NITROSO-DI-N-PROPYLAMINE	2.0E+00	(mg/m ³) ⁻¹	7.0E+00	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
PENTACHLOROPHENOL	4.6E-03	(mg/m ³) ⁻¹	1.6E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
PHENANTHRENE	NA	NA	NA	NA	D	IRIS	02/05/2008
PHENOL	NA	NA	NA	NA	D	IRIS	02/05/2008
PYRENE	NA	NA	NA	NA	D	IRIS	02/05/2008
VOC							
1,1,2,2-TETRACHLOROETHANE	5.8E-02	(mg/m ³) ⁻¹	2.0E-01	(mg/kg-day) ⁻¹	C	IRIS	02/05/2008
1,1,2-TRICHLOROETHANE	1.6E-02	(mg/m ³) ⁻¹	5.6E-02	(mg/kg-day) ⁻¹	C	IRIS	02/05/2008
1,2,3-TRICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA
1,2,4-TRICHLOROBENZENE	NA	NA	NA	NA	D	IRIS	02/05/2008
1,2,4-TRIMETHYLBENZENE	NA	NA	NA	NA	NA	NA	NA
1,2-DICHLOROBENZENE	NA	NA	NA	NA	D	IRIS	02/05/2008
1,2-DICHLOROETHANE	2.6E-02	(mg/m ³) ⁻¹	9.1E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
1,2-DICHLOROPROPANE	1.0E-02	(mg/m ³) ⁻¹	3.6E-02	(mg/kg-day) ⁻¹	NA	CalEPA (STSC)	04/29/2008
1,3,5-TRICHLOROBENZENE	NA	NA	NA	NA	NA	NA	NA
1,3,5-TRIMETHYLBENZENE	NA	NA	NA	NA	NA	NA	NA
1,3-DICHLOROBENZENE	NA	NA	NA	NA	D	IRIS	02/05/2008
1,4-DICHLOROBENZENE	1.1E-02	(mg/m ³) ⁻¹	4.0E-02	(mg/kg-day) ⁻¹	B2	CalEPA (STSC)	04/29/2008
2-HEXANONE	NA	NA	NA	NA	NA	NA	NA
ACETONE	NA	NA	NA	NA	D	IRIS	02/05/2008
BENZENE	7.8E-03	(mg/m ³) ⁻¹	2.7E-02	(mg/kg-day) ⁻¹	A	IRIS	02/05/2008
BROMODICHLOROMETHANE	3.7E-02	(mg/m ³) ⁻¹	1.3E-01	(mg/kg-day) ⁻¹	B2	CalEPA (STSC)	04/29/2008
BROMOMETHANE	NA	NA	NA	NA	D	IRIS	02/05/2008
CARBON DISULFIDE	NA	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.5E-02	(mg/m ³) ⁻¹	5.3E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
CHLOROBENZENE	NA	NA	NA	NA	D	IRIS	02/05/2008
CHLORODIBROMOMETHANE	NA	NA	NA	NA	C	IRIS	02/05/2008
CHLOROETHANE	NA	NA	NA	NA	NA	NA	NA
CHLOROFORM	2.3E-02	(mg/m ³) ⁻¹	8.1E-02	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
CIS-1,3-DICHLOROPROPENE	NA	NA	NA	NA	NA	NA	NA

TABLE 6.2
CANCER TOXICITY DATA -- INHALATION
HONEYWELL, WASTEBED B/HARBOR BROOK SITE, GEDDES AND SYRACUSE, NEW YORK

Chemical of Potential Concern	Inhalation Unit Risk		Inhalation Cancer Slope Factor (1)		Weight of Evidence/ Cancer Guideline Description (2)	Unit Risk : Inhalation CSF	
	Value	Units	Value	Units		Source(s)	Date(s) (MM/DD/YYYY)
DICHLOROBENZENES	NA	NA	NA	NA	NA	NA	02/05/2008
DODECANE	NA	NA	NA	NA	NA	NA	NA
ETHYLBENZENE	NA	NA	NA	NA	D	IRIS	02/05/2008
ISOPROPYLBENZENE	NA	NA	NA	NA	D	IRIS	02/05/2008
METHYLENE CHLORIDE	4.7E-04	(mg/m ³) ⁻¹	1.7E-03	(mg/kg-day) ⁻¹	B2	IRIS	02/05/2008
P-ISOPROPYLTOLUENE	NA	NA	NA	NA	NA	NA	NA
SEC-BUTYLBENZENE	NA	NA	NA	NA	NA	NA	NA
STYRENE	NA	NA	NA	NA	NA	NA	NA
TETRACHLOROETHENE	5.9E-06	(mg/m ³) ⁻¹	2.1E-05	(mg/kg-day) ⁻¹	B2	USEPA 2003 (STSC)	6/12/2003
TOLUENE	NA	NA	NA	NA	NA	NA	NA
TRANS-1,3-DICHLOROPROPENE	NA	NA	NA	NA	NA	NA	NA
TRICHLOROETHENE	1.1E-01	(mg/m ³) ⁻¹	4.0E-01	(mg/kg-day) ⁻¹	A2	NCEA (STSC)	01/01/2001
VINYL CHLORIDE ^a	8.8E-03	(mg/m ³) ⁻¹	3.1E-02	(mg/kg-day) ⁻¹	A	IRIS	02/05/2008
VINYL CHLORIDE ^f	4.4E-03	(mg/m ³) ⁻¹	1.5E-02	(mg/kg-day) ⁻¹	A	IRIS	02/05/2008
XYLENES, TOTAL	NA	NA	NA	NA	NA	NA	NA

Notes:

(1) Inhalation Cancer Slope Factor = Inhalation Unit Risk * (70 kg / 20 m³); USEPA (1989) RAGS Part A.

efficiencies in Exhibit 4-1, the highest value is reported.

(2) Codes for Weight of Evidence: A - Human Carcinogen; B - Probable Human Carcinogen; C - Possible Human Carcinogen; D - Not Classifiable as to Human Carcinogenicity;

E - Evidence of Non-Carcinogenicity in Humans.

a = Because chromium was not speciated, the inhalation unit risk value for chromium VI was utilized

b = Includes all detected Aroclors. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

c = Less chlorinated includes Aroclors 1221, 1232, 1016, and 1242. RfD values for Aroclor-1016 (CAS# 126741120) utilized.

d = Highly Chlorinated includes Aroclors 1248, 1254, 1260 [and higher if reported]. RfD values for Aroclor-1254 (CAS# 11097691) utilized.

e = Cancer slope factor/unit risk for continuous exposure to Vinyl Chloride from birth. To be used in calculation of risk to receptors <18 years of age only.

f = Cancer slope factor/unit risk for continuous exposure to Vinyl Chloride from adulthood. To be used in calculation of risk to receptors >18 years of age only.

NA - Not available

Sources:

Tier 1 - IRIS - United States Environmental Protection Agency (USEPA) Integrated Risk Information System (Available at: <http://www.epa.gov/iris>).

Tier 2 - PPRTV - USEPA Provisional Peer Reviewed Toxicity Values from the Office of Research and Development/National Center for Environmental Assessment/Superfund Health Risk Technical Support Center.

Tier 3 - Tox values approved by Superfund Technical Support Center. ATSDR - Agency for Toxic Substances and Disease Registry Minimal Risk Levels (MRLs, Available at: <http://www.atsdr.cdc.gov/mrls/index.html>);

CALEPA - California Environmental Protection Agency toxicity criteria database (Available at: <http://www.oehha.ca.gov/risk/chemicalDB/index.asp>); HEAST - USEPA Health Effects Assessment Summary Tables from the USEPA STSC;

NCEA - National Center for Environmental Assessment;USEPA (2003). Memo from Southerland. OSWER Directive 9285.7-75. USEPA (1993) Provisional Guidance for Quantitative Risk Assessment of

Polycyclic Aromatic Hydrocarbons, EPA/600/R-93/089).

STSC - Indicates that the associated value was provided for this assessment by the Superfund Technical Support Center.

RAGS Table 7 RME Series

TABLE 7.1 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	Ingestion	2,3,7,8-TCDD Equivalent	2E-05	mg/kg	5E-10	mg/kg-day	2E+05	1/(mg/kg-day)	8E-05	6E-09	mg/kg-day	1E-09	mg/kg-day	6E+00
				ANTIMONY	1E+00	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	4E-04	mg/kg-day	7E-01
				ARSENIC	8E-02	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	3E-06	2E-05	mg/kg-day	3E-04	mg/kg-day	8E-02
				CHROMIUM	6E-01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-03	mg/kg-day	6E-02
				CYANIDE	6E+00	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-02	mg/kg-day	8E-02
				MANGANESE	3E+00	mg/kg	8E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	1E-01	mg/kg-day	7E-03
				MERCURY (AS METHYLMERCURY)	1E+00	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-04	mg/kg-day	3E+00
				SELENIUM	2E+00	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	5E-03	mg/kg-day	9E-02
				VANADIUM	6E-01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	9E-03	mg/kg-day	2E-02
				ZINC	4E+01	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	3E-01	mg/kg-day	4E-02
				HIGHLY CHLORINATED PCBs	6E-01	mg/kg	1E-05	mg/kg-day	2E+00	1/(mg/kg-day)	3E-05	2E-04	mg/kg-day	2E-05	mg/kg-day	9E+00
				LESS CHLORINATED PCBs	5E-01	mg/kg	1E-05	mg/kg-day	2E+00	1/(mg/kg-day)	2E-05	1E-04	mg/kg-day	7E-05	mg/kg-day	2E+00
				4,4-DDD	1E-02	mg/kg	3E-07	mg/kg-day	2E-01	1/(mg/kg-day)	8E-08	4E-06	mg/kg-day		mg/kg-day	
				4,4'-DDT	1E-02	mg/kg	2E-07	mg/kg-day	3E-01	1/(mg/kg-day)	8E-08	3E-06	mg/kg-day	5E-04	mg/kg-day	6E-03
				ALDRIN	3E-03	mg/kg	6E-08	mg/kg-day	2E+01	1/(mg/kg-day)	1E-06	7E-07	mg/kg-day	3E-05	mg/kg-day	2E-02
				DELTA-BHC	3E-03	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day		mg/kg-day	
				DIELDRIN	4E-03	mg/kg	1E-07	mg/kg-day	2E+01	1/(mg/kg-day)	2E-06	1E-06	mg/kg-day	5E-05	mg/kg-day	2E-02
				HEPTACHLOR EPOXIDE	4E-03	mg/kg	1E-07	mg/kg-day	9E+00	1/(mg/kg-day)	1E-06	1E-06	mg/kg-day	1E-05	mg/kg-day	9E-02
				BIS(2-ETHYLHEXYL)PHTHALATE	2E+00	mg/kg	6E-05	mg/kg-day	1E-02	1/(mg/kg-day)	8E-07	7E-04	mg/kg-day	2E-02	mg/kg-day	3E-02
				HEXACHLORO BENZENE	1E-02	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	4E-06	mg/kg-day	8E-04	mg/kg-day	5E-03
			Exp. Route Total								1E-04					2E+01
		Exp. Point Total									1E-04					2E+01
	Exp. Medium Total										1E-04					2E+01
Medium Total											1E-04					2E+01
Sediment	Surface Sediment	Exposure Unit 1	Dermal	2,3,7,8-TCDD Equivalent	7E-05	mg/kg	6E-12	mg/kg-day	2E+05	1/(mg/kg-day)	8E-07	7E-11	mg/kg-day	1E-09	mg/kg-day	7E-02
				ARSENIC	7E+00	mg/kg	6E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	7E-06	mg/kg-day	3E-04	mg/kg-day	2E-02
				CADMIUM	1E+00	mg/kg	4E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	3E-05	mg/kg-day	2E-03
				CHROMIUM	5E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	5E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				THALLIUM	6E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	6E-07	4E-06	mg/kg-day	2E-05	mg/kg-day	2E-01
				DIELDRIN	2E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				ENDRIN KETONE	5E-02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-04	mg/kg-day	
				HEPTACHLOR EPOXIDE	8E-03	mg/kg		mg/kg-day	9E+00	1/(mg/kg-day)			mg/kg-day	1E-05	mg/kg-day	
				1-METHYLNAPHTHALENE	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day		mg/kg-day	
				2-METHYLNAPHTHALENE	3E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	4E-03	mg/kg-day	3E-02
				ACENAPHTHYLENE	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	5E-04
				BENZ(A)ANTHRACENE	3E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	6E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	9E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	4E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-02	mg/kg-day	6E-03
				BENZO(K)FLUORANTHENE	4E+01	mg/kg		mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	4E+01	mg/kg	1E-05	mg/kg-day	1E-02	1/(mg/kg-day)		1E-04	mg/kg-day	2E-02	mg/kg-day	6E-03
				CARBAZOLE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day		mg/kg-day	
				CHRYSENE	9E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-07	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	1E-03	mg/kg-day	7E-02
				FLUORANTHENE	1E+02	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	4E-02	mg/kg-day	1E-02
				HEXACHLORO BENZENE	1E-01	mg/kg	4E-08	mg/kg-day	2E+00	1/(mg/kg-day)	6E-08	4E-07	mg/kg-day	8E-04	mg/kg-day	5E-04
				INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	6E+01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	2E-02	mg/kg-day	1E-02
				PHENANTHRENE	1E+02	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	3E-02	mg/kg-day	2E-02
				PYRENE	2E+02	mg/kg	6E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day	3E-02	mg/kg-day	2E-02
				1,2,3-TRICHLORO BENZENE	5E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLORO BENZENE	5E-01	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLORO BENZENE	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	

TABLE 7.1 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations				Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Sediment	Surface Sediment	Exposure Unit 1	Dermal	1,3,5-TRICHLOROBENZENE	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	2E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				BENZENE	4E+00	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	2E-02	mg/kg-day	
				CHLORO BENZENE	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day	
				METHYLENE CHLORIDE	4E-01	mg/kg		mg/kg-day	8E-03	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				N-HEXADACANE	8E-01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	
				P-ISOPROPYL TOLUENE	9E-03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				TOLUENE	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-02	mg/kg-day	
				XYLENES, TOTAL	3E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
			Exp. Route Total								7E-04					5E-01
			Ingestion	2,3,7,8-TCDD Equivalent	7E-05	mg/kg	1E-12	mg/kg-day	2E+05	1/(mg/kg-day)	2E-07	1E-11	mg/kg-day	1E-09	mg/kg-day	1E-02
				ARSENIC	7E+00	mg/kg	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	1E-06	mg/kg-day	3E-04	mg/kg-day	5E-03
				CADMIUM	1E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-05	mg/kg-day	1E-02
				CHROMIUM	5E+01	mg/kg	9E-07	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	8E-05	mg/kg-day	1E-01
				IRON	1E+04	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	7E-01	mg/kg-day	3E-03
				LEAD	1E+02	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	6E-03	mg/kg-day	1E-02
				MERCURY	5E+00	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	2E-05	mg/kg-day	5E-02
				THALLIUM	6E-01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	8E-05	mg/kg-day	2E-03
				VANADIUM	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	2E-04	mg/kg-day	1E-02
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	2E-07	mg/kg-day	2E-05	mg/kg-day	8E-03
				DIELDRIN	2E-02	mg/kg	3E-10	mg/kg-day	2E+01	1/(mg/kg-day)	5E-09	3E-09	mg/kg-day	5E-05	mg/kg-day	7E-05
				ENDRIN KETONE	5E-02	mg/kg	9E-10	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	3E-04	mg/kg-day	3E-05
				HEPTACHLOR EPOXIDE	8E-03	mg/kg	1E-10	mg/kg-day	9E+00	1/(mg/kg-day)	1E-09	2E-09	mg/kg-day	1E-05	mg/kg-day	1E-04
				1-METHYLNAPHTHALENE	4E+00	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day		mg/kg-day	
				2-METHYLNAPHTHALENE	3E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	4E-03	mg/kg-day	1E-03
				ACENAPHTHYLENE	4E+00	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	3E-02	mg/kg-day	3E-05
				BENZ(A)ANTHRACENE	3E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-06	6E-05	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	6E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	1E-05	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	9E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-06	2E-05	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	4E+01	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-02	mg/kg-day	3E-04
				BENZO(K)FLUORANTHENE	4E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-06	7E-06	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	4E+01	mg/kg	7E-07	mg/kg-day	1E-02	1/(mg/kg-day)	9E-09	8E-06	mg/kg-day	2E-02	mg/kg-day	4E-04
				CARBAZOLE	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	9E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-08	2E-05	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-06	2E-06	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	1E-03	mg/kg-day	4E-03
				FLUORANTHENE	1E+02	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	4E-02	mg/kg-day	7E-04
				HEXACHLOROBENZENE	1E-01	mg/kg	2E-09	mg/kg-day	2E+00	1/(mg/kg-day)	4E-09	3E-08	mg/kg-day	8E-04	mg/kg-day	3E-05
				INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	7E-06	mg/kg-day		mg/kg-day	
				NAPHTHALENE	6E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	2E-02	mg/kg-day	6E-04
				PHENANTHRENE	1E+02	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	8E-04
				PYRENE	2E+02	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				1,2,3-TRICHLOROBENZENE	5E-01	mg/kg	9E-09	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	5E-01	mg/kg	9E-09	mg/kg-day	4E-03	1/(mg/kg-day)	3E-11	1E-07	mg/kg-day	1E-02	mg/kg-day	1E-05
				1,2-DICHLOROBENZENE	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	
				1,3,5-TRICHLOROBENZENE	2E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	
				1,4-DICHLOROBENZENE	2E+01	mg/kg	4E-07	mg/kg-day	5E-03	1/(mg/kg-day)	2E-09	5E-06	mg/kg-day	7E-02	mg/kg-day	7E-05
				BENZENE	4E+00	mg/kg	7E-08	mg/kg-day	6E-02	1/(mg/kg-day)	4E-09	8E-07	mg/kg-day	4E-03	mg/kg-day	2E-04
				CHLORO BENZENE	2E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	2E-02	mg/kg-day	2E-04
				METHYLENE CHLORIDE	4E-01	mg/kg	7E-09	mg/kg-day	8E-03	1/(mg/kg-day)	5E-11	8E-08	mg/kg-day	6E-02	mg/kg-day	1E-06
				N-HEXADACANE	8E-01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day		mg/kg-day	
				P-ISOPROPYL TOLUENE	9E-03	mg/kg	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				TOLUENE	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	8E-02	mg/kg-day	3E-05
				XYLENES, TOTAL	3E+01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day	2E-01	mg/kg-day	4E-05
			Exp. Route Total								4E-05					3E-01
		Exp. Point Total									7E-04					7E-01
	Exp. Medium Total										7E-04					7E-01
Medium Total											7E-04					7E-01

TABLE 7.1 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface soil	Exposure Unit 1	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	4E-11	mg/kg-day	2E+05	1/(mg/kg-day)	7E-06	5E-10	mg/kg-day	1E-09	mg/kg-day	5E-01
				ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ANTIMONY	7E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-05	mg/kg-day	
				ARSENIC	9E+00	mg/kg	8E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	9E-06	mg/kg-day	3E-04	mg/kg-day	3E-02
				BARIIUM	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				CADMIUM	2E+01	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	3E-05	mg/kg-day	3E-02
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				SILVER	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				THALLIUM	7E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	1E+00	mg/kg	6E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	7E-06	mg/kg-day	2E-05	mg/kg-day	3E-01
				LESS CHLORINATED PCBs	9E-01	mg/kg	4E-07	mg/kg-day	2E+00	1/(mg/kg-day)	7E-07	4E-06	mg/kg-day	7E-05	mg/kg-day	6E-02
				DIELDRIN	1E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				2-METHYLNAPHTHALENE	9E+00	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	4E-03	mg/kg-day	9E-03
				ACENAPHTHYLENE	5E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	8E-04
				BENZ(A)ANTHRACENE	2E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	8E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				BENZO(K)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-07	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	2E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	3E+00	mg/kg	9E-07	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02
				FLUORANTHENE	3E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	4E-02	mg/kg-day	4E-03
				HEXACHLOROBNZENE	7E-01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	2E-06	mg/kg-day	8E-04	mg/kg-day	3E-03
				INDENO(1,2,3-CD)PYRENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-06	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	2E-02	mg/kg-day	3E-03
				PHENANTHRENE	2E+01	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	3E-03
				1,2,3-TRICHLOROBNZENE	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBNZENE	3E+00	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBNZENE	5E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBNZENE	2E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				BENZENE	4E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	3E-03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				Exp. Route Total							1E-04					1E+00
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	9E-12	mg/kg-day	2E+05	1/(mg/kg-day)	1E-06	1E-10	mg/kg-day	1E-09	mg/kg-day	1E-01
				ALUMINIUM	7E+03	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	1E+00	mg/kg-day	1E-03
				ANTIMONY	7E-01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	4E-04	mg/kg-day	3E-04
				ARSENIC	9E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	2E-06	mg/kg-day	3E-04	mg/kg-day	6E-03
				BARIIUM	3E+02	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	2E-01	mg/kg-day	3E-04
				CADMIUM	2E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	1E-03	mg/kg-day	5E-03
				CHROMIUM	1E+02	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-03	mg/kg-day	8E-03
				COPPER	2E+02	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	4E-02	mg/kg-day	1E-03
				IRON	1E+04	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	7E-01	mg/kg-day	4E-03
				LEAD	4E+02	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	1E-01	mg/kg-day	5E-04
				MERCURY	8E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-04	mg/kg-day	6E-03
				SILVER	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	5E-03	mg/kg-day	4E-04
				THALLIUM	7E-01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	8E-05	mg/kg-day	2E-03
				VANADIUM	2E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	9E-03	mg/kg-day	5E-04
				HIGHLY CHLORINATED PCBs	1E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	3E-07	mg/kg-day	2E-05	mg/kg-day	1E-02
				LESS CHLORINATED PCBs	9E-01	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	2E-07	mg/kg-day	7E-05	mg/kg-day	3E-03
				DIELDRIN	1E-02	mg/kg	2E-10	mg/kg-day	2E+01	1/(mg/kg-day)	3E-09	2E-09	mg/kg-day	5E-05	mg/kg-day	5E-05
				2-METHYLNAPHTHALENE	9E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	4E-03	mg/kg-day	4E-04
				ACENAPHTHYLENE	5E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	4E-05

TABLE 7.1 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface soil	Exposure Unit 1	Ingestion	BENZ(A)ANTHRACENE	2E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-07	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-07	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	7E-05
				BENZO(K)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-08	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	2E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-09	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	3E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	1E-03	mg/kg-day	7E-04
				FLUORANTHENE	3E+01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day	4E-02	mg/kg-day	2E-04
				HEXACHLOROBENZENE	7E-01	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-08	1E-07	mg/kg-day	8E-04	mg/kg-day	2E-04
				INDENO(1,2,3-CD)PYRENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-07	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	2E-02	mg/kg-day	2E-04
				PHENANTHRENE	2E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				1,2,3-TRICHLOROBENZENE	2E+00	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	3E+00	mg/kg	5E-08	mg/kg-day	4E-03	1/(mg/kg-day)	2E-10	6E-07	mg/kg-day	1E-02	mg/kg-day	6E-05
				1,4-DICHLOROBENZENE	5E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	9E-02	mg/kg-day	1E-05
				1,4-DICHLOROBENZENE	2E+01	mg/kg	3E-07	mg/kg-day	5E-03	1/(mg/kg-day)	2E-09	4E-06	mg/kg-day	7E-02	mg/kg-day	6E-05
				BENZENE	4E-01	mg/kg	6E-09	mg/kg-day	6E-02	1/(mg/kg-day)	3E-10	7E-08	mg/kg-day	4E-03	mg/kg-day	2E-05
				P-ISOPROPYLTOLUENE	3E-03	mg/kg	5E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day	
			Exp. Route Total								9E-06					2E-01
		Exp. Point Total									2E-04					1E+00
	Exp. Medium Total										2E-04					1E+00
Medium Total											2E-04					1E+00
Surface Soil	Outdoor Air	Exposure Unit 1	Inhalation	2,3,7,8-TCDD Equivalent	4E-07	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day	
				ALUMINUM	5E-06	mg/m3	4E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day		mg/kg-day	
				ANTIMONY	5E-10	mg/m3	4E-13	mg/kg-day		1/(mg/kg-day)		5E-12	mg/kg-day		mg/kg-day	
				ARSENIC	7E-09	mg/m3	6E-12	mg/kg-day	2E+01	1/(mg/kg-day)	9E-11	7E-11	mg/kg-day	1E-05	mg/kg-day	5E-06
				BARIUM	2E-07	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	1E-04	mg/kg-day	1E-05
				CADMIUM	2E-08	mg/m3	1E-11	mg/kg-day	6E+00	1/(mg/kg-day)	9E-11	2E-10	mg/kg-day		mg/kg-day	
				CHROMIUM	8E-08	mg/m3	7E-11	mg/kg-day	4E+01	1/(mg/kg-day)	3E-09	8E-10	mg/kg-day	3E-05	mg/kg-day	3E-05
				COPPER	1E-07	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				IRON	1E-05	mg/m3	9E-09	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day	
				LEAD	3E-07	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day	
				MANGANESE	2E-07	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	1E-05	mg/kg-day	2E-04
				MERCURY	6E-09	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		6E-11	mg/kg-day	9E-05	mg/kg-day	7E-07
				SILVER	7E-09	mg/m3	6E-12	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day	
				THALLIUM	5E-10	mg/m3	4E-13	mg/kg-day		1/(mg/kg-day)		5E-12	mg/kg-day		mg/kg-day	
				VANADIUM	2E-08	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	1E-09	mg/m3	9E-13	mg/kg-day	2E+00	1/(mg/kg-day)	2E-12	1E-11	mg/kg-day		mg/kg-day	
				LESS CHLORINATED PCBs	7E-10	mg/m3	6E-13	mg/kg-day	2E+00	1/(mg/kg-day)	1E-12	6E-12	mg/kg-day		mg/kg-day	
				DIELDRIN	8E-12	mg/m3	7E-15	mg/kg-day	2E+01	1/(mg/kg-day)	1E-13	8E-14	mg/kg-day		mg/kg-day	
				2-METHYLNAPHTHALENE	6E-09	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		6E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	4E-09	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	1E-08	mg/m3	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-12	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	1E-08	mg/m3	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-11	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	9E-09	mg/m3	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-12	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	7E-09	mg/m3	6E-12	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	9E-09	mg/m3	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-13	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	1E-08	mg/m3	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-14	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E-09	mg/m3	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-12	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E-09	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				FLUORANTHENE	2E-08	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				HEXACHLOROBENZENE	5E-10	mg/m3	4E-13	mg/kg-day	2E+00	1/(mg/kg-day)	6E-13	5E-12	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	7E-09	mg/m3	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-12	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	1E-08	mg/m3	1E-11	mg/kg-day	1E-01	1/(mg/kg-day)	1E-12	1E-10	mg/kg-day	9E-04	mg/kg-day	1E-07
				PHENANTHRENE	2E-08	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				1,2,3-TRICHLOROBENZENE	9E-05	mg/m3	7E-08	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E-04	mg/m3	8E-08	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				1,2-DICHLOROBENZENE	6E-04	mg/m3	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	4E-02	mg/kg-day	1E-04
				1,4-DICHLOROBENZENE	2E-03	mg/m3	2E-06	mg/kg-day	4E-02	1/(mg/kg-day)	8E-08	2E-05	mg/kg-day	2E-01	mg/kg-day	1E-04

TABLE 7.1 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Surface Soil	Outdoor Air	Exposure Unit 1	Inhalation	BENZENE	2E-04	mg/m3	2E-07	mg/kg-day	3E-02	1/(mg/kg-day)	5E-09	2E-06	mg/kg-day	9E-03	mg/kg-day	2E-04	
			P-ISOPROPYLTOLUENE			1/(mg/kg-day)	6E-09	mg/kg-day		mg/kg-day		mg/kg-day					
		DODECANE	6E-07	mg/m3	5E-10	mg/kg-day	1/(mg/kg-day)										
			Exp. Route Total								9E-08					7E-04	
		Exp. Point Total									9E-08					7E-04	
	Exp. Medium Total										9E-08					7E-04	
Medium Total											9E-08					7E-04	
Water	Surface water	Exposure Unit 1	Dermal	ANTIMONY	2E+00	ug/l	7E-09	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day	6E-05	mg/kg-day	1E-03	
				ARSENIC	3E+00	ug/l	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-08	1E-07	mg/kg-day	3E-04	mg/kg-day	5E-04	
				CHROMIUM	6E+00	ug/l	5E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	8E-05	mg/kg-day	7E-03	
				IRON	6E+03	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	7E-01	mg/kg-day	4E-04	
				LEAD	1E+01	ug/l	4E-09	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day		
				MANGANESE	4E+02	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	6E-03	mg/kg-day	3E-03	
				MERCURY	1E-01	ug/l	4E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day	2E-05	mg/kg-day	2E-04	
				THALLIUM	4E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	8E-05	mg/kg-day	2E-03	
				VANADIUM	2E+00	ug/l	6E-09	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day	2E-04	mg/kg-day	3E-04	
				ZINC	3E+02	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-01	mg/kg-day	3E-05	
				2,4-DIMETHYLPHENOL	5E+01	ug/l	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	2E-02	mg/kg-day	2E-03	
				2-METHYLNAPHTHALENE	6E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day		
				3&4-METHYLPHENOL	6E+01	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	5E-02	mg/kg-day	5E-04	
				ACENAPHTHENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day		
				ACENAPHTHYLENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				BENZ(A)ANTHRACENE	4E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	2E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	3E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	2E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				BENZO(K)FLUORANTHENE	2E+00	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+00	ug/l	3E-06	mg/kg-day	1E-02	1/(mg/kg-day)	4E-08	3E-05	mg/kg-day	2E-02	mg/kg-day	1E-03	
				CARBAZOLE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				CHRYSENE	3E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-07	(a)	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day		
				FLUORENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day		
				INDENO(1,2,3-CD)PYRENE	1E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-05	(a)	mg/kg-day		mg/kg-day		
				NAPHTHALENE	1E+03	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	2E-02	mg/kg-day	1E-01	
				PHENANTHRENE	2E+01	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-02	mg/kg-day	5E-03	
				PYRENE	6E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				1,2,4-TRIMETHYLBENZENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				1,3,5-TRIMETHYLBENZENE	9E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				1,4-DICHLOROBBENZENE	8E+00	ug/l	2E-06	mg/kg-day	5E-03	1/(mg/kg-day)	9E-09	2E-05	mg/kg-day	7E-02	mg/kg-day	3E-04	
				BENZENE	4E+01	ug/l	3E-06	mg/kg-day	6E-02	1/(mg/kg-day)	1E-07	3E-05	mg/kg-day	4E-03	mg/kg-day	8E-03	
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day		
				TOLUENE	2E+02	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	8E-02	mg/kg-day	4E-03	
				XYLENES, TOTAL	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day		
		Exp. Route Total								3E-04					2E-01		
		Exp. Point Total									3E-04				2E-01		
	Exp. Medium Total										3E-04				2E-01		
Medium Total											3E-04				2E-01		
Total of Receptor Risks Across All Media											1E-03	Total of Receptor Hazards Across All Media					2E+01

Notes:
(a) See Table 7.1 RME Supplement A for the intake and toxicity values for COPCs with a Mutagenic Mode of Action.

TABLE 7.1 RME Supplement A
 CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
 REASONABLE MAXIMUM EXPOSURE
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Older Child Trespasser
Receptor Age:	12 to < 18 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							12-16 yrs	16-18 yrs		12-16 yrs (ADAF=3)	16-18 yrs (ADAF=1)		
Soil	Surface Soil	EU-1	Ingestion	Benz(a)anthracene	1.5E+01	mg/kg	1.9E-07	8.0E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	5.E-07
				Benzo(a)pyrene	1.5E+01	mg/kg	1.9E-07	7.8E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	5.E-06
				Benzo(b)fluoranthene	1.3E+01	mg/kg	1.6E-07	6.8E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	4.E-07
				Benzo(k)fluoranthene	1.2E+01	mg/kg	1.5E-07	6.3E-08	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	4.E-08
				Chrysene	1.5E+01	mg/kg	1.9E-07	7.9E-08	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	5.E-09
				Dibenz(a,h)anthracene	3.3E+00	mg/kg	4.1E-08	1.7E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	1.E-06
				Indeno(1,2,3-cd)pyrene	9.1E+00	mg/kg	1.1E-07	4.8E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	3.E-07
			Dermal	Benz(a)anthracene	1.5E+01	mg/kg	3.7E-06	1.7E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	9.E-06
				Benzo(a)pyrene	1.5E+01	mg/kg	3.6E-06	1.7E-06	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	9.E-05
				Benzo(b)fluoranthene	1.3E+01	mg/kg	3.1E-06	1.5E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	8.E-06
				Benzo(k)fluoranthene	1.2E+01	mg/kg	2.9E-06	1.4E-06	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	7.E-07
				Chrysene	1.5E+01	mg/kg	3.7E-06	1.7E-06	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	9.E-08
				Dibenz(a,h)anthracene	3.3E+00	mg/kg	8.0E-07	3.7E-07	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	2.E-05
				Indeno(1,2,3-cd)pyrene	9.1E+00	mg/kg	2.2E-06	1.0E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	6.E-06
	Fugitive Dust	EU-1	Inhalation	Benz(a)anthracene	1.1E-08	mg/m³	3.2E-12	1.4E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	1.1E-08	mg/m³	3.2E-12	1.4E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	9.4E-09	mg/m³	2.7E-12	1.2E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	8.8E-09	mg/m³	2.5E-12	1.1E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	1.1E-08	mg/m³	3.2E-12	1.4E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	2.4E-09	mg/m³	6.9E-13	3.0E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	6.6E-09	mg/m³	1.9E-12	8.3E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
Sediment	Surface Sediment	EU-1	Ingestion	Benz(a)anthracene	2.8E+02	mg/kg	3.5E-06	1.4E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	9.E-06
				Benzo(a)pyrene	6.3E+01	mg/kg	8.0E-07	3.3E-07	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	2.E-05
				Benzo(b)fluoranthene	9.4E+01	mg/kg	1.2E-06	5.0E-07	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	3.E-06
				Benzo(k)fluoranthene	3.5E+01	mg/kg	4.5E-07	1.9E-07	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	1.E-07
				Chrysene	8.7E+01	mg/kg	1.1E-06	4.6E-07	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	3.E-08
				Dibenz(a,h)anthracene	1.1E+01	mg/kg	1.4E-07	5.7E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	3.E-06
				Indeno(1,2,3-cd)pyrene	3.3E+01	mg/kg	4.1E-07	1.7E-07	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	1.E-06
			Dermal	Benz(a)anthracene	2.8E+02	mg/kg	6.7E-05	3.1E-05	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	2.E-04
				Benzo(a)pyrene	6.3E+01	mg/kg	1.5E-05	7.2E-06	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	4.E-04
				Benzo(b)fluoranthene	9.4E+01	mg/kg	2.3E-05	1.1E-05	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	6.E-05
				Benzo(k)fluoranthene	3.5E+01	mg/kg	8.6E-06	4.0E-06	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	2.E-06
				Chrysene	8.7E+01	mg/kg	2.1E-05	9.9E-06	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	5.E-07
				Dibenz(a,h)anthracene	1.1E+01	mg/kg	2.7E-06	1.2E-06	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	7.E-05
				Indeno(1,2,3-cd)pyrene	3.3E+01	mg/kg	8.0E-06	3.7E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	2.E-05

TABLE 7.1 RME Supplement A
 CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
 REASONABLE MAXIMUM EXPOSURE
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Older Child Trespasser
Receptor Age:	12 to < 18 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							12-16 yrs	16-18 yrs		12-16 yrs (ADAF=3)	16-18 yrs (ADAF=1)		
Water	Surface Water	EU-1	Dermal	Benz(a)anthracene	3.8E+00	µg/L	8.7E-06	4.1E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	2.E-05
				Benzo(a)pyrene	2.2E+00	µg/L	8.7E-06	4.1E-06	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	2.E-04
				Benzo(b)fluoranthene	2.9E+00	µg/L	1.2E-05	5.4E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	3.E-05
				Benzo(k)fluoranthene	1.6E+00	µg/L			mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	
				Chrysene	2.9E+00	µg/L	6.6E-06	3.1E-06	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	2.E-07
				Indeno(1,2,3-cd)pyrene	1.4E+00	µg/L	5.7E-06	2.6E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	1.E-05

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup

TABLE 7.2 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	Ingestion	2,3,7,8-TCDD Equivalent	2E-05	mg/kg	3E-09	mg/kg-day	2E+05	1/(mg/kg-day)	5E-04	7E-09	mg/kg-day	1E-09	mg/kg-day	7E+00		
				ANTIMONY	1E+00	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	4E-04	mg/kg-day	9E-01		
				ARSENIC	8E-02	mg/kg	1E-05	mg/kg-day	2E+00	1/(mg/kg-day)	2E-05	3E-05	mg/kg-day	3E-04	mg/kg-day	1E-01		
				CHROMIUM	6E-01	mg/kg	9E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-03	mg/kg-day	7E-02		
				CYANIDE	6E+00	mg/kg	9E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-02	mg/kg-day	1E-01		
				MANGANESE	3E+00	mg/kg	5E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	1E-01	mg/kg-day	8E-03		
				MERCURY (AS METHYLMERCURY)	1E+00	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	1E-04	mg/kg-day	4E+00		
				SELENIUM	2E+00	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	5E-03	mg/kg-day	1E-01		
				VANADIUM	6E-01	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	9E-03	mg/kg-day	3E-02		
				ZINC	4E+01	mg/kg	7E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	3E-01	mg/kg-day	5E-02		
				HIGHLY CHLORINATED PCBs	6E-01	mg/kg	9E-05	mg/kg-day	2E+00	1/(mg/kg-day)	2E-04	2E-04	mg/kg-day	2E-05	mg/kg-day	1E+01		
				LESS CHLORINATED PCBs	5E-01	mg/kg	7E-05	mg/kg-day	2E+00	1/(mg/kg-day)	1E-04	2E-04	mg/kg-day	7E-05	mg/kg-day	2E+00		
				4,4-DDD	1E-02	mg/kg	2E-06	mg/kg-day	2E-01	1/(mg/kg-day)	5E-07	5E-06	mg/kg-day		mg/kg-day			
				4,4'-DDT	1E-02	mg/kg	1E-06	mg/kg-day	3E-01	1/(mg/kg-day)	5E-07	3E-06	mg/kg-day	5E-04	mg/kg-day	7E-03		
				ALDRIN	3E-03	mg/kg	4E-07	mg/kg-day	2E+01	1/(mg/kg-day)	7E-06	9E-07	mg/kg-day	3E-05	mg/kg-day	3E-02		
				DELTA-BHC	3E-03	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day		mg/kg-day			
				DIELDRIN	4E-03	mg/kg	6E-07	mg/kg-day	2E+01	1/(mg/kg-day)	9E-06	1E-06	mg/kg-day	5E-05	mg/kg-day	3E-02		
				HEPTACHLOR EPOXIDE	4E-03	mg/kg	6E-07	mg/kg-day	9E+00	1/(mg/kg-day)	6E-06	1E-06	mg/kg-day	1E-05	mg/kg-day	1E-01		
				BIS(2-ETHYLHEXYL)PHTHALATE	2E+00	mg/kg	4E-04	mg/kg-day	1E-02	1/(mg/kg-day)	5E-06	8E-04	mg/kg-day	2E-02	mg/kg-day	4E-02		
				HEXACHLOROBENZENE	1E-02	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	3E-06	5E-06	mg/kg-day	8E-04	mg/kg-day	6E-03		
				Exp. Route Total						8E-04					3E+01			
				Exp. Point Total						8E-04					3E+01			
				Exp. Medium Total						8E-04					3E+01			
Medium Total							8E-04					3E+01						
Sediment	Surface Sediment	Exposure Unit 1	Dermal	2,3,7,8-TCDD Equivalent	7E-05	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	4E-07	6E-12	mg/kg-day	1E-09	mg/kg-day	6E-03		
				ARSENIC	7E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	4E-07	6E-07	mg/kg-day	3E-04	mg/kg-day	2E-03		
				CADMIUM	1E+00	mg/kg	2E-09	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day	3E-05	mg/kg-day	2E-04		
				CHROMIUM	5E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day			
				LEAD	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day			
				MERCURY	5E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day			
				THALLIUM	6E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	3E-07	mg/kg-day	2E-05	mg/kg-day	1E-02		
				DIELDRIN	2E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day			
				ENDRIN KETONE	5E-02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-04	mg/kg-day			
				HEPTACHLOR EPOXIDE	8E-03	mg/kg		mg/kg-day	9E+00	1/(mg/kg-day)			mg/kg-day	1E-05	mg/kg-day			
				1-METHYLNAPHTHALENE	4E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day			
				2-METHYLNAPHTHALENE	3E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03		
				ACENAPHTHYLENE	4E+00	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05		
				BENZ(A)ANTHRACENE	3E+02	mg/kg	4E-05	mg/kg-day	7E-01	1/(mg/kg-day)	3E-05	1E-04	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	6E+01	mg/kg	1E-05	mg/kg-day	7E+00	1/(mg/kg-day)	7E-05	2E-05	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	9E+01	mg/kg	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	1E-05	3E-05	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	4E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	5E-04		
				BENZO(K)FLUORANTHENE	4E+01	mg/kg	6E-06	mg/kg-day	7E-02	1/(mg/kg-day)	4E-07	1E-05	mg/kg-day		mg/kg-day			
				BIS(2-ETHYLHEXYL)PHTHALATE	4E+01	mg/kg	5E-06	mg/kg-day	1E-02	1/(mg/kg-day)	6E-08	1E-05	mg/kg-day	2E-02	mg/kg-day	5E-04		
				CARBAZOLE	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day			
				CHRYSENE	9E+01	mg/kg	1E-05	mg/kg-day	7E-03	1/(mg/kg-day)	1E-07	3E-05	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	4E-06	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	2E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	1E-03	mg/kg-day	6E-03		
				FLUORANTHENE	1E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	4E-02	mg/kg-day	1E-03		
				HEXACHLOROBENZENE	1E-01	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-08	4E-08	mg/kg-day	8E-04	mg/kg-day	4E-05		
				INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	5E-06	mg/kg-day	7E-01	1/(mg/kg-day)	4E-06	1E-05	mg/kg-day		mg/kg-day			
				NAPHTHALENE	6E+01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day			
				PHENANTHRENE	1E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-02	mg/kg-day	1E-03		
				PYRENE	2E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	3E-02	mg/kg-day	2E-03		
				1,2,3-TRICHLOROBENZENE	5E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	5E-01	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day			

TABLE 7.2 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Sediment	Surface Sediment	Exposure Unit 1	Dermal	1,2-DICHLOROBENZENE	1E+01	mg/kg												
				1,3,5-TRICHLOROBENZENE	2E+01	mg/kg												
				1,4-DICHLOROBENZENE	2E+01	mg/kg					5E-03	1/(mg/kg-day)				7E-02	mg/kg-day	
				BENZENE	4E+00	mg/kg					6E-02	1/(mg/kg-day)				4E-03	mg/kg-day	
				CHLOROBENZENE	2E+01	mg/kg						1/(mg/kg-day)				2E-02	mg/kg-day	
				METHYLENE CHLORIDE	4E-01	mg/kg					8E-03	1/(mg/kg-day)				6E-02	mg/kg-day	
				N-HEXADACANE	8E-01	mg/kg						1/(mg/kg-day)					mg/kg-day	
				P-ISOPROPYLTOLUENE	9E-03	mg/kg						1/(mg/kg-day)					mg/kg-day	
				TOLUENE	1E+01	mg/kg						1/(mg/kg-day)				8E-02	mg/kg-day	
				XYLENES, TOTAL	3E+01	mg/kg						1/(mg/kg-day)				2E-01	mg/kg-day	
			Exp. Route Total										1E-04					4E-02
			Ingestion	2,3,7,8-TCDD Equivalent	7E-05	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	3E-07	5E-12	mg/kg-day	1E-09	mg/kg-day		5E-03	
				ARSENIC	7E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	5E-07	mg/kg-day	3E-04	mg/kg-day		2E-03	
				CADMIUM	1E+00	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-05	mg/kg-day		5E-03	
				CHROMIUM	5E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	8E-05	mg/kg-day		5E-02	
				IRON	1E+04	mg/kg	4E-04	mg/kg-day		1/(mg/kg-day)		9E-04	mg/kg-day	7E-01	mg/kg-day		1E-03	
				LEAD	1E+02	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day		mg/kg-day			
				MANGANESE	3E+02	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	6E-03	mg/kg-day		4E-03	
				MERCURY	5E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	2E-05	mg/kg-day		2E-02	
				THALLIUM	6E-01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	8E-05	mg/kg-day		6E-04	
				VANADIUM	1E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	2E-04	mg/kg-day		5E-03	
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	6E-08	mg/kg-day	2E-05	mg/kg-day		3E-03	
				DIELDRIN	2E-02	mg/kg	6E-10	mg/kg-day	2E+01	1/(mg/kg-day)	9E-09	1E-09	mg/kg-day	5E-05	mg/kg-day		3E-05	
				ENDRIN KETONE	5E-02	mg/kg	2E-09	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day	3E-04	mg/kg-day		1E-05	
				HEPTACHLOR EPOXIDE	8E-03	mg/kg	3E-10	mg/kg-day	9E+00	1/(mg/kg-day)	2E-09	6E-10	mg/kg-day	1E-05	mg/kg-day		5E-05	
				1-METHYLNAPHTHALENE	4E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day			
				2-METHYLNAPHTHALENE	3E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	4E-03	mg/kg-day		6E-04	
				ACENAPHTHYLENE	4E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-02	mg/kg-day		1E-05	
				BENZ(A)ANTHRACENE	3E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	6E+01	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	2E-05	5E-06	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	9E+01	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	8E-06	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	4E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day		1E-04	
				BENZO(K)FLUORANTHENE	4E+01	mg/kg	1E-06	mg/kg-day	7E-02	1/(mg/kg-day)	9E-08	3E-06	mg/kg-day		mg/kg-day			
				BIS(2-ETHYLHEXYL)PHTHALATE	4E+01	mg/kg	1E-06	mg/kg-day	1E-02	1/(mg/kg-day)	2E-08	3E-06	mg/kg-day	2E-02	mg/kg-day		2E-04	
				CARBAZOLE	1E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day			
				CHRYSENE	9E+01	mg/kg	3E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	7E-06	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	4E-07	mg/kg-day	7E+00	1/(mg/kg-day)	3E-06	9E-07	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	2E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	1E-03	mg/kg-day		2E-03	
				FLUORANTHENE	1E+02	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-02	mg/kg-day		3E-04	
				HEXACHLOROBENZENE	1E-01	mg/kg	4E-09	mg/kg-day	2E+00	1/(mg/kg-day)	7E-09	1E-08	mg/kg-day	8E-04	mg/kg-day		1E-05	
				INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	8E-07	3E-06	mg/kg-day		mg/kg-day			
				NAPHTHALENE	6E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	2E-02	mg/kg-day		2E-04	
				PHENANTHRENE	1E+02	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-02	mg/kg-day		3E-04	
				PYRENE	2E+02	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day		5E-04	
				1,2,3-TRICHLOROBENZENE	5E-01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	5E-01	mg/kg	2E-08	mg/kg-day	4E-03	1/(mg/kg-day)	7E-11	4E-08	mg/kg-day	1E-02	mg/kg-day		4E-06	
				1,2-DICHLOROBENZENE	1E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day			
				1,3,5-TRICHLOROBENZENE	2E+01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day			
				1,4-DICHLOROBENZENE	2E+01	mg/kg	8E-07	mg/kg-day	5E-03	1/(mg/kg-day)	4E-09	2E-06	mg/kg-day	7E-02	mg/kg-day		3E-05	
				BENZENE	4E+00	mg/kg	1E-07	mg/kg-day	6E-02	1/(mg/kg-day)	8E-09	3E-07	mg/kg-day	4E-03	mg/kg-day		8E-05	
				CHLOROBENZENE	2E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	2E-02	mg/kg-day		9E-05	
				METHYLENE CHLORIDE	4E-01	mg/kg	1E-08	mg/kg-day	8E-03	1/(mg/kg-day)	1E-10	3E-08	mg/kg-day	6E-02	mg/kg-day		6E-07	
				N-HEXADACANE	8E-01	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day		mg/kg-day			
				P-ISOPROPYLTOLUENE	9E-03	mg/kg	3E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day			
				TOLUENE	1E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	8E-02	mg/kg-day		1E-05	
				XYLENES, TOTAL	3E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	2E-01	mg/kg-day		1E-05	
			Exp. Route Total										3E-05					1E-01
			Exp. Point Total										2E-04					1E-01
			Exp. Medium Total										2E-04					1E-01
			Medium Total										2E-04					1E-01

TABLE 7.2 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 1	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	2E-11	mg/kg-day	2E+05	1/(mg/kg-day)	3E-06	4E-11	mg/kg-day	1E-09	mg/kg-day	4E-02
				ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ANTIMONY	7E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-05	mg/kg-day	
				ARSENIC	9E+00	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	8E-07	mg/kg-day	3E-04	mg/kg-day	3E-03
				BARIIUM	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				CADMIUM	2E+01	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day	3E-05	mg/kg-day	3E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				SILVER	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				THALLIUM	7E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	1E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	6E-07	mg/kg-day	2E-05	mg/kg-day	3E-02
				LESS CHLORINATED PCBs	9E-01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	4E-07	mg/kg-day	7E-05	mg/kg-day	5E-03
				DIELDRIN	1E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				2-METHYLNAPHTHALENE	9E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	4E-03	mg/kg-day	8E-04
				ACENAPHTHYLENE	5E+00	mg/kg	9E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	7E-05
				BENZ(A)ANTHRACENE	2E+01	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	6E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	1E+01	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	2E-05	5E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	5E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZO(K)FLUORANTHENE	1E+01	mg/kg	2E-06	mg/kg-day	7E-02	1/(mg/kg-day)	1E-07	4E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	2E+01	mg/kg	2E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	5E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	mg/kg	5E-07	mg/kg-day	7E+00	1/(mg/kg-day)	4E-06	1E-06	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	3E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	1E-03	mg/kg-day	9E-04
				FLUORANTHENE	3E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-02	mg/kg-day	3E-04
				HEXACHLOROBENZENE	7E-01	mg/kg	8E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	2E-07	mg/kg-day	8E-04	mg/kg-day	2E-04
				INDENO(1,2,3-CD)PYRENE	9E+00	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	3E-06	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	2E-02	mg/kg-day	3E-04
				PHENANTHRENE	2E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-02	mg/kg-day	3E-04
				1,2,3-TRICHLOROBENZENE	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	3E+00	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBENZENE	5E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	2E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				BENZENE	4E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	3E-03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				Exp. Route Total							3E-05					9E-02
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	2E-11	mg/kg-day	2E+05	1/(mg/kg-day)	3E-06	4E-11	mg/kg-day	1E-09	mg/kg-day	4E-02
				ALUMINUM	7E+03	mg/kg	3E-04	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	1E+00	mg/kg-day	6E-04
				ANTIMONY	7E-01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	4E-04	mg/kg-day	1E-04
				ARSENIC	9E+00	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	8E-07	mg/kg-day	3E-04	mg/kg-day	3E-03
				BARIIUM	3E+02	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-01	mg/kg-day	1E-04
				CADMIUM	2E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	1E-03	mg/kg-day	2E-03
				CHROMIUM	1E+02	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-03	mg/kg-day	3E-03
				COPPER	2E+02	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	4E-02	mg/kg-day	4E-04
				IRON	1E+04	mg/kg	5E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	7E-01	mg/kg-day	2E-03
				LEAD	4E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	1E-01	mg/kg-day	2E-04
				MERCURY	8E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				SILVER	1E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	5E-03	mg/kg-day	2E-04
				THALLIUM	7E-01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	8E-05	mg/kg-day	7E-04
				VANADIUM	2E+01	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	9E-03	mg/kg-day	2E-04
				HIGHLY CHLORINATED PCBs	1E+00	mg/kg	5E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	1E-07	mg/kg-day	2E-05	mg/kg-day	6E-03
				LESS CHLORINATED PCBs	9E-01	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	6E-08	7E-08	mg/kg-day	7E-05	mg/kg-day	1E-03
				DIELDRIN	1E-02	mg/kg	4E-10	mg/kg-day	2E+01	1/(mg/kg-day)	6E-09	9E-10	mg/kg-day	5E-05	mg/kg-day	2E-05
				2-METHYLNAPHTHALENE	9E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	4E-03	mg/kg-day	2E-04

TABLE 7.2 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Soil	Surface Soil	Exposure Unit 1	Ingestion	ACENAPHTHYLENE	5E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day		mg/kg-day	1E-05	
				BENZ(A)ANTHRACENE	2E+01	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	4E-07	1E-06	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	1E+01	mg/kg	5E-07	mg/kg-day	7E+00	1/(mg/kg-day)	4E-06	1E-06	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	3E-07	1E-06	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	1E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	3E-02	mg/kg-day	3E-05	
				BENZO(K)FLUORANTHENE	1E+01	mg/kg	4E-07	mg/kg-day	7E-02	1/(mg/kg-day)	3E-08	1E-06	mg/kg-day		mg/kg-day		
				CHRYSENE	2E+01	mg/kg	5E-07	mg/kg-day	7E-03	1/(mg/kg-day)	4E-09	1E-06	mg/kg-day		mg/kg-day		
				DIBENZ(A,H)ANTHRACENE	3E+00	mg/kg	1E-07	mg/kg-day	7E+00	1/(mg/kg-day)	8E-07	3E-07	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	3E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	1E-03	mg/kg-day	3E-04	
				FLUORANTHENE	3E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	4E-02	mg/kg-day	7E-05	
				HEXACHLORO BENZENE	7E-01	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	5E-08	mg/kg-day	8E-04	mg/kg-day	7E-05	
				INDENO(1,2,3-CD)PYRENE	9E+00	mg/kg	3E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	7E-07	mg/kg-day		mg/kg-day		
				NAPHTHALENE	2E+01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	2E-02	mg/kg-day	6E-05	
				PHENANTHRENE	2E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	6E-05	
				1,2,3-TRICHLORO BENZENE	2E+00	mg/kg	8E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day		mg/kg-day		
				1,2,4-TRICHLORO BENZENE	3E+00	mg/kg	1E-07	mg/kg-day	4E-03	1/(mg/kg-day)	3E-10	2E-07	mg/kg-day	1E-02	mg/kg-day	2E-05	
				1,2-DICHLORO BENZENE	5E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	9E-02	mg/kg-day	5E-06	
				1,4-DICHLORO BENZENE	2E+01	mg/kg	7E-07	mg/kg-day	5E-03	1/(mg/kg-day)	4E-09	2E-06	mg/kg-day	7E-02	mg/kg-day	2E-05	
				BENZENE	4E-01	mg/kg	1E-08	mg/kg-day	6E-02	1/(mg/kg-day)	7E-10	3E-08	mg/kg-day	4E-03	mg/kg-day	7E-06	
				P-ISOPROPYLTOLUENE	3E-03	mg/kg	9E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day		
				DODECANE	8E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day		mg/kg-day		
				Exp. Route Total									9E-06				6E-02
				Exp. Point Total									4E-05				1E-01
				Exp. Medium Total									4E-05				1E-01
Medium Total									4E-05				1E-01				
Surface Soil	Outdoor Air	Exposure Unit 1	Inhalation	2,3,7,8-TCDD Equivalent	4E-07	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day		
				ALUMINUM	5E-06	mg/m3	2E-08	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	1E-03	mg/kg-day	4E-05	
				ANTIMONY	5E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		5E-12	mg/kg-day		mg/kg-day		
				ARSENIC	7E-09	mg/m3	3E-11	mg/kg-day	2E+01	1/(mg/kg-day)	5E-10	7E-11	mg/kg-day	1E-05	mg/kg-day	5E-06	
				BARIUM	2E-07	mg/m3	9E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	1E-04	mg/kg-day	1E-05	
				CADMIUM	2E-08	mg/m3	8E-11	mg/kg-day	6E+00	1/(mg/kg-day)	5E-10	2E-10	mg/kg-day		mg/kg-day		
				CHROMIUM	8E-08	mg/m3	4E-10	mg/kg-day	4E+01	1/(mg/kg-day)	2E-08	9E-10	mg/kg-day	3E-05	mg/kg-day	3E-05	
				COPPER	1E-07	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day		
				IRON	1E-05	mg/m3	5E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day		
				LEAD	3E-07	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day		
				MANGANESE	2E-07	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	1E-05	mg/kg-day	2E-04	
				MERCURY	6E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		6E-11	mg/kg-day	9E-05	mg/kg-day	7E-07	
				SILVER	7E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		8E-11	mg/kg-day		mg/kg-day		
				THALLIUM	5E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		5E-12	mg/kg-day		mg/kg-day		
				VANADIUM	2E-08	mg/m3	7E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day		
				HIGHLY CHLORINATED PCBs	1E-09	mg/m3	5E-12	mg/kg-day	2E+00	1/(mg/kg-day)	9E-12	1E-11	mg/kg-day		mg/kg-day		
				LESS CHLORINATED PCBs	7E-10	mg/m3	3E-12	mg/kg-day	2E+00	1/(mg/kg-day)	6E-12	7E-12	mg/kg-day		mg/kg-day		
				DIELDRIN	8E-12	mg/m3	4E-14	mg/kg-day	2E+01	1/(mg/kg-day)	6E-13	9E-14	mg/kg-day		mg/kg-day		
				2-METHYLNAPHTHALENE	6E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day		
				ACENAPHTHYLENE	4E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day		
				BENZ(A)ANTHRACENE	1E-08	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	1E-08	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	9E-09	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	7E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		8E-11	mg/kg-day		mg/kg-day		
				BENZO(K)FLUORANTHENE	9E-09	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		9E-11	mg/kg-day		mg/kg-day		
				CHRYSENE	1E-08	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day		
				DIBENZ(A,H)ANTHRACENE	2E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	2E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day		
				FLUORANTHENE	2E-08	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day		
				HEXACHLORO BENZENE	5E-10	mg/m3	2E-12	mg/kg-day	2E+00	1/(mg/kg-day)	3E-12	5E-12	mg/kg-day		mg/kg-day		
				INDENO(1,2,3-CD)PYRENE	7E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day		
				NAPHTHALENE	1E-08	mg/m3	5E-11	mg/kg-day	1E-01	1/(mg/kg-day)	6E-12	1E-10	mg/kg-day	9E-04	mg/kg-day	1E-07	
				PHENANTHRENE	2E-08	mg/m3	8E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day		
				1,2,3-TRICHLORO BENZENE	9E-05	mg/m3	4E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day		mg/kg-day		
				1,2,4-TRICHLORO BENZENE	1E-04	mg/m3	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day		

TABLE 7.2 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations							
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient			
							Value	Units	Value	Units		Value	Units	Value	Units				
Surface Soil	Outdoor Air	Exposure Unit 1	Inhalation	1,2-DICHLOROBENZENE	6E-04	mg/m3	3E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	4E-02	mg/kg-day	2E-04			
				1,4-DICHLOROBENZENE	2E-03	mg/m3	1E-05	mg/kg-day	4E-02	1/(mg/kg-day)	4E-07	3E-05	mg/kg-day	2E-01	mg/kg-day	1E-04			
				BENZENE	2E-04	mg/m3	9E-07	mg/kg-day	3E-02	1/(mg/kg-day)	3E-08	2E-06	mg/kg-day	9E-03	mg/kg-day	3E-04			
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day				
				DODECANE	6E-07	mg/m3	3E-09	mg/kg-day		1/(mg/kg-day)		6E-09	mg/kg-day		mg/kg-day				
				Exp. Route Total							5E-07					8E-04			
		Exp. Point Total							5E-07						8E-04				
	Exp. Medium Total								5E-07						8E-04				
Medium Total											5E-07				8E-04				
Surface Water	Surface Water	Exposure Unit 1	Dermal	ANTIMONY	2E+00	ug/l	3E-08	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day	6E-05	mg/kg-day	1E-03			
				ARSENIC	3E+00	ug/l	5E-08	mg/kg-day	2E+00	1/(mg/kg-day)	8E-08	1E-07	mg/kg-day	3E-04	mg/kg-day	4E-04			
				CHROMIUM	6E+00	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	8E-05	mg/kg-day	6E-03			
				IRON	6E+03	ug/l	9E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	7E-01	mg/kg-day	3E-04			
				LEAD	1E+01	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day				
				MANGANESE	4E+02	ug/l	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	6E-03	mg/kg-day	3E-03			
				MERCURY	1E-01	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day	2E-05	mg/kg-day	2E-04			
				THALLIUM	4E+00	ug/l	6E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	8E-05	mg/kg-day	2E-03			
				VANADIUM	2E+00	ug/l	3E-08	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	2E-04	mg/kg-day	3E-04			
				ZINC	3E+02	ug/l	3E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-01	mg/kg-day	3E-05			
				2,4-DIMETHYLPHENOL	5E+01	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	2E-02	mg/kg-day	1E-03			
				2-METHYLNAPHTHALENE	6E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day				
				3&4-METHYLPHENOL	6E+01	ug/l	8E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	5E-02	mg/kg-day	4E-04			
				ACENAPHTHENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day				
				ACENAPHTHYLENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day				
				BENZ(A)ANTHRACENE	4E+00	ug/l	6E-05	mg/kg-day	7E-01	1/(mg/kg-day)	4E-05	1E-04	mg/kg-day		mg/kg-day				
				BENZO(A)PYRENE	2E+00	ug/l	5E-05	mg/kg-day	7E+00	1/(mg/kg-day)	4E-04	1E-04	mg/kg-day		mg/kg-day				
				BENZO(B)FLUORANTHENE	3E+00	ug/l	7E-05	mg/kg-day	7E-01	1/(mg/kg-day)	5E-05	2E-04	mg/kg-day		mg/kg-day				
				BENZO(G,H,I)PERYLENE	2E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day				
				BENZO(K)FLUORANTHENE	2E+00	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day				
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+00	ug/l	1E-05	mg/kg-day	1E-02	1/(mg/kg-day)	2E-07	3E-05	mg/kg-day	2E-02	mg/kg-day	1E-03			
				CARBAZOLE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day				
				CHRYSENE	3E+00	ug/l	4E-05	mg/kg-day	7E-03	1/(mg/kg-day)	3E-07	1E-04	mg/kg-day		mg/kg-day				
				DIBENZOFURAN	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day				
				FLUORENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day				
				INDENO(1,2,3-CD)PYRENE	1E+00	ug/l	4E-05	mg/kg-day	7E-01	1/(mg/kg-day)	3E-05	8E-05	mg/kg-day		mg/kg-day				
				NAPHTHALENE	1E+03	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-02	mg/kg-day	1E-01			
				PHENANTHRENE	2E+01	ug/l	6E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	5E-03			
				PYRENE	6E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day				
				1,2,4-TRIMETHYLBENZENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day				
				1,3,5-TRIMETHYLBENZENE	9E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day				
				1,4-DICHLOROBENZENE	8E+00	ug/l	7E-06	mg/kg-day	5E-03	1/(mg/kg-day)	4E-08	2E-05	mg/kg-day	7E-02	mg/kg-day	2E-04			
				BENZENE	4E+01	ug/l	1E-05	mg/kg-day	6E-02	1/(mg/kg-day)	6E-07	3E-05	mg/kg-day	4E-03	mg/kg-day	7E-03			
				DICHLOROENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day				
				TOLUENE	2E+02	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	8E-02	mg/kg-day	4E-03			
				XYLENES, TOTAL	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day				
						Exp. Route Total							5E-04						1E-01
						Exp. Point Total							5E-04						1E-01
					Exp. Medium Total								5E-04						1E-01
				Medium Total											5E-04				1E-01
				Total of Receptor Risks Across All Media										2E-03	Total of Receptor Hazards Across All Media				

TABLE 7.3. RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	Inhalation	CARBAZOLE	7E-05	mg/m3	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	9E-05	mg/m3	3E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	8E-06	mg/m3	3E-08	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	1E-04	mg/m3	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				FLUORANTHENE	3E-04	mg/m3	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	
				FLUORENE	2E-04	mg/m3	6E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day		mg/kg-day	
				HEXACHLOROBENZENE	8E-07	mg/m3	3E-09	mg/kg-day	2E+00	1/(mg/kg-day)	4E-09	8E-09	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	2E-05	mg/m3	8E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day		mg/kg-day	
				NAPHTHALENE	1E-03	mg/m3	4E-06	mg/kg-day	1E-01	1/(mg/kg-day)	5E-07	1E-05	mg/kg-day	9E-04	mg/kg-day	1E-02
				PHENANTHRENE	5E-04	mg/m3	2E-06	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day		mg/kg-day	
				PYRENE	2E-04	mg/m3	8E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	6E-04	mg/m3	2E-06	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day		mg/kg-day	
				1,4-DICHLOROBENZENE	1E-02	mg/m3	5E-05	mg/kg-day	4E-02	1/(mg/kg-day)	2E-06	1E-04	mg/kg-day	2E-01	mg/kg-day	6E-04
				BENZENE	5E-03	mg/m3	2E-05	mg/kg-day	3E-02	1/(mg/kg-day)	5E-07	5E-05	mg/kg-day	9E-03	mg/kg-day	6E-03
				BROMOMETHANE	6E-04	mg/m3	2E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	1E-03	mg/kg-day	4E-03
				TOLUENE	9E-03	mg/m3	3E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	1E+00	mg/kg-day	6E-05
				XYLENES, TOTAL	8E-02	mg/m3	3E-04	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	3E-02	mg/kg-day	7E-01
				DODECANE	9E-04	mg/m3	3E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day		mg/kg-day	
				Exp. Point Total		Exp. Route Total						2E-05				
		Exp. Medium Total							2E-05					1E+00		
							2E-05					1E+00				
							2E-05					1E+00				
Medium Total									2E-05					1E+00		
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	Dermal	ALUMINUM	4E+03	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	1E+00	mg/kg-day	7E-05
				ANTIMONY	2E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	6E-05	mg/kg-day	8E-04
				ARSENIC	6E+00	ug/l	5E-08	mg/kg-day	2E+00	1/(mg/kg-day)	7E-08	1E-07	mg/kg-day	3E-04	mg/kg-day	4E-04
				BARIUM	4E+03	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	1E-02	mg/kg-day	5E-03
				CADMIUM	2E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day	3E-05	mg/kg-day	2E-03
				CHROMIUM	2E+01	ug/l	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	8E-05	mg/kg-day	1E-02
				CYANIDE	3E+01	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	2E-02	mg/kg-day	3E-05
				IRON	1E+04	ug/l	8E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	7E-01	mg/kg-day	3E-04
				LEAD	1E+01	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day	
				MANGANESE	1E+03	ug/l	9E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	6E-03	mg/kg-day	5E-03
				MERCURY	1E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	2E-05	mg/kg-day	1E-03
				SILVER	2E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	2E-04	mg/kg-day	2E-04
				VANADIUM	1E+01	ug/l	7E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	2E-04	mg/kg-day	9E-04
				HIGHLY CHLORINATED PCBs	7E-02	ug/l		mg/kg-day	2E+00	1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				4,4'-DDT	2E-02	ug/l	9E-08	mg/kg-day	3E-01	1/(mg/kg-day)	3E-08	2E-07	mg/kg-day	5E-04	mg/kg-day	5E-04
				1,1'-BIPHENYL	2E-02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	5E-02	mg/kg-day	
				2,4-DICHLOROPHENOL	1E-02	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		7E-09	mg/kg-day	3E-03	mg/kg-day	2E-06
				2,4-DIMETHYLPHENOL	8E-01	ug/l	8E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	2E-02	mg/kg-day	1E-05
				2-METHYLNAPHTHALENE	1E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				2-METHYLPHENOL	2E+00	ug/l	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	5E-02	mg/kg-day	6E-06
				2-NITROPHENOL	3E-03	ug/l	1E-10	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day	
				3,4-METHYLPHENOL	6E+00	ug/l	3E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	5E-02	mg/kg-day	2E-05
				4-METHYLPHENOL	3E+00	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	5E-02	mg/kg-day	9E-06
				4-NITROPHENOL	8E-03	ug/l	3E-10	mg/kg-day		1/(mg/kg-day)		9E-10	mg/kg-day		mg/kg-day	
				ACENAPHTHENE	2E-01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day	
				ACENAPHTHYLENE	1E-01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				ANTHRACENE	1E-01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-01	mg/kg-day	
				BENZ(A)ANTHRACENE	4E-02	ug/l	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	5E-07	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-02	ug/l	3E-07	mg/kg-day	7E+00	1/(mg/kg-day)	2E-06	8E-07	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E-02	ug/l	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	6E-07	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	3E-02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				BENZO(K)FLUORANTHENE	7E-02	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)	2E-10	3E-08	mg/kg-day	2E-02	mg/kg-day	2E-06
				BIS(2-ETHYLHEXYL)PHthalate	2E-02	ug/l	1E-08	mg/kg-day	1E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				CARBAZOLE	7E-02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				CHRYSENE	3E-02	ug/l	2E-07	mg/kg-day	7E-03	1/(mg/kg-day)	1E-09	5E-07	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E-02	ug/l	1E-07	mg/kg-day	7E+00	1/(mg/kg-day)	9E-07	4E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E-01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day	
				FLUORANTHENE	4E-01	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	4E-02	mg/kg-day	5E-05
				FLUORENE	4E-01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	

TABLE 7.3. RME
 CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
 REASONABLE MAXIMUM EXPOSURE
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Surface Water	Surface Water	Exposure Unit 1	Dermal	1,4-DICHLOROBENZENE	8E+00	ug/l	3E-06	mg/kg-day	5E-03	1/(mg/kg-day)	1E-08	7E-06	mg/kg-day	7E-02	mg/kg-day	1E-04		
				BENZENE	4E+01	ug/l	5E-06	mg/kg-day	6E-02	1/(mg/kg-day)	3E-07	1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03		
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day			
				TOLUENE	2E+02	ug/l	5E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	8E-02	mg/kg-day	2E-03		
				XYLENES, TOTAL	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day			
		Exp. Route Total									2E-04				7E-02			
		Exp. Point Total									2E-04				7E-02			
	Exp. Medium Total									2E-04				7E-02				
Medium Total											2E-04				7E-02			
							Total of Receptor Risks Across All Media					4E-04	Total of Receptor Hazards Across All Media					8E+00

TABLE 7.3a. RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations							
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient			
							Value	Units	Value	Units		Value	Units	Value	Units				
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	3E-13	mg/kg-day	2E+05	1/(mg/kg-day)	4E-08	7E-13	mg/kg-day	1E-09	mg/kg-day	7E-04			
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day				
				ARSENIC	6E+00	mg/kg	5E-08	mg/kg-day	2E+00	1/(mg/kg-day)	7E-08	1E-07	mg/kg-day	3E-04	mg/kg-day	5E-04			
				CADMIUM	2E+01	mg/kg	5E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	3E-05	mg/kg-day	6E-04			
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day				
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day				
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day				
				LEAD	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day				
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day				
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day				
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day				
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	4E-08	mg/kg-day	2E+00	1/(mg/kg-day)	7E-08	1E-07	mg/kg-day	2E-05	mg/kg-day	5E-03			
				LESS CHLORINATED PCBs	3E-02	mg/kg	1E-09	mg/kg-day	2E+00	1/(mg/kg-day)	2E-09	3E-09	mg/kg-day	7E-05	mg/kg-day	4E-05			
				ACENAPHTHYLENE	2E+00	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	8E-06			
				BENZ(A)ANTHRACENE	9E+00	mg/kg	3E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	9E-07	mg/kg-day		mg/kg-day				
				BENZO(A)PYRENE	7E+00	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	2E-06	7E-07	mg/kg-day		mg/kg-day				
				BENZO(B)FLUORANTHENE	9E+00	mg/kg	3E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	9E-07	mg/kg-day		mg/kg-day				
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	8E-06			
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	1E-07	mg/kg-day	7E-02	1/(mg/kg-day)	8E-09	3E-07	mg/kg-day		mg/kg-day				
				CHRYSENE	9E+00	mg/kg	3E-07	mg/kg-day	7E-03	1/(mg/kg-day)	2E-09	9E-07	mg/kg-day		mg/kg-day				
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	2E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	6E-08	mg/kg-day		mg/kg-day				
				DIBENZOFURAN	1E+00	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	1E-03	mg/kg-day	1E-04			
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	7E-08	mg/kg-day	7E-01	1/(mg/kg-day)	5E-08	2E-07	mg/kg-day		mg/kg-day				
			NAPHTHALENE	2E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	2E-02	mg/kg-day	9E-06				
			PHENANTHRENE	1E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05				
			BENZENE	2E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day					
						Exp. Route Total					3E-06						7E-03		
						Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	3E-12	mg/kg-day	2E+05	1/(mg/kg-day)	4E-07	8E-12	mg/kg-day	1E-09	mg/kg-day	8E-03
					ALUMINUM		5E+03	mg/kg	5E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	1E+00	mg/kg-day	1E-03	
					ARSENIC		6E+00	mg/kg	6E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	2E-06	mg/kg-day	3E-04	mg/kg-day	5E-03	
					CADMIUM		2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	1E-03	mg/kg-day	5E-03	
					CHROMIUM		1E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-03	mg/kg-day	1E-02	
					COPPER		1E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	4E-02	mg/kg-day	8E-04	
					IRON		1E+04	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	7E-01	mg/kg-day	5E-03	
					LEAD		2E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day		mg/kg-day		
					MANGANESE		3E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	1E-01	mg/kg-day	6E-04	
					MERCURY		2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	3E-04	mg/kg-day	2E-03	
					VANADIUM		1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	9E-03	mg/kg-day	4E-04	
					HIGHLY CHLORINATED PCBs		9E-01	mg/kg	9E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	2E-07	mg/kg-day	2E-05	mg/kg-day	1E-02	
					LESS CHLORINATED PCBs		3E-02	mg/kg	3E-09	mg/kg-day	2E+00	1/(mg/kg-day)	5E-09	7E-09	mg/kg-day	7E-05	mg/kg-day	1E-04	
					ACENAPHTHYLENE		2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	3E-02	mg/kg-day	2E-05	
					BENZ(A)ANTHRACENE		9E+00	mg/kg	8E-07	mg/kg-day	7E-01	1/(mg/kg-day)	6E-07	2E-06	mg/kg-day		mg/kg-day		
					BENZO(A)PYRENE		7E+00	mg/kg	6E-07	mg/kg-day	7E+00	1/(mg/kg-day)	5E-06	2E-06	mg/kg-day		mg/kg-day		
					BENZO(B)FLUORANTHENE		9E+00	mg/kg	9E-07	mg/kg-day	7E-01	1/(mg/kg-day)	6E-07	2E-06	mg/kg-day		mg/kg-day		
					BENZO(G,H,I)PERYLENE		2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	3E-02	mg/kg-day	2E-05	
					BENZO(K)FLUORANTHENE		3E+00	mg/kg	3E-07	mg/kg-day	7E-02	1/(mg/kg-day)	2E-08	8E-07	mg/kg-day		mg/kg-day		
					CHRYSENE		9E+00	mg/kg	8E-07	mg/kg-day	7E-03	1/(mg/kg-day)	6E-09	2E-06	mg/kg-day		mg/kg-day		
					DIBENZ(A,H)ANTHRACENE		6E-01	mg/kg	6E-08	mg/kg-day	7E+00	1/(mg/kg-day)	4E-07	2E-07	mg/kg-day		mg/kg-day		
					DIBENZOFURAN		1E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	1E-03	mg/kg-day	4E-04	
					INDENO(1,2,3-CD)PYRENE		2E+00	mg/kg	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	5E-07	mg/kg-day		mg/kg-day		
					NAPHTHALENE	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	2E-02	mg/kg-day	2E-05		
					PHENANTHRENE	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04		
					BENZENE	2E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)	9E-12	5E-10	mg/kg-day	4E-03	mg/kg-day	1E-07		
						Exp. Route Total						8E-06					5E-02		
		Exp. Point Total							1E-05					6E-02					
	Exp. Medium Total								1E-05					6E-02					
Medium Total										1E-05				6E-02					

TABLE 7.3a. RME
 CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
 REASONABLE MAXIMUM EXPOSURE
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
				PYRENE	7E+00	ug/l										
				1,4-DICHLOROBENZENE	3E-01	ug/l	1E-07	mg/kg-day	5E-03	1/(mg/kg-day)	6E-10	3E-07	mg/kg-day	7E-02	mg/kg-day	4E-06
				BENZENE	9E-01	ug/l	1E-07	mg/kg-day	6E-02	1/(mg/kg-day)	5E-09	3E-07	mg/kg-day	4E-03	mg/kg-day	7E-05
		Exp. Point Total	Exp. Route Total							4E-04					1E-01	
	Exp. Medium Total									4E-04					1E-01	
Medium Total										4E-04					1E-01	
					Total of Receptor Risks Across All Media					4E-04	Total of Receptor Hazards Across All Media					2E-01

TABLE 7.4 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Surface Water	Surface Water	Exposure Unit 1	Dermal	ANTIMONY	2E+00	ug/l	7E-09	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	6E-05	mg/kg-day	8E-03	
				ARSENIC	3E+00	ug/l	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-08	8E-07	mg/kg-day	3E-04	mg/kg-day	3E-03	
				CHROMIUM	6E+00	ug/l	4E-08	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	8E-05	mg/kg-day	4E-02	
				IRON	6E+03	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	7E-01	mg/kg-day	2E-03	
				LEAD	1E+01	ug/l	4E-09	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day		
				MANGANESE	4E+02	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	6E-03	mg/kg-day	2E-02	
				MERCURY	1E-01	ug/l	4E-10	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	2E-05	mg/kg-day	1E-03	
				THALLIUM	4E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	8E-05	mg/kg-day	1E-02	
				VANADIUM	2E+00	ug/l	6E-09	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	2E-04	mg/kg-day	2E-03	
				ZINC	3E+02	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	3E-01	mg/kg-day	2E-04	
				2,4-DIMETHYLPHENOL	5E+01	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	2E-02	mg/kg-day	8E-03	
				2-METHYLNAPHTHALENE	6E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day		
				3&4-METHYLPHENOL	6E+01	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	5E-02	mg/kg-day	2E-03	
				ACENAPHTHENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day		
				ACENAPHTHYLENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				BENZ(A)ANTHRACENE	4E+00	ug/l	9E-06	mg/kg-day	7E-01	1/(mg/kg-day)	7E-06	6E-04	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	2E+00	ug/l	8E-06	mg/kg-day	7E+00	1/(mg/kg-day)	6E-05	6E-04	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	3E+00	ug/l	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	9E-06	8E-04	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	2E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				BENZO(K)FLUORANTHENE	2E+00	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+00	ug/l	2E-06	mg/kg-day	1E-02	1/(mg/kg-day)	2E-08	1E-04	mg/kg-day	2E-02	mg/kg-day	6E-03	
				CARBAZOLE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				CHRYSENE	3E+00	ug/l	7E-06	mg/kg-day	7E-03	1/(mg/kg-day)	5E-08	5E-04	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day		
				FLUORENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day		
				INDENO(1,2,3-CD)PYRENE	1E+00	ug/l	6E-06	mg/kg-day	7E-01	1/(mg/kg-day)	4E-06	4E-04	mg/kg-day		mg/kg-day		
				NAPHTHALENE	1E+03	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	2E-02	mg/kg-day	7E-01	
				PHENANTHRENE	2E+01	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day	3E-02	mg/kg-day	2E-02	
				PYRENE	6E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				1,2,4-TRIMETHYLBENZENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				1,3,5-TRIMETHYLBENZENE	9E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				1,4-DICHLOROBENZENE	8E+00	ug/l	1E-06	mg/kg-day	5E-03	1/(mg/kg-day)	7E-09	9E-05	mg/kg-day	7E-02	mg/kg-day	1E-03	
				BENZENE	4E+01	ug/l	2E-06	mg/kg-day	6E-02	1/(mg/kg-day)	1E-07	2E-04	mg/kg-day	4E-03	mg/kg-day	4E-02	
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day		
				TOLUENE	2E+02	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	8E-02	mg/kg-day	2E-02	
				XYLENES, TOTAL	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day		
						Exp. Route Total								8E-05			
				Exp. Point Total									8E-05				9E-01
			Exp. Medium Total										8E-05				9E-01
		Medium Total											8E-05				9E-01
Total of Receptor Risks Across All Media											2E-04	Total of Receptor Hazards Across All Media					3E+01

TABLE 7.4a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS -SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day			
				ALUMINUM	2E-06	mg/m3	8E-09	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	1E-03	mg/kg-day	4E-04		
				ARSENIC	3E-09	mg/m3	9E-12	mg/kg-day	2E+01	1/(mg/kg-day)	1E-10	7E-10	mg/kg-day	1E-05	mg/kg-day	5E-05		
				CADMIUM	8E-09	mg/m3	3E-11	mg/kg-day	6E+00	1/(mg/kg-day)	2E-10	2E-09	mg/kg-day		mg/kg-day			
				CHROMIUM	6E-08	mg/m3	2E-10	mg/kg-day	4E+01	1/(mg/kg-day)	1E-08	2E-08	mg/kg-day	3E-05	mg/kg-day	6E-04		
				COPPER	5E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day			
				IRON	6E-06	mg/m3	2E-08	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day			
				LEAD	7E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day		mg/kg-day			
				MANGANESE	1E-07	mg/m3	5E-10	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day	1E-05	mg/kg-day	2E-03		
				MERCURY	8E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day	9E-05	mg/kg-day	2E-06		
				VANADIUM	6E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day			
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	1E-12	mg/kg-day	2E+00	1/(mg/kg-day)	3E-12	1E-10	mg/kg-day		mg/kg-day			
				LESS CHLORINATED PCBs	1E-11	mg/m3	4E-14	mg/kg-day	2E+00	1/(mg/kg-day)	9E-14	3E-12	mg/kg-day		mg/kg-day			
				ACENAPHTHYLENE	1E-09	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				BENZ(A)ANTHRACENE	4E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	3E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				CHRYSENE	4E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	6E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				NAPHTHALENE	8E-10	mg/m3	3E-12	mg/kg-day	1E-01	1/(mg/kg-day)	3E-13	2E-10	mg/kg-day	9E-04	mg/kg-day	2E-07		
				PHENANTHRENE	6E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day			
				BENZENE	9E-07	mg/m3	3E-09	mg/kg-day	3E-02	1/(mg/kg-day)	9E-11	2E-07	mg/kg-day	9E-03	mg/kg-day	3E-05		
				Exp. Point Total			Exp. Route Total							1E-08				4E-03
												1E-08				4E-03		
		Exp. Medium Total										1E-08				4E-03		
		Medium Total											1E-08				4E-03	
		Ground Water	Shallow Ground Water	Exposure Unit 9	Dermal	ALUMINUM	4E+04	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	1E+00	mg/kg-day	1E-02
						ANTIMONY	6E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	6E-05	mg/kg-day	2E-02
	ARSENIC					2E+01	ug/l	7E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	5E-06	mg/kg-day	3E-04	mg/kg-day	2E-02	
BARIUM	8E+02					ug/l	3E-06	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-02	mg/kg-day	2E-02		
CADMIUM	1E+01					ug/l	4E-08	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-05	mg/kg-day	1E-01		
CHROMIUM	2E+02					ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	8E-05	mg/kg-day	1E+00		
COPPER	3E+02					ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	4E-02	mg/kg-day	2E-03		
IRON	5E+04					ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	7E-01	mg/kg-day	2E-02		
LEAD	7E+02					ug/l	3E-07	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day			
MANGANESE	1E+03					ug/l	4E-06	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	6E-03	mg/kg-day	5E-02		
MERCURY	2E+00					ug/l	8E-09	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	2E-05	mg/kg-day	3E-02		
NICKEL	7E+01					ug/l	5E-08	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	8E-04	mg/kg-day	4E-03		
SELENIUM	1E+01					ug/l	4E-08	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	5E-03	mg/kg-day	5E-04		
THALLIUM	2E+01					ug/l	8E-08	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	8E-05	mg/kg-day	7E-02		
VANADIUM	7E+01					ug/l	3E-07	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-04	mg/kg-day	8E-02		
ZINC	5E+02					ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	3E-01	mg/kg-day	2E-04		
4-NITROPHENOL	1E+00					ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day		mg/kg-day			
ACENAPHTHENE	1E+01					ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day			
ACENAPHTHYLENE	4E+00					ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day			
ATRAZINE	5E+01					ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day			
BENZ(A)ANTHRACENE	5E+00					ug/l	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	9E-06	8E-04	mg/kg-day		mg/kg-day			
BENZO(A)PYRENE	6E+00					ug/l	2E-05	mg/kg-day	7E+00	1/(mg/kg-day)	2E-04	2E-03	mg/kg-day		mg/kg-day			
BENZO(B)FLUORANTHENE	7E+00					ug/l	3E-05	mg/kg-day	7E-01	1/(mg/kg-day)	2E-05	2E-03	mg/kg-day		mg/kg-day			
BENZO(G,H,I)PERYLENE	5E+00					ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day			
BENZO(K)FLUORANTHENE	5E+00					ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day			
BIS(2-ETHYLHEXYL)PHTHALATE	5E+00					ug/l	2E-06	mg/kg-day	1E-02	1/(mg/kg-day)	2E-08	1E-04	mg/kg-day	2E-02	mg/kg-day	6E-03		
CARBAZOLE	4E+00					ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
CHRYSENE	5E+00			ug/l	1E-05	mg/kg-day	7E-03	1/(mg/kg-day)	9E-08	8E-04	mg/kg-day		mg/kg-day					
NAPHTHALENE	1E+02			ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-02	mg/kg-day	7E-02				

TABLE 7.4a RME
 CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS -SYW-12
 REASONABLE MAXIMUM EXPOSURE
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Shallow Ground Water	Exposure Unit 9	Dermal	PHENANTHRENE	7E+00	ug/l	4E-06	mg/kg-day	1/(mg/kg-day)			3E-04	mg/kg-day	3E-02	mg/kg-day	9E-03
				PYRENE	7E+00	ug/l		1/(mg/kg-day)								
				1,4-DICHLOROBENZENE	3E-01	ug/l	5E-08	mg/kg-day	1/(mg/kg-day)	3E-10	4E-06	mg/kg-day	7E-02	mg/kg-day	5E-05	
				BENZENE	9E-01	ug/l	5E-08	mg/kg-day	1/(mg/kg-day)	3E-09	3E-06	mg/kg-day	4E-03	mg/kg-day	9E-04	
Exp. Medium Total																
Exp. Point Total																
Exp. Route Total																
Medium Total																

TABLE 7.6 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Ditch Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Water	Surface Water	Exposure Unit 3	Dermal	2-METHYLNAPHTHALENE	9E+01	ug/l	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-03	mg/kg-day	3E-04
				3&4-METHYLPHENOL	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	5E-02	mg/kg-day	
				ACENAPHTHYLENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				CARBAZOLE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+01	ug/l	mg/kg-day	1/(mg/kg-day)	mg/kg-day	1E-03	mg/kg-day	2E-02				
				FLUORENE	2E+01	ug/l	mg/kg-day	1/(mg/kg-day)	mg/kg-day	4E-02	mg/kg-day					
				NAPHTHALENE	8E+02	ug/l	mg/kg-day	1/(mg/kg-day)	mg/kg-day	2E-02	mg/kg-day					
				PHENANTHRENE	2E+01	ug/l	mg/kg-day	1/(mg/kg-day)	mg/kg-day	3E-02	mg/kg-day					
				1,2,4-TRIMETHYLBENZENE	7E+01	ug/l	4E-06	mg/kg-day	1/(mg/kg-day)	2E-07		mg/kg-day		mg/kg-day	3E-03	
				1,3,5-TRIMETHYLBENZENE	3E+01	ug/l		mg/kg-day	1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				BENZENE	7E+01	ug/l		mg/kg-day	6E-02	1/(mg/kg-day)	1E-05	mg/kg-day	4E-03	mg/kg-day		
				TOLUENE	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)		mg/kg-day	8E-02	mg/kg-day		
				XYLENES, TOTAL	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)		mg/kg-day	2E-01	mg/kg-day		
			Exp. Route Total								2E-07					3E-02
		Exp. Point Total								2E-07					3E-02	
	Exp. Medium Total								2E-07					3E-02		
Medium Total								2E-07					3E-02			
Total of Receptor Risks Across All Media							2E-06	Total of Receptor Hazards Across All Media							4E-02	

TABLE 7.7 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 4	Inhalation	ALUMINUM	1E-05	mg/m3	2E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	1E-03	mg/kg-day	3E-04
				ARSENIC	2E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day	1E-05	mg/kg-day	4E-05
				BARIIUM	3E-07	mg/m3	5E-09	mg/kg-day	2E+01	1/(mg/kg-day)	3E-09	1E-08	mg/kg-day	1E-04	mg/kg-day	9E-05
				CHROMIUM	2E-08	mg/m3	3E-10	mg/kg-day	4E+01	1/(mg/kg-day)	1E-08	9E-10	mg/kg-day	3E-05	mg/kg-day	3E-05
				IRON	2E-05	mg/m3	3E-07	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day		mg/kg-day	
				LEAD	6E-07	mg/m3	8E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day		mg/kg-day	
				MANGANESE	5E-07	mg/m3	6E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-05	mg/kg-day	1E-03
				MERCURY	1E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day	9E-05	mg/kg-day	5E-07
				VANADIUM	3E-08	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		9E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	7E-11	mg/m3	9E-13	mg/kg-day	2E+00	1/(mg/kg-day)	2E-12	3E-12	mg/kg-day		mg/kg-day	
				LESS CHLORINATED PCBs	4E-12	mg/m3	5E-14	mg/kg-day	2E+00	1/(mg/kg-day)	1E-13	1E-13	mg/kg-day		mg/kg-day	
				DIELDRIN	6E-11	mg/m3	8E-13	mg/kg-day	2E+01	1/(mg/kg-day)	1E-11	2E-12	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	2E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		8E-12	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	5E-10	mg/m3	7E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	5E-10	mg/m3	6E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	6E-10	mg/m3	8E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	3E-10	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	4E-10	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		6E-12	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	3E-10	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-10	mg/m3	8E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZENE	4E-07	mg/m3	6E-09	mg/kg-day	3E-02	1/(mg/kg-day)	2E-10	2E-08	mg/kg-day	9E-03	mg/kg-day	2E-06
				P-ISOPROPYLTOLUENE	5E-12	mg/m3	7E-14	mg/kg-day		1/(mg/kg-day)		2E-13	mg/kg-day		mg/kg-day	
			Exp. Route Total								2E-08					2E-03
		Exp. Point Total									2E-08					2E-03
	Exp. Medium Total										2E-08					2E-03
Medium Total											2E-08					2E-03
Soil	Surface Soil	Exposure Unit 4	Dermal	ALUMINUM	9E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)	1E-06	2E-06	mg/kg-day	3E-04	mg/kg-day	7E-03
				BARIIUM	3E+02	mg/kg	7E-07	mg/kg-day	2E+00	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				CHROMIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				IRON	2E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	5E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	1E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	6E-02	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	4E-08	mg/kg-day	2E-05	mg/kg-day	2E-03
				LESS CHLORINATED PCBs	3E-03	mg/kg	7E-10	mg/kg-day	2E+00	1/(mg/kg-day)	1E-09	2E-09	mg/kg-day	7E-05	mg/kg-day	3E-05
				DIELDRIN	5E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				ACENAPHTHYLENE	2E-01	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day	4E-06
				BENZ(A)ANTHRACENE	4E-01	mg/kg	9E-08	mg/kg-day	7E-01	1/(mg/kg-day)	7E-08	3E-07	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	4E-01	mg/kg	9E-08	mg/kg-day	7E+00	1/(mg/kg-day)	7E-07	3E-07	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	5E-01	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	8E-08	3E-07	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E-01	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	5E-06
				BENZO(K)FLUORANTHENE	3E-01	mg/kg	7E-08	mg/kg-day	7E-02	1/(mg/kg-day)	5E-09	2E-07	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E-01	mg/kg	3E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	9E-08	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	2E-01	mg/kg	6E-08	mg/kg-day	7E-01	1/(mg/kg-day)	4E-08	2E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	5E-01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-02	mg/kg-day	1E-05
				BENZENE	1E-03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
			Exp. Route Total								2E-06					9E-03

TABLE 7.7 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Soil	Surface Soil	Exposure Unit 4	Ingestion	ALUMINUM	9E+03	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)	5E-06	7E-03	mg/kg-day	1E+00	mg/kg-day	7E-03		
				ARSENIC	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-04	mg/kg-day	3E-02		
				BARIUM	3E+02	mg/kg	8E-05	mg/kg-day	2E+00	1/(mg/kg-day)		2E-04	mg/kg-day	2E-01	mg/kg-day	1E-03		
				CHROMIUM	2E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-03	mg/kg-day	5E-03		
				IRON	2E+04	mg/kg	5E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	7E-01	mg/kg-day	2E-02		
				LEAD	5E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day		mg/kg-day			
				MANGANESE	4E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-01	mg/kg-day	2E-03		
				MERCURY	1E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	3E-04	mg/kg-day	2E-03		
				VANADIUM	2E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	9E-03	mg/kg-day	2E-03		
				HIGHLY CHLORINATED PCBs	6E-02	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	4E-08	mg/kg-day	2E-05	mg/kg-day	2E-03		
				LESS CHLORINATED PCBs	3E-03	mg/kg	8E-10	mg/kg-day	2E+00	1/(mg/kg-day)	2E-09	2E-09	mg/kg-day	7E-05	mg/kg-day	3E-05		
				DIELDRIN	5E-02	mg/kg	1E-08	mg/kg-day	2E+01	1/(mg/kg-day)	2E-07	4E-08	mg/kg-day	5E-05	mg/kg-day	7E-04		
				ACENAPHTHYLENE	2E-01	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-02	mg/kg-day	4E-06		
				BENZ(A)ANTHRACENE	4E-01	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	8E-08	3E-07	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	4E-01	mg/kg	1E-07	mg/kg-day	7E+00	1/(mg/kg-day)	8E-07	3E-07	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	5E-01	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	4E-07	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	2E-01	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	6E-06		
				BENZO(K)FLUORANTHENE	3E-01	mg/kg	8E-08	mg/kg-day	7E-02	1/(mg/kg-day)	6E-09	2E-07	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	1E-01	mg/kg	4E-08	mg/kg-day	7E+00	1/(mg/kg-day)	3E-07	1E-07	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	2E-01	mg/kg	7E-08	mg/kg-day	7E-01	1/(mg/kg-day)	5E-08	2E-07	mg/kg-day		mg/kg-day			
				PHENANTHRENE	5E-01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-02	mg/kg-day	1E-05		
				BENZENE	1E-03	mg/kg	3E-10	mg/kg-day	6E-02	1/(mg/kg-day)	1E-11	7E-10	mg/kg-day	4E-03	mg/kg-day	2E-07		
				P-ISOPROPYLTOLUENE	4E-03	mg/kg	1E-09	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day			
						Exp. Route Total								7E-06				7E-02
				Exp. Point Total								9E-06				8E-02		
		Exp. Medium Total										9E-06				8E-02		
Medium Total										9E-06				8E-02				
							Total of Receptor Risks Across All Media					9E-06	Total of Receptor Hazards Across All Media					8E-02

TABLE 7.7a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	3E-08	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	4E-11	mg/kg-day	2E+01	1/(mg/kg-day)	5E-10	1E-10	mg/kg-day	1E-03	mg/kg-day	6E-05
				CADMIUM	8E-09	mg/m3	1E-10	mg/kg-day	6E+00	1/(mg/kg-day)	6E-10	3E-10	mg/kg-day	1E-05	mg/kg-day	7E-06
				CHROMIUM	5E-08	mg/m3	7E-10	mg/kg-day	4E+01	1/(mg/kg-day)	3E-08	2E-09	mg/kg-day	3E-05	mg/kg-day	7E-05
				COPPER	5E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	8E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		5E-09	mg/kg-day	1E-05	mg/kg-day	4E-04
				MERCURY	8E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day	9E-05	mg/kg-day	3E-07
				VANADIUM	6E-09	mg/m3	8E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	5E-12	mg/kg-day	2E+00	1/(mg/kg-day)	1E-11	1E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				BENZO(A)ANTHRACENE	4E-09	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		5E-11	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		9E-12	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	8E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	7E-09	mg/kg-day	3E-02	1/(mg/kg-day)	2E-10	2E-08	mg/kg-day	9E-03	mg/kg-day	2E-06
			Exp. Route Total								3E-08					5E-04
		Exp. Point Total									3E-08					5E-04
	Exp. Medium Total										3E-08					5E-04
Medium Total											3E-08					5E-04
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	2E-07	5E-12	mg/kg-day	1E-09	mg/kg-day	5E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	9E-07	mg/kg-day	3E-04	mg/kg-day	3E-03
				CADMIUM	2E+01	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day	3E-05	mg/kg-day	3E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	6E-07	mg/kg-day	2E-05	mg/kg-day	3E-02
				ACENAPHTHYLENE	2E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	4E-05
				BENZO(A)ANTHRACENE	9E+00	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	6E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	1E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	4E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	6E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	7E-07	mg/kg-day	7E-02	1/(mg/kg-day)	5E-08	2E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	2E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	6E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	1E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	4E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	1E-03	mg/kg-day	8E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	4E-07	mg/kg-day	7E-01	1/(mg/kg-day)	3E-07	1E-06	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-02	mg/kg-day	3E-04
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								2E-05					4E-02

TABLE 7.7a RME
 CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
 REASONABLE MAXIMUM EXPOSURE
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	8E-12	mg/kg-day	2E+05	1/(mg/kg-day)	1E-06	2E-11	mg/kg-day	1E-09	mg/kg-day	2E-02
				ALUMINUM	5E+03	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	1E+00	mg/kg-day	4E-03
				ARSENIC	6E+00	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	5E-06	mg/kg-day	3E-04	mg/kg-day	2E-02
				CADMIUM	2E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02
				CHROMIUM	1E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	3E-03	mg/kg-day	3E-02
				COPPER	1E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	4E-02	mg/kg-day	2E-03
				IRON	1E+04	mg/kg	4E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	7E-01	mg/kg-day	1E-02
				MANGANESE	3E+02	mg/kg	9E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	2E-03
				MERCURY	2E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-04	mg/kg-day	5E-03
				VANADIUM	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	1E-03
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	7E-07	mg/kg-day	2E-05	mg/kg-day	3E-02
				ACENAPHTHYLENE	2E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	7E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	5E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	7E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	6E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	9E-07	mg/kg-day	7E-02	1/(mg/kg-day)	6E-08	2E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	7E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	4E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	3E-07	1E-06	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	4E-04
				BENZENE	1E-03	mg/kg	3E-10	mg/kg-day	6E-02	1/(mg/kg-day)	1E-11	7E-10	mg/kg-day	4E-03	mg/kg-day	2E-07
						Exp. Route Total								2E-05		
				Exp. Point Total								4E-05				2E-01
		Exp. Medium Total									4E-05				2E-01	
Medium Total										4E-05				2E-01		
Total of Receptor Risks Across All Media											4E-05	Total of Receptor Hazards Across All Media				2E-01

TABLE 7.8 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface soil	Exposure Unit 5	Ingestion	INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	7E-06	3E-05	mg/kg-day		mg/kg-day	
				NAPHTHALENE	7E+00	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day	2E-02	mg/kg-day	4E-04
				PHENANTHRENE	4E+01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				BENZENE	8E-03	mg/kg	3E-09	mg/kg-day	6E-02	1/(mg/kg-day)	2E-10	8E-09	mg/kg-day	4E-03	mg/kg-day	2E-06
				P-ISOPROPYLTOLUENE	4E-03	mg/kg	1E-09	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day	
			Exp. Route Total								2E-04					4E-01
			Dermal	ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ANTIMONY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-05	mg/kg-day	
				ARSENIC	2E+01	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	4E-06	mg/kg-day	3E-04	mg/kg-day	1E-02
				CHROMIUM	4E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				IRON	2E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				THALLIUM	1E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	6E+00	mg/kg	3E-06	mg/kg-day	2E+00	1/(mg/kg-day)	6E-06	8E-06	mg/kg-day	2E-05	mg/kg-day	4E-01
				ENDOSULFAN SULFATE	1E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				ENDRIN ALDEHYDE	7E-02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-04	mg/kg-day	
				3&4-METHYLPHENOL	4E-02	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day	5E-02	mg/kg-day	9E-07
				ACENAPHTHYLENE	1E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	6E-04
				BENZ(A)ANTHRACENE	4E+01	mg/kg	2E-05	mg/kg-day	7E-01	1/(mg/kg-day)	1E-05	5E-05	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E+01	mg/kg	2E-05	mg/kg-day	7E+00	1/(mg/kg-day)	1E-04	4E-05	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	3E+01	mg/kg	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	1E-05	4E-05	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	3E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				BENZO(K)FLUORANTHENE	4E+01	mg/kg	2E-05	mg/kg-day	7E-02	1/(mg/kg-day)	1E-06	5E-05	mg/kg-day		mg/kg-day	
				CHRYSENE	3E+01	mg/kg	2E-05	mg/kg-day	7E-03	1/(mg/kg-day)	1E-07	4E-05	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	4E-06	mg/kg-day	7E+00	1/(mg/kg-day)	3E-05	1E-05	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	6E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	1E-03	mg/kg-day	6E-03
				FLUORANTHENE	8E+01	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	4E-02	mg/kg-day	2E-03
				INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	9E-06	4E-05	mg/kg-day		mg/kg-day	
				NAPHTHALENE	7E+00	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	2E-02	mg/kg-day	5E-04
				PHENANTHRENE	4E+01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	3E-02	mg/kg-day	2E-03
				BENZENE	8E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
			Exp. Route Total								2E-04					4E-01
			Exp. Point Total								3E-04					9E-01
			Exp. Medium Total								3E-04					9E-01
Medium Total								3E-04					9E-01			
Total of Receptor Risks Across All Media											3E-04	Total of Receptor Hazards Across All Media				9E-01

TABLE 7.9 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 7	Inhalation	2,3,7,8-TCDD Equivalent	3E-07	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day	
				ALUMINUM	4E-06	mg/m3	2E-07	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	1E-03	mg/kg-day	3E-04
				ANTIMONY	4E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		5E-11	mg/kg-day		mg/kg-day	
				ARSENIC	5E-09	mg/m3	2E-10	mg/kg-day	2E+01	1/(mg/kg-day)	4E-09	7E-10	mg/kg-day	1E-05	mg/kg-day	5E-05
				BARIIUM	2E-07	mg/m3	8E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-04	mg/kg-day	2E-04
				CADMIUM	2E-08	mg/m3	7E-10	mg/kg-day	6E+00	1/(mg/kg-day)	5E-09	2E-09	mg/kg-day		mg/kg-day	
				CHROMIUM	5E-08	mg/m3	2E-09	mg/kg-day	4E+01	1/(mg/kg-day)	1E-07	7E-09	mg/kg-day	3E-05	mg/kg-day	2E-04
				COPPER	1E-07	mg/m3	6E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day		mg/kg-day	
				IRON	8E-06	mg/m3	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				LEAD	3E-07	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day	
				MANGANESE	2E-07	mg/m3	8E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-05	mg/kg-day	1E-03
				MERCURY	5E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day	9E-05	mg/kg-day	8E-06
				SILVER	7E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day	
				THALLIUM	4E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		5E-11	mg/kg-day		mg/kg-day	
				VANADIUM	1E-08	mg/m3	5E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	8E-10	mg/m3	4E-11	mg/kg-day	2E+00	1/(mg/kg-day)	7E-11	1E-10	mg/kg-day		mg/kg-day	
				LESS CHLORINATED PCBs	4E-10	mg/m3	2E-11	mg/kg-day	2E+00	1/(mg/kg-day)	4E-11	5E-11	mg/kg-day		mg/kg-day	
				DIELDRIN	6E-11	mg/m3	3E-12	mg/kg-day	2E+01	1/(mg/kg-day)	5E-11	8E-12	mg/kg-day		mg/kg-day	
				2-METHYLNAPHTHALENE	6E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	4E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	1E-08	mg/m3	5E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	1E-08	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		9E-10	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	7E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	8E-09	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				CHRYSENE	1E-08	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E-09	mg/m3	8E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day	
				FLUORANTHENE	2E-08	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day	
				HEXACHLOROBENZENE	5E-10	mg/m3	2E-11	mg/kg-day	2E+00	1/(mg/kg-day)	3E-11	6E-11	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	6E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day	
				NAPHTHALENE	1E-08	mg/m3	5E-10	mg/kg-day	1E-01	1/(mg/kg-day)	6E-11	1E-09	mg/kg-day	9E-04	mg/kg-day	2E-06
				PHENANTHRENE	2E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				1,2,3-TRICHLOROBENZENE	1E-04	mg/m3	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E-04	mg/m3	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day		mg/kg-day	
				1,2-DICHLOROBENZENE	7E-04	mg/m3	3E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	4E-02	mg/kg-day	2E-03
				1,4-DICHLOROBENZENE	3E-03	mg/m3	1E-04	mg/kg-day	4E-02	1/(mg/kg-day)	5E-06	4E-04	mg/kg-day	2E-01	mg/kg-day	2E-03
				BENZENE	2E-04	mg/m3	1E-05	mg/kg-day	3E-02	1/(mg/kg-day)	3E-07	3E-05	mg/kg-day	9E-03	mg/kg-day	3E-03
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	5E-07	mg/m3	2E-08	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day		mg/kg-day	
				Exp. Route Total						5E-06				9E-03		
				Exp. Point Total						5E-06				9E-03		
				Exp. Medium Total						5E-06				9E-03		
				Medium Total						5E-06				9E-03		
Soil	Surface soil	Exposure Unit 7	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	5E-11	mg/kg-day	2E+05	1/(mg/kg-day)	8E-06	2E-10	mg/kg-day	1E-09	mg/kg-day	2E-01
				ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ANTIMONY	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-05	mg/kg-day	
				ARSENIC	9E+00	mg/kg	1E-06	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	3E-06	mg/kg-day	3E-04	mg/kg-day	9E-03
				BARIIUM	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				CADMIUM	3E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-05	mg/kg-day	1E-02
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	5E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				SILVER	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	

TABLE 7.9 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface soil	Exposure Unit 7	Dermal	THALLIUM	7E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	1E+00	mg/kg	7E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	2E-06	mg/kg-day	2E-05	mg/kg-day	1E-01
				LESS CHLORINATED PCBs	7E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	7E-07	1E-06	mg/kg-day	7E-05	mg/kg-day	1E-02
				DIELDRIN	1E-01	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				2-METHYLNAPHTHALENE	1E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03
				ACENAPHTHYLENE	6E+00	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-02	mg/kg-day	3E-04
				BENZ(A)ANTHRACENE	2E+01	mg/kg	8E-06	mg/kg-day	7E-01	1/(mg/kg-day)	6E-06	2E-05	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+01	mg/kg	8E-06	mg/kg-day	7E+00	1/(mg/kg-day)	6E-05	2E-05	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	6E-06	mg/kg-day	7E-01	1/(mg/kg-day)	4E-06	2E-05	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	5E-04
				BENZO(K)FLUORANTHENE	1E+01	mg/kg	6E-06	mg/kg-day	7E-02	1/(mg/kg-day)	5E-07	2E-05	mg/kg-day		mg/kg-day	
				CHRYSENE	2E+01	mg/kg	8E-06	mg/kg-day	7E-03	1/(mg/kg-day)	6E-08	2E-05	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	mg/kg	1E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	4E-06	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	1E-03	mg/kg-day	4E-03
				FLUORANTHENE	4E+01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	4E-02	mg/kg-day	1E-03
				HEXACHLOROBENZENE	8E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	8E-07	mg/kg-day	8E-04	mg/kg-day	1E-03
				INDENO(1,2,3-CD)PYRENE	1E+01	mg/kg	5E-06	mg/kg-day	7E-01	1/(mg/kg-day)	3E-06	1E-05	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-02	mg/kg-day	1E-03
				PHENANTHRENE	3E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				1,2,3-TRICHLOROBENZENE	3E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	3E+00	mg/kg		mg/kg-day	4E-03				mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBENZENE	6E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	2E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				BENZENE	4E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	3E-03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
			Exp. Route Total								9E-05					3E-01
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	2E-10	mg/kg-day	2E+05	1/(mg/kg-day)	3E-05	5E-10	mg/kg-day	1E-09	mg/kg-day	5E-01
				ALUMINUM	7E+03	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	1E+00	mg/kg-day	7E-03
				ANTIMONY	8E-01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	4E-04	mg/kg-day	2E-03
				ARSENIC	9E+00	mg/kg	3E-06	mg/kg-day	2E+00	1/(mg/kg-day)	5E-06	9E-06	mg/kg-day	3E-04	mg/kg-day	3E-02
				BARIUM	3E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	2E-01	mg/kg-day	1E-03
				CADMIUM	3E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	1E-03	mg/kg-day	3E-02
				CHROMIUM	1E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	3E-03	mg/kg-day	3E-02
				COPPER	2E+02	mg/kg	8E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	6E-03
				IRON	1E+04	mg/kg	5E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	7E-01	mg/kg-day	2E-02
				LEAD	5E+02	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-01	mg/kg-day	2E-03
				MERCURY	1E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-04	mg/kg-day	3E-02
				SILVER	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	5E-03	mg/kg-day	2E-03
				THALLIUM	7E-01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	8E-05	mg/kg-day	9E-03
				VANADIUM	2E+01	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	9E-03	mg/kg-day	2E-03
				HIGHLY CHLORINATED PCBs	1E+00	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	1E-06	mg/kg-day	2E-05	mg/kg-day	7E-02
				LESS CHLORINATED PCBs	7E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	7E-07	mg/kg-day	7E-05	mg/kg-day	1E-02
				DIELDRIN	1E-01	mg/kg	4E-08	mg/kg-day	2E+01	1/(mg/kg-day)	6E-07	1E-07	mg/kg-day	5E-05	mg/kg-day	2E-03
				2-METHYLNAPHTHALENE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03
				ACENAPHTHYLENE	6E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZ(A)ANTHRACENE	2E+01	mg/kg	6E-06	mg/kg-day	7E-01	1/(mg/kg-day)	5E-06	2E-05	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+01	mg/kg	6E-06	mg/kg-day	7E+00	1/(mg/kg-day)	4E-05	2E-05	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	4E-06	mg/kg-day	7E-01	1/(mg/kg-day)	3E-06	1E-05	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	4E-04
				BENZO(K)FLUORANTHENE	1E+01	mg/kg	5E-06	mg/kg-day	7E-02	1/(mg/kg-day)	4E-07	1E-05	mg/kg-day		mg/kg-day	
				CHRYSENE	2E+01	mg/kg	6E-06	mg/kg-day	7E-03	1/(mg/kg-day)	4E-08	2E-05	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	mg/kg	1E-06	mg/kg-day	7E+00	1/(mg/kg-day)	8E-06	3E-06	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	1E-03	mg/kg-day	4E-03

TABLE 7.9 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Soil	Surface soil	Exposure Unit 7	Ingestion	FLUORANTHENE	4E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	4E-02	mg/kg-day	9E-04		
				HEXACHLOROBENZENE	8E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	8E-07	mg/kg-day	8E-04	mg/kg-day	1E-03		
				INDENO(1,2,3-CD)PYRENE	1E+01	mg/kg	4E-06	mg/kg-day	7E-01	1/(mg/kg-day)	3E-06	1E-05	mg/kg-day		mg/kg-day			
				NAPHTHALENE	2E+01	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-02	mg/kg-day	9E-04		
				PHENANTHRENE	3E+01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-02	mg/kg-day	9E-04		
				1,2,3-TRICHLOROBENZENE	3E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	3E+00	mg/kg	1E-06	mg/kg-day	4E-03	1/(mg/kg-day)	4E-09	3E-06	mg/kg-day	1E-02	mg/kg-day	3E-04		
				1,2-DICHLOROBENZENE	6E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	9E-02	mg/kg-day	7E-05		
				1,4-DICHLOROBENZENE	2E+01	mg/kg	8E-06	mg/kg-day	5E-03	1/(mg/kg-day)	4E-08	2E-05	mg/kg-day	7E-02	mg/kg-day	3E-04		
				BENZENE	4E-01	mg/kg	1E-07	mg/kg-day	6E-02	1/(mg/kg-day)	8E-09	4E-07	mg/kg-day	4E-03	mg/kg-day	1E-04		
				P-ISOPROPYLTOLUENE	3E-03	mg/kg	9E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day			
				DODECANE	8E+02	mg/kg	3E-04	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day		mg/kg-day			
				Exp. Route Total											1E-04			8E-01
				Exp. Point Total											2E-04			1E+00
Exp. Medium Total											2E-04			1E+00				
Medium Total											2E-04			1E+00				
Water	Potable Water (All GW)	Exposure Unit 8	Ingestion	ALUMINUM	2E+04	ug/l	2E-01	mg/kg-day		1/(mg/kg-day)		5E-01	mg/kg-day	1E+00	mg/kg-day	5E-01		
				ANTIMONY	2E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	4E-04	mg/kg-day	1E-01		
				ARSENIC	9E+00	ug/l	7E-05	mg/kg-day	2E+00	1/(mg/kg-day)	1E-04	2E-04	mg/kg-day	3E-04	mg/kg-day	6E-01		
				BARIIUM	1E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	2E-01	mg/kg-day	1E-01		
				BERYLLIUM	8E-01	ug/l	6E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-03	mg/kg-day	8E-03		
				CADMIUM	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	1E-03	mg/kg-day	4E-02		
				CHROMIUM	7E+01	ug/l	5E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	3E-03	mg/kg-day	5E-01		
				COBALT	1E+01	ug/l	8E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day			
				COPPER	9E+01	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	4E-02	mg/kg-day	5E-02		
				CYANIDE	3E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	2E-02	mg/kg-day	3E-02		
				IRON	4E+04	ug/l	3E-01	mg/kg-day		1/(mg/kg-day)		8E-01	mg/kg-day	7E-01	mg/kg-day	1E+00		
				LEAD	6E+01	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day		mg/kg-day			
				MANGANESE	2E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	1E-01	mg/kg-day	3E-01		
				MERCURY	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-04	mg/kg-day	1E-01		
				NICKEL	5E+01	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-02	mg/kg-day	5E-02		
				SELENIUM	4E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	5E-03	mg/kg-day	1E-02		
				SILVER	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	5E-03	mg/kg-day	8E-03		
				THALLIUM	7E+00	ug/l	5E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	8E-05	mg/kg-day	2E+00		
				VANADIUM	4E+01	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		9E-04	mg/kg-day	9E-03	mg/kg-day	1E-01		
				ZINC	1E+02	ug/l	7E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-01	mg/kg-day	6E-03		
				HIGHLY CHLORINATED PCBs	7E-02	ug/l	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	1E-06	mg/kg-day	2E-05	mg/kg-day	7E-02		
				4,4'-DDD	9E-02	ug/l	6E-07	mg/kg-day	2E-01	1/(mg/kg-day)	1E-07	2E-06	mg/kg-day		mg/kg-day			
				4,4'-DDT	1E+00	ug/l	8E-06	mg/kg-day	3E-01	1/(mg/kg-day)	3E-06	2E-05	mg/kg-day	5E-04	mg/kg-day	4E-02		
				ALDRIN	3E-02	ug/l	2E-07	mg/kg-day	2E+01	1/(mg/kg-day)	4E-06	7E-07	mg/kg-day	3E-05	mg/kg-day	2E-02		
				ALPHA-BHC	2E-01	ug/l	1E-06	mg/kg-day	6E+00	1/(mg/kg-day)	8E-06	4E-06	mg/kg-day		mg/kg-day			
				ENDOSULFAN II	6E-02	ug/l	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	6E-03	mg/kg-day	2E-04		
				ENDOSULFAN SULFATE	2E-02	ug/l	1E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	6E-03	mg/kg-day	7E-05		
				HEPTACHLOR EPOXIDE	1E-02	ug/l	7E-08	mg/kg-day		1/(mg/kg-day)	6E-07	2E-07	mg/kg-day	1E-05	mg/kg-day	2E-02		
				1,1'-BIPHENYL	1E+01	ug/l	9E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	5E-02	mg/kg-day	5E-03		
				2,4-DICHLOROPHENOL	1E+01	ug/l	7E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-03	mg/kg-day	6E-02		
				2,4-DIMETHYLPHENOL	4E+03	ug/l	3E-02	mg/kg-day		1/(mg/kg-day)		8E-02	mg/kg-day	2E-02	mg/kg-day	4E+00		
				2-METHYLNAPHTHALENE	6E+02	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	4E-03	mg/kg-day	3E+00		
				2-METHYLPHENOL	1E+03	ug/l	7E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	5E-02	mg/kg-day	4E-01		
				2-NITROPHENOL	6E+00	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day			
				3&4-METHYLPHENOL	4E+03	ug/l	3E-02	mg/kg-day		1/(mg/kg-day)		8E-02	mg/kg-day	5E-02	mg/kg-day	2E+00		
				4-CHLORO-3-METHYLPHENOL	1E+00	ug/l	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day			
				4-METHYLPHENOL	8E+03	ug/l	6E-02	mg/kg-day		1/(mg/kg-day)		2E-01	mg/kg-day	5E-02	mg/kg-day	3E+00		
				4-NITROPHENOL	1E+01	ug/l	7E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day			
				ACENAPHTHENE	1E+02	ug/l	7E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	6E-02	mg/kg-day	3E-02		
				ACENAPHTHYLENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-02	mg/kg-day	1E-01		

TABLE 7.9 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations							
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient			
							Value	Units	Value	Units		Value	Units	Value	Units				
Water	Potable Water (All GW)	Exposure Unit 8	Ingestion	ANTHRACENE	1E+02	ug/l	8E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-01	mg/kg-day	7E-03			
				ATRAZINE	5E+01	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	4E-02	mg/kg-day	3E-02			
				BENZ(A)ANTHRACENE	5E+01	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)	3E-04	1E-03	mg/kg-day		mg/kg-day				
				BENZO(A)PYRENE	2E+01	ug/l	1E-04	mg/kg-day	7E+00	1/(mg/kg-day)	1E-03	4E-04	mg/kg-day		mg/kg-day				
				BENZO(B)FLUORANTHENE	2E+01	ug/l	1E-04	mg/kg-day	7E-01	1/(mg/kg-day)	1E-04	4E-04	mg/kg-day		mg/kg-day				
				BENZO(G,H,I)PERYLENE	5E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	3E-03			
				BENZO(K)FLUORANTHENE	2E+01	ug/l	1E-04	mg/kg-day	7E-02	1/(mg/kg-day)	9E-06	4E-04	mg/kg-day		mg/kg-day				
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	7E-05	mg/kg-day	1E-02	1/(mg/kg-day)	1E-06	2E-04	mg/kg-day	2E-02	mg/kg-day	1E-02			
				CARBAZOLE	1E+02	ug/l	7E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day		mg/kg-day				
				CHRYSENE	4E+01	ug/l	2E-04	mg/kg-day	7E-03	1/(mg/kg-day)	2E-06	7E-04	mg/kg-day		mg/kg-day				
				DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	2E-05	mg/kg-day	7E+00	1/(mg/kg-day)	1E-04	6E-05	mg/kg-day		mg/kg-day				
				DIBENZOFURAN	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	1E-03	mg/kg-day	4E+00			
				FLUORANTHENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	4E-02	mg/kg-day	8E-02			
				FLUORENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	4E-02	mg/kg-day	9E-02			
				HEXACHLOROBUTADIENE	1E+00	ug/l	7E-06	mg/kg-day		1/(mg/kg-day)	5E-07	2E-05	mg/kg-day		mg/kg-day				
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	6E-05	mg/kg-day	7E-01	1/(mg/kg-day)	4E-05	2E-04	mg/kg-day		mg/kg-day				
				NAPHTHALENE	4E+03	ug/l	3E-02	mg/kg-day		1/(mg/kg-day)		8E-02	mg/kg-day	2E-02	mg/kg-day	4E+00			
				NITROBENZENE	3E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	5E-04	mg/kg-day	1E-01			
				PHENANTHRENE	4E+02	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		8E-03	mg/kg-day	3E-02	mg/kg-day	3E-01			
				PHENOL	2E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	3E-01	mg/kg-day	1E-01			
				PYRENE	1E+02	ug/l	7E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-02	mg/kg-day	7E-02			
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l	8E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day				
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	9E-05	mg/kg-day		1/(mg/kg-day)	3E-07	3E-04	mg/kg-day	1E-02	mg/kg-day	3E-02			
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day		mg/kg-day				
				1,2-DICHLOROBENZENE	5E+02	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	9E-02	mg/kg-day	1E-01			
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day		mg/kg-day				
				1,3-DICHLOROBENZENE	5E+00	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day				
				1,4-DICHLOROBENZENE	5E+02	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)	2E-05	9E-03	mg/kg-day	7E-02	mg/kg-day	1E-01			
				2-HEXANONE	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	2E-01	mg/kg-day	2E-04			
				ACETONE	8E+01	ug/l	5E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	9E-01	mg/kg-day	2E-03			
				BENZENE	6E+03	ug/l	4E-02	mg/kg-day		1/(mg/kg-day)	2E-03	1E-01	mg/kg-day	4E-03	mg/kg-day	3E+01			
				BROMODICHLOROMETHANE	3E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)	1E-06	6E-05	mg/kg-day	2E-02	mg/kg-day	3E-03			
				CARBON DISULFIDE	1E+01	ug/l	9E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	2E-03			
				CHLOROBENZENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	2E-02	mg/kg-day	2E-01			
				CHLOROETHANE	5E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day		mg/kg-day				
				ETHYLBENZENE	1E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	1E-01	mg/kg-day	3E-02			
				ISOPROPYLBENZENE	4E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	1E-01	mg/kg-day	8E-04			
				METHYLENE CHLORIDE	7E-01	ug/l	5E-06	mg/kg-day		1/(mg/kg-day)	4E-08	1E-05	mg/kg-day	6E-02	mg/kg-day	2E-04			
				P-ISOPROPYLTOLUENE	3E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day		mg/kg-day				
				SEC-BUTYLBENZENE	1E+01	ug/l	8E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day				
				STYRENE	8E+02	ug/l	6E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	2E-01	mg/kg-day	8E-02			
				TETRACHLOROETHENE	3E-01	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)	1E-06	6E-06	mg/kg-day	1E-02	mg/kg-day	6E-04			
				TOLUENE	1E+03	ug/l	9E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	8E-02	mg/kg-day	3E-01			
				VINYL CHLORIDE	1E+00	ug/l	8E-06	mg/kg-day		1/(mg/kg-day)	6E-06	2E-05	mg/kg-day	3E-03	mg/kg-day	7E-03			
				XYLENES, TOTAL	1E+03	ug/l	7E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	2E-01	mg/kg-day	9E-02			
								Exp. Route Total										6E+01	
								Exp. Point Total										6E+01	
								Exp. Medium Total										6E+01	
				Medium Total											4E-03			6E+01	
											Total of Receptor Risks Across All Media					4E-03	Total of Receptor Hazards Across All Media		

TABLE 7.9a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	6E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	1E-10	mg/kg-day	2E+01	1/(mg/kg-day)	2E-09	3E-10	mg/kg-day	1E-03	mg/kg-day	2E-04
				CADMIUM	8E-09	mg/m3	3E-10	mg/kg-day	6E+00	1/(mg/kg-day)	2E-09	9E-10	mg/kg-day	1E-05	mg/kg-day	2E-05
				CHROMIUM	5E-08	mg/m3	2E-09	mg/kg-day	4E+01	1/(mg/kg-day)	1E-07	7E-09	mg/kg-day	3E-05	mg/kg-day	2E-04
				COPPER	5E-08	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		6E-09	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	6E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-05	mg/kg-day	1E-03
				MERCURY	8E-10	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day	9E-05	mg/kg-day	1E-06
				VANADIUM	6E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	2E-11	mg/kg-day	2E+00	1/(mg/kg-day)	4E-11	5E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		9E-11	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	2E-08	mg/kg-day	3E-02	1/(mg/kg-day)	6E-10	6E-08	mg/kg-day	9E-03	mg/kg-day	8E-06
			Exp. Route Total								1E-07					2E-03
		Exp. Point Total									1E-07					2E-03
	Exp. Medium Total										1E-07					2E-03
Medium Total											1E-07					2E-03
Soil	Surface soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	3E-12	mg/kg-day	2E+05	1/(mg/kg-day)	5E-07	9E-12	mg/kg-day	1E-09	mg/kg-day	9E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	7E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	2E-06	mg/kg-day	3E-04	mg/kg-day	6E-03
				CADMIUM	2E+01	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-05	mg/kg-day	7E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	9E-07	1E-06	mg/kg-day	2E-05	mg/kg-day	6E-02
				ACENAPHTHYLENE	2E+00	mg/kg	9E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	9E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	4E-06	mg/kg-day	7E-01	1/(mg/kg-day)	3E-06	1E-05	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	3E-06	mg/kg-day	7E+00	1/(mg/kg-day)	2E-05	8E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	4E-06	mg/kg-day	7E-01	1/(mg/kg-day)	3E-06	1E-05	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	1E-06	mg/kg-day	7E-02	1/(mg/kg-day)	1E-07	4E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	4E-06	mg/kg-day	7E-03	1/(mg/kg-day)	3E-08	1E-05	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	3E-07	mg/kg-day	7E+00	1/(mg/kg-day)	2E-06	7E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	1E-03	mg/kg-day	2E-03
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	8E-07	mg/kg-day	7E-01	1/(mg/kg-day)	6E-07	2E-06	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	6E-04
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								3E-05					9E-02

TABLE 7.9a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Soil	Surface soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	1E-11	mg/kg-day	2E+05	1/(mg/kg-day)	2E-06	3E-11	mg/kg-day	1E-09	mg/kg-day	3E-02	
				ALUMINUM	5E+03	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	1E+00	mg/kg-day	5E-03	
				ARSENIC	6E+00	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	3E-06	6E-06	mg/kg-day	3E-04	mg/kg-day	2E-02	
				CADMIUM	2E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	1E-03	mg/kg-day	2E-02	
				CHROMIUM	1E+02	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-03	mg/kg-day	4E-02	
				COPPER	1E+02	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	4E-02	mg/kg-day	3E-03	
				IRON	1E+04	mg/kg	5E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	7E-01	mg/kg-day	2E-02	
				MANGANESE	3E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-01	mg/kg-day	2E-03	
				MERCURY	2E+00	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-04	mg/kg-day	6E-03	
				VANADIUM	1E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	2E-03	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	7E-07	9E-07	mg/kg-day	2E-05	mg/kg-day	5E-02	
				ACENAPHTHYLENE	2E+00	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	7E-05	
				BENZ(A)ANTHRACENE	9E+00	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	9E-06	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	7E+00	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	2E-05	6E-06	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	9E-06	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	8E-05	
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	1E-06	mg/kg-day	7E-02	1/(mg/kg-day)	8E-08	3E-06	mg/kg-day		mg/kg-day		
				CHRYSENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	9E-06	mg/kg-day		mg/kg-day		
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	6E-07	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	2E+00	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	1E-03	mg/kg-day	2E-03	
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	6E-07	mg/kg-day	7E-01	1/(mg/kg-day)	5E-07	2E-06	mg/kg-day		mg/kg-day		
				PHENANTHRENE	1E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	5E-04	
				BENZENE	1E-03	mg/kg	3E-10	mg/kg-day	6E-02	1/(mg/kg-day)	2E-11	1E-09	mg/kg-day	4E-03	mg/kg-day	2E-07	
				Exp. Route Total											3E-05		
		Exp. Point Total											6E-05				3E-01
	Exp. Medium Total											6E-05				3E-01	
Medium Total											6E-05				3E-01		
Total of Receptor Risks Across All Media											6E-05		Total of Receptor Hazards Across All Media				3E-01

TABLE 7.10 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	Ingestion	2,3,7,8-TCDD Equivalent	2E-05	mg/kg	9E-10	mg/kg-day	2E+05	1/(mg/kg-day)	1E-04	1E-08	mg/kg-day	1E-09	mg/kg-day	1E+01
				ANTIMONY	1E+00	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	4E-04	mg/kg-day	1E+00
				ARSENIC	8E-02	mg/kg	4E-06	mg/kg-day	2E+00	1/(mg/kg-day)	6E-06	4E-05	mg/kg-day	3E-04	mg/kg-day	1E-01
				CHROMIUM	6E-01	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	3E-03	mg/kg-day	1E-01
				CYANIDE	6E+00	mg/kg	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	2E-02	mg/kg-day	2E-01
				MANGANESE	3E+00	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E-01	mg/kg-day	1E-02
				MERCURY (AS METHYLMERCURY)	1E+00	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	1E-04	mg/kg-day	6E+00
				SELENIUM	2E+00	mg/kg	7E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	5E-03	mg/kg-day	2E-01
				VANADIUM	6E-01	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	9E-03	mg/kg-day	4E-02
				ZINC	4E+01	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	3E-01	mg/kg-day	8E-02
				HIGHLY CHLORINATED PCBs	6E-01	mg/kg	3E-05	mg/kg-day	2E+00	1/(mg/kg-day)	6E-05	3E-04	mg/kg-day	2E-05	mg/kg-day	2E+01
				LESS CHLORINATED PCBs	5E-01	mg/kg	2E-05	mg/kg-day	2E+00	1/(mg/kg-day)	5E-05	3E-04	mg/kg-day	7E-05	mg/kg-day	4E+00
				4,4-DDD	1E-02	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day		mg/kg-day	
				4,4'-DDT	1E-02	mg/kg	5E-07	mg/kg-day	3E-01	1/(mg/kg-day)	2E-07	5E-06	mg/kg-day	5E-04	mg/kg-day	1E-02
				ALDRIN	3E-03	mg/kg	1E-07	mg/kg-day	2E+01	1/(mg/kg-day)	2E-06	1E-06	mg/kg-day	3E-05	mg/kg-day	5E-02
				DELTA-BHC	3E-03	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				DIELDRIN	4E-03	mg/kg	2E-07	mg/kg-day	2E+01	1/(mg/kg-day)	3E-06	2E-06	mg/kg-day	5E-05	mg/kg-day	4E-02
				HEPTACHLOR EPOXIDE	4E-03	mg/kg	2E-07	mg/kg-day	9E+00	1/(mg/kg-day)	2E-06	2E-06	mg/kg-day	1E-05	mg/kg-day	2E-01
				BIS(2-ETHYLHEXYL)PHTHALATE	2E+00	mg/kg	1E-04	mg/kg-day	1E-02	1/(mg/kg-day)	2E-06	1E-03	mg/kg-day	2E-02	mg/kg-day	6E-02
				HEXACHLOROBENZENE	1E-02	mg/kg	6E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	7E-06	mg/kg-day	8E-04	mg/kg-day	9E-03
				Exp. Route Total							3E-04					4E+01
		Exp. Point Total									3E-04					4E+01
	Exp. Medium Total										3E-04					4E+01
Medium Total											3E-04					4E+01
Sediment	Surface Sediment	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	1E-04	mg/kg	2E-11	mg/kg-day	2E+05	1/(mg/kg-day)	3E-06	2E-10	mg/kg-day	1E-09	mg/kg-day	2E-01
				ARSENIC	1E+01	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	3E-06	2E-05	mg/kg-day	3E-04	mg/kg-day	7E-02
				CADMIUM	5E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-05	mg/kg-day	1E-02
				CHROMIUM	7E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				THALLIUM	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)	1E-06	7E-06	mg/kg-day	2E-05	mg/kg-day	3E-01
				DIELDRIN	2E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				ENDRIN KETONE	5E-02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-04	mg/kg-day	
				HEPTACHLOR EPOXIDE	1E-02	mg/kg		mg/kg-day	9E+00	1/(mg/kg-day)			mg/kg-day	1E-05	mg/kg-day	
				2-METHYLNAPHTHALENE	4E+01	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	4E-03	mg/kg-day	8E-02
				ACENAPHTHYLENE	6E+00	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	3E-02	mg/kg-day	2E-03
				BENZ(A)ANTHRACENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	1E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-03	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	8E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	7E+01	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	3E-02	mg/kg-day	2E-02
				BENZO(K)FLUORANTHENE	6E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+01	mg/kg	3E-05	mg/kg-day	1E-02	1/(mg/kg-day)	5E-07	4E-04	mg/kg-day	2E-02	mg/kg-day	2E-02
				CARBAZOLE	2E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day	
				CHRYSENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-04	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	3E+01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-03	mg/kg-day	2E-01
				FLUORANTHENE	1E+02	mg/kg	9E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	4E-02	mg/kg-day	3E-02
				HEXACHLOROBENZENE	1E-01	mg/kg	7E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	8E-07	mg/kg-day	8E-04	mg/kg-day	1E-03
				INDENO(1,2,3-CD)PYRENE	5E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	7E+01	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	2E-02	mg/kg-day	3E-02
				PHENANTHRENE	2E+02	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-02	mg/kg-day	7E-02
				PYRENE	4E+02	mg/kg	3E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	3E-02	mg/kg-day	1E-01
				1,2,4-TRICHLOROBENZENE	8E-01	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	5E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	

TABLE 7.10 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Sediment	Surface Sediment	Exposure Unit 6	Dermal	BENZENE	8E+00	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day			
				CHLOROBENZENE	5E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-02	mg/kg-day			
				METHYLENE CHLORIDE	7E-01	mg/kg		mg/kg-day	8E-03	1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day			
				TOLUENE	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-02	mg/kg-day			
				XYLENES, TOTAL	7E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day			
			Exp. Route Total								6E-03				1E+00			
			Ingestion	2,3,7,8-TCDD Equivalent	1E-04	mg/kg	1E-11	mg/kg-day	2E+05	1/(mg/kg-day)	2E-06	2E-10	mg/kg-day	1E-09	mg/kg-day	2E-01		
				ARSENIC	1E+01	mg/kg	1E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	2E-05	mg/kg-day	3E-04	mg/kg-day	5E-02		
				CADMIUM	5E+00	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day	3E-05	mg/kg-day	3E-01		
				CHROMIUM	7E+01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	8E-05	mg/kg-day	1E+00		
				IRON	1E+04	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	7E-01	mg/kg-day	3E-02		
				LEAD	1E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day			
				MANGANESE	2E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	6E-03	mg/kg-day	7E-02		
				MERCURY	8E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	2E-05	mg/kg-day	6E-01		
				THALLIUM	8E-01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	8E-05	mg/kg-day	1E-02		
				VANADIUM	1E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-04	mg/kg-day	1E-01		
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	1E-06	mg/kg-day	2E-05	mg/kg-day	6E-02		
				DIELDRIN	2E-02	mg/kg	3E-09	mg/kg-day	2E+01	1/(mg/kg-day)	4E-08	3E-08	mg/kg-day	5E-05	mg/kg-day	6E-04		
				ENDRIN KETONE	5E-02	mg/kg	7E-09	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day	3E-04	mg/kg-day	3E-04		
				HEPTACHLOR EPOXIDE	1E-02	mg/kg	1E-09	mg/kg-day	9E+00	1/(mg/kg-day)	1E-08	2E-08	mg/kg-day	1E-05	mg/kg-day	1E-03		
				2-METHYLNAPHTHALENE	4E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	4E-03	mg/kg-day	1E-02		
				ACENAPHTHYLENE	6E+00	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-02	mg/kg-day	3E-04		
				BENZ(A)ANTHRACENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-04	(a)	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	1E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-04	(a)	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	7E+01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	3E-03		
				BENZO(K)FLUORANTHENE	6E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-06	(a)	mg/kg-day		mg/kg-day			
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+01	mg/kg	8E-06	mg/kg-day	1E-02	1/(mg/kg-day)	1E-07	9E-05	mg/kg-day	2E-02	mg/kg-day	5E-03		
				CARBAZOLE	2E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day		mg/kg-day			
				CHRYSENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	8E-05	(a)	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	3E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	1E-03	mg/kg-day	4E-02		
				FLUORANTHENE	1E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	5E-03		
				HEXACHLOROBENZENE	1E-01	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	2E-07	mg/kg-day	8E-04	mg/kg-day	2E-04		
				INDENO(1,2,3-CD)PYRENE	5E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-05	(a)	mg/kg-day		mg/kg-day			
				NAPHTHALENE	7E+01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	2E-02	mg/kg-day	5E-03		
				PHENANTHRENE	2E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	3E-02	mg/kg-day	1E-02		
				PYRENE	4E+02	mg/kg	6E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day	3E-02	mg/kg-day	2E-02		
				1,2,4-TRICHLOROBENZENE	8E-01	mg/kg	1E-07	mg/kg-day	4E-03	1/(mg/kg-day)	4E-10	1E-06	mg/kg-day	1E-02	mg/kg-day	1E-04		
				1,4-DICHLOROBENZENE	5E+01	mg/kg	7E-06	mg/kg-day	5E-03	1/(mg/kg-day)	4E-08	8E-05	mg/kg-day	7E-02	mg/kg-day	1E-03		
				BENZENE	8E+00	mg/kg	1E-06	mg/kg-day	6E-02	1/(mg/kg-day)	6E-08	1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03		
				CHLOROBENZENE	5E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	2E-02	mg/kg-day	4E-03		
				METHYLENE CHLORIDE	7E-01	mg/kg	1E-07	mg/kg-day	8E-03	1/(mg/kg-day)	7E-10	1E-06	mg/kg-day	6E-02	mg/kg-day	2E-05		
				TOLUENE	2E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	8E-02	mg/kg-day	4E-04		
				XYLENES, TOTAL	7E+01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	2E-01	mg/kg-day	5E-04		
				Exp. Route Total								1E-03				3E+00		
				Exp. Point Total								7E-03				4E+00		
				Exp. Medium Total								7E-03				4E+00		
				Medium Total									7E-03				4E+00	
				Soil	Surface Soil	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	9E-11	mg/kg-day	2E+05	1/(mg/kg-day)	1E-05	1E-09	mg/kg-day	1E+00
ALUMINUM	7E+03	mg/kg							mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day		
ARSENIC	8E+00	mg/kg	1E-06					mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	2E-05	mg/kg-day	3E-04	mg/kg-day	5E-02		
BARIIUM	4E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day			
CADMIUM	4E+01	mg/kg	2E-07					mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-05	mg/kg-day	9E-02		
CHROMIUM	1E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
COPPER	2E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day			
IRON	1E+04	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day			
LEAD	7E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			

TABLE 7.10 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Dermal	MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				SILVER	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				THALLIUM	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	1E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	1E-05	mg/kg-day	2E-05	mg/kg-day	7E-01
				LESS CHLORINATED PCBs	7E-01	mg/kg	6E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	7E-06	mg/kg-day	7E-05	mg/kg-day	9E-02
				DIELDRIN	1E-01	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				2-METHYLNAPHTHALENE	1E+01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	4E-03	mg/kg-day	3E-02
				ACENAPHTHYLENE	4E+00	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				BENZ(A)ANTHRACENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-07	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	1E-03	mg/kg-day	3E-02
				HEXACHLOROBENZENE	1E+00	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	6E-06	mg/kg-day	8E-04	mg/kg-day	8E-03
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	2E-02	mg/kg-day	1E-02
				PHENANTHRENE	2E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-02	mg/kg-day	5E-03
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBENZENE	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	3E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				BENZENE	5E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				Exp. Route Total							5E-05					2E+00
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	7E-11	mg/kg-day	2E+05	1/(mg/kg-day)	1E-05	8E-10	mg/kg-day	1E-09	mg/kg-day	8E-01
				ALUMINUM	7E+03	mg/kg	9E-04	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	1E+00	mg/kg-day	1E-02
				ARSENIC	8E+00	mg/kg	1E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	1E-05	mg/kg-day	3E-04	mg/kg-day	4E-02
				BARIUM	4E+02	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	2E-01	mg/kg-day	3E-03
				CADMIUM	4E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	1E-03	mg/kg-day	5E-02
				CHROMIUM	1E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-03	mg/kg-day	6E-02
				COPPER	2E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	4E-02	mg/kg-day	9E-03
				IRON	1E+04	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	7E-01	mg/kg-day	3E-02
				LEAD	7E+02	mg/kg	9E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	1E-01	mg/kg-day	3E-03
				MERCURY	1E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-04	mg/kg-day	6E-02
				SILVER	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	5E-03	mg/kg-day	5E-03
				THALLIUM	8E-01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	8E-05	mg/kg-day	2E-02
				VANADIUM	2E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	9E-03	mg/kg-day	4E-03
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	4E-07	2E-06	mg/kg-day	2E-05	mg/kg-day	1E-01
				LESS CHLORINATED PCBs	7E-01	mg/kg	9E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	1E-06	mg/kg-day	7E-05	mg/kg-day	2E-02
				DIELDRIN	1E-01	mg/kg	1E-08	mg/kg-day	2E+01	1/(mg/kg-day)	2E-07	2E-07	mg/kg-day	5E-05	mg/kg-day	3E-03
				2-METHYLNAPHTHALENE	1E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	4E-03	mg/kg-day	5E-03
				ACENAPHTHYLENE	4E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZ(A)ANTHRACENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-02	mg/kg-day	3E-04
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-07	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	1E-03	mg/kg-day	6E-03

TABLE 7.10 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Water	Surface Water	Exposure Unit 6	Dermal	ANTIMONY	2E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)	2E-08	2E-07	mg/kg-day	6E-05	mg/kg-day	3E-03
				ARSENIC	2E+00	ug/l	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)		2E-07	mg/kg-day	3E-04	mg/kg-day	5E-04
				CHROMIUM	5E+00	ug/l	8E-08	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	8E-05	mg/kg-day	1E-02
				IRON	5E+03	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	7E-01	mg/kg-day	6E-04
				LEAD	8E+00	ug/l	6E-09	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day		mg/kg-day	
				MERCURY	1E-01	ug/l	8E-10	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	2E-05	mg/kg-day	5E-04
				THALLIUM	4E+00	ug/l	3E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	8E-05	mg/kg-day	4E-03
				2,4-DIMETHYLPHENOL	1E+02	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	2E-02	mg/kg-day	7E-03
				2-METHYLNAPHTHALENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				3&4-METHYLPHENOL	2E+02	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	5E-02	mg/kg-day	3E-03
				ACENAPHTHENE	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day	
				ACENAPHTHYLENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				BENZ(A)ANTHRACENE	4E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-03	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	3E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	8E+00	ug/l	6E-06	mg/kg-day	1E-02	1/(mg/kg-day)	9E-08	8E-05	mg/kg-day	2E-02	mg/kg-day	4E-03
				CARBAZOLE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)		mg/kg-day		mg/kg-day		
				CHRYSENE	4E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-07	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)		mg/kg-day	1E-03	mg/kg-day		
				FLUORENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)		mg/kg-day	4E-02	mg/kg-day		
				NAPHTHALENE	2E+03	ug/l	7E-04	mg/kg-day		1/(mg/kg-day)		8E-03	mg/kg-day	2E-02	mg/kg-day	4E-01
				PHENANTHRENE	3E+01	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	3E-02	mg/kg-day	2E-02
				PYRENE	8E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	8E+00	ug/l	3E-06	mg/kg-day	5E-03	1/(mg/kg-day)	2E-08	4E-05	mg/kg-day	7E-02	mg/kg-day	5E-04
				BENZENE	7E+01	ug/l	9E-06	mg/kg-day	6E-02	1/(mg/kg-day)	5E-07	1E-04	mg/kg-day	4E-03	mg/kg-day	3E-02
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				TOLUENE	4E+02	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	8E-02	mg/kg-day	1E-02
				XYLENES, TOTAL	5E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
						Exp. Route Total							1E-03			
					Exp. Point Total							1E-03				5E-01
				Exp. Medium Total							1E-03				5E-01	
Medium Total										1E-03				5E-01		
Total of Receptor Risks Across All Media											9E-03	Total of Receptor Hazards Across All Media				5E+01

Notes:
(a) See Table 7.10 RME Supplement A for the intake and toxicity values for COPCs with an MMOA

TABLE 7.10.RME Supplement A
 CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
 REASONABLE MAXIMUM EXPOSURE
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Recreator
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	EU-6	Ingestion	Benz(a)anthracene	7.5E+00	mg/kg	4.9E-07	6.2E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	5.E-06
				Benzo(a)pyrene	9.0E+00	mg/kg	5.9E-07	7.4E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	6.E-05
				Benzo(b)fluoranthene	6.6E+00	mg/kg	4.3E-07	5.4E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	4.E-06
				Benzo(k)fluoranthene	5.7E+00	mg/kg	3.7E-07	4.6E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	4.E-07
				Chrysene	8.0E+00	mg/kg	5.2E-07	6.6E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	5.E-08
				Dibenz(a,h)anthracene	1.6E+00	mg/kg	1.1E-07	1.3E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-05
				Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	3.0E-07	3.7E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-06
		Dermal	Benz(a)anthracene	7.5E+00	mg/kg	1.7E-07	2.3E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-06	
			Benzo(a)pyrene	9.0E+00	mg/kg	2.0E-07	2.8E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-05	
			Benzo(b)fluoranthene	6.6E+00	mg/kg	1.5E-07	2.0E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-06	
			Benzo(k)fluoranthene	5.7E+00	mg/kg	1.3E-07	1.7E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	1.E-07	
			Chrysene	8.0E+00	mg/kg	1.8E-07	2.5E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	2.E-08	
			Dibenz(a,h)anthracene	1.6E+00	mg/kg	3.6E-08	5.0E-08	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	4.E-06	
			Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	1.0E-07	1.4E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-06	
	Fugitive Dust	EU-6	Inhalation	Benz(a)anthracene	1.9E-09	mg/m ³	5.7E-13	1.0E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	2.3E-09	mg/m ³	6.9E-13	1.2E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	1.7E-09	mg/m ³	5.0E-13	8.9E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	1.4E-09	mg/m ³	4.3E-13	7.6E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.0E-09	mg/m ³	6.1E-13	1.1E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	4.1E-10	mg/m ³	1.3E-13	2.2E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	1.2E-09	mg/m ³	3.5E-13	6.2E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	Na
Sediment	Surface Sediment	EU-6	Ingestion	Benz(a)anthracene	1.5E+02	mg/kg	9.9E-06	1.2E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-04
				Benzo(a)pyrene	1.1E+02	mg/kg	6.9E-06	8.7E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	7.E-04
				Benzo(b)fluoranthene	2.4E+02	mg/kg	1.6E-05	2.0E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-04
				Benzo(k)fluoranthene	5.9E+01	mg/kg	3.8E-06	4.8E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	4.E-06
				Chrysene	2.2E+02	mg/kg	1.4E-05	1.8E-05	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	1.E-06
				Dibenz(a,h)anthracene	1.3E+01	mg/kg	8.4E-07	1.1E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	8.E-05
				Indeno(1,2,3-cd)pyrene	5.4E+01	mg/kg	3.6E-06	4.5E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	4.E-05
			Dermal	Benz(a)anthracene	1.5E+02	mg/kg	5.0E-05	7.0E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	5.E-04
				Benzo(a)pyrene	1.1E+02	mg/kg	3.5E-05	4.9E-05	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	4.E-03
				Benzo(b)fluoranthene	2.4E+02	mg/kg	7.9E-05	1.1E-04	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	8.E-04
				Benzo(k)fluoranthene	5.9E+01	mg/kg	1.9E-05	2.7E-05	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	2.E-05
				Chrysene	2.2E+02	mg/kg	7.2E-05	1.0E-04	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	7.E-06
				Dibenz(a,h)anthracene	1.3E+01	mg/kg	4.3E-06	5.9E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	4.E-04
				Indeno(1,2,3-cd)pyrene	5.4E+01	mg/kg	1.8E-05	2.5E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-04
Water	Surface Water	EU-6	Dermal	Benz(a)anthracene	4.0E+00	µg/L	1.3E-05	1.8E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-04
				Benzo(a)pyrene	2.0E+00	µg/L	1.1E-05	1.5E-05	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-03
				Benzo(b)fluoranthene	3.0E+00	µg/L	1.6E-05	2.3E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-04
				Chrysene	2.9E+00	µg/L	9.0E-06	1.3E-05	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	9.E-07

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include Carcinogens Described in the Supplemental Guidance as having a Mutagenic Mode of Action.

TABLE 7.10a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	5E-12	mg/kg-day	2E+05	1/(mg/kg-day)	8E-07	6E-11	mg/kg-day	1E-09	mg/kg-day	6E-02
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	1E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	1E-05	mg/kg-day	3E-04	mg/kg-day	4E-02
				CADMIUM	2E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-05	mg/kg-day	4E-02
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	7E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	8E-06	mg/kg-day	2E-05	mg/kg-day	4E-01
				ACENAPHTHYLENE	2E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	6E-04
				BENZ(A)ANTHRACENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	7E-04
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-07	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	9E-07	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-06	(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	4E-03
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				Exp. Route Total							3E-04					6E-01
			Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	4E-12	mg/kg-day	2E+05	1/(mg/kg-day)	6E-07	5E-11	mg/kg-day	1E-09	mg/kg-day	5E-02
				ALUMINUM	5E+03	mg/kg	7E-04	mg/kg-day		1/(mg/kg-day)		8E-03	mg/kg-day	1E+00	mg/kg-day	8E-03
				ARSENIC	6E+00	mg/kg	8E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	1E-05	mg/kg-day	3E-04	mg/kg-day	3E-02
				CADMIUM	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	1E-03	mg/kg-day	3E-02
				CHROMIUM	1E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-03	mg/kg-day	6E-02
				COPPER	1E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	5E-03
				IRON	1E+04	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	7E-01	mg/kg-day	3E-02
				MANGANESE	3E+02	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	1E-01	mg/kg-day	4E-03
				MERCURY	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-04	mg/kg-day	1E-02
				VANADIUM	1E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	9E-03	mg/kg-day	2E-03
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	1E-06	mg/kg-day	2E-05	mg/kg-day	7E-02
				ACENAPHTHYLENE	2E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZ(A)ANTHRACENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-07	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	1E-03	mg/kg-day	3E-03
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	8E-04
				BENZENE	1E-03	mg/kg	1E-10	mg/kg-day	6E-02	1/(mg/kg-day)	7E-12	1E-09	mg/kg-day	4E-03	mg/kg-day	4E-07
				Exp. Route Total							6E-05					3E-01
				Exp. Point Total							4E-04					9E-01
				Exp. Medium Total							4E-04					9E-01
				Medium Total							4E-04					9E-01

TABLE 7.10a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	7E-09	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	9E-12	mg/kg-day	2E+01	1/(mg/kg-day)	1E-10	1E-10	mg/kg-day	1E-03	mg/kg-day	6E-05
				CADMIUM	8E-09	mg/m3	2E-11	mg/kg-day	6E+00	1/(mg/kg-day)	2E-10	3E-10	mg/kg-day	1E-05	mg/kg-day	7E-06
				CHROMIUM	5E-08	mg/m3	2E-10	mg/kg-day	4E+01	1/(mg/kg-day)	7E-09	2E-09	mg/kg-day	3E-05	mg/kg-day	7E-05
				COPPER	5E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	5E-10	mg/kg-day		1/(mg/kg-day)		5E-09	mg/kg-day	1E-05	mg/kg-day	4E-04
				MERCURY	8E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day	9E-05	mg/kg-day	3E-07
				VANADIUM	6E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	1E-12	mg/kg-day	2E+00	1/(mg/kg-day)	3E-12	1E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	2E-09	mg/kg-day	3E-02	1/(mg/kg-day)	4E-11	2E-08	mg/kg-day	9E-03	mg/kg-day	2E-06
			Exp. Route Total								7E-09					5E-04
		Exp. Point Total									7E-09					5E-04
	Exp. Medium Total										7E-09					5E-04
Medium Total											7E-09					5E-04
Total of Receptor Risks Across All Media											4E-04	Total of Receptor Hazards Across All Media				9E-01

Notes:
(a) See Table 7.10a RME Supplement A for the intake and toxicity values for COPCs with an MMOA

TABLE 7.10a.RME Supplement A
CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Recreator
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	EU-9	Ingestion	Benz(a)anthracene	9.3E+00	mg/kg	6.1E-07	7.6E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	6.E-06
				Benzo(a)pyrene	6.6E+00	mg/kg	4.3E-07	5.4E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	4.E-05
				Benzo(b)fluoranthene	9.6E+00	mg/kg	6.2E-07	7.8E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	6.E-06
				Benzo(k)fluoranthene	3.3E+00	mg/kg	2.1E-07	2.7E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	2.E-07
				Chrysene	9.5E+00	mg/kg	6.2E-07	7.8E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	6.E-08
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	3.8E-08	4.8E-08	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	4.E-06
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	1.2E-07	1.5E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-06
			Dermal	Benz(a)anthracene	9.3E+00	mg/kg	3.1E-06	4.3E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
				Benzo(a)pyrene	6.6E+00	mg/kg	2.2E-06	3.1E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-04
				Benzo(b)fluoranthene	9.6E+00	mg/kg	3.2E-06	4.4E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
				Benzo(k)fluoranthene	3.3E+00	mg/kg	1.1E-06	1.5E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	1.E-06
				Chrysene	9.5E+00	mg/kg	3.1E-06	4.4E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	3.E-07
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	1.9E-07	2.7E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-05
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	6.0E-07	8.3E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	6.E-06
	Fugitive Dust	EU-9	Inhalation	Benz(a)anthracene	2.7E-08	mg/m³	8.1E-12	1.4E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	1.9E-08	mg/m³	5.8E-12	1.0E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	2.8E-08	mg/m³	8.3E-12	1.5E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	9.4E-09	mg/m³	2.8E-12	5.0E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.7E-08	mg/m³	8.3E-12	1.5E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	1.7E-09	mg/m³	5.1E-13	9.0E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	5.2E-09	mg/m³	1.6E-12	2.8E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include Carcinogens Described in the Supplemental Guidance as having a Mutagenic Mode of Action.

TABLE 7.11 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Water	Surface Water	Exposure Unit 6	Dermal	ANTIMONY	2E+00	ug/l	3E-08	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day	6E-05	mg/kg-day	1E-03
				ARSENIC	2E+00	ug/l	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	7E-08	mg/kg-day	3E-04	mg/kg-day	2E-04
				CHROMIUM	5E+00	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	8E-05	mg/kg-day	5E-03
				IRON	5E+03	ug/l	8E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	7E-01	mg/kg-day	3E-04
				LEAD	8E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day	
				MERCURY	1E-01	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day	2E-05	mg/kg-day	2E-04
				THALLIUM	4E+00	ug/l	6E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	8E-05	mg/kg-day	2E-03
				2,4-DIMETHYLPHENOL	1E+02	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	2E-02	mg/kg-day	3E-03
				2-METHYLNAPHTHALENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				3&4-METHYLPHENOL	2E+02	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	5E-02	mg/kg-day	1E-03
				ACENAPHTHENE	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day	
				ACENAPHTHYLENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				BENZ(A)ANTHRACENE	4E+00	ug/l	6E-05	mg/kg-day	7E-01	1/(mg/kg-day)	4E-05	1E-04	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+00	ug/l	5E-05	mg/kg-day	7E+00	1/(mg/kg-day)	4E-04	1E-04	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	3E+00	ug/l	8E-05	mg/kg-day	7E-01	1/(mg/kg-day)	6E-05	2E-04	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	8E+00	ug/l	1E-05	mg/kg-day	1E-02	1/(mg/kg-day)	2E-07	3E-05	mg/kg-day	2E-02	mg/kg-day	2E-03
				CARBAZOLE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				CHRYSENE	4E+00	ug/l	6E-05	mg/kg-day	7E-03	1/(mg/kg-day)	4E-07	1E-04	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day	
				FLUORENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				NAPHTHALENE	2E+03	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	2E-02	mg/kg-day	2E-01
				PHENANTHRENE	3E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-02	mg/kg-day	8E-03
				PYRENE	8E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	8E+00	ug/l	7E-06	mg/kg-day	5E-03	1/(mg/kg-day)	4E-08	2E-05	mg/kg-day	7E-02	mg/kg-day	2E-04
				BENZENE	7E+01	ug/l	2E-05	mg/kg-day	6E-02	1/(mg/kg-day)	1E-06	4E-05	mg/kg-day	4E-03	mg/kg-day	1E-02
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				TOLUENE	4E+02	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	8E-02	mg/kg-day	6E-03
				XYLENES, TOTAL	5E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
							Exp. Route Total								5E-04	
					Exp. Point Total								5E-04			2E-01
				Exp. Medium Total								5E-04			2E-01	
											5E-04			2E-01		
Medium Total				Total of Receptor Risks Across All Media							2E-03	Total of Receptor Hazards Across All Media				3E+01

TABLE 7.11a. RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	1E-11	mg/kg-day	2E+01	1/(mg/kg-day)	2E-10	3E-11	mg/kg-day	1E-03	mg/kg-day	2E-05
				CADMIUM	8E-09	mg/m3	3E-11	mg/kg-day	6E+00	1/(mg/kg-day)	2E-10	8E-11	mg/kg-day	1E-05	mg/kg-day	2E-06
				CHROMIUM	5E-08	mg/m3	2E-10	mg/kg-day	4E+01	1/(mg/kg-day)	1E-08	6E-10	mg/kg-day	3E-05	mg/kg-day	2E-05
				COPPER	5E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	3E-08	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	1E-05	mg/kg-day	1E-04
				MERCURY	8E-10	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		9E-12	mg/kg-day	9E-05	mg/kg-day	1E-07
				VANADIUM	6E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	2E-12	mg/kg-day	2E+00	1/(mg/kg-day)	4E-12	4E-12	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		9E-12	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	6E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		3E-12	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		8E-12	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		8E-12	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	2E-09	mg/kg-day	3E-02	1/(mg/kg-day)	6E-11	5E-09	mg/kg-day	9E-03	mg/kg-day	6E-07
				Exp. Route Total							1E-08					1E-04
				Exp. Point Total							1E-08					1E-04
				Exp. Medium Total							1E-08					1E-04
				Medium Total							1E-08					1E-04
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	1E-12	mg/kg-day	2E+05	1/(mg/kg-day)	2E-07	3E-12	mg/kg-day	1E-09	mg/kg-day	3E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	5E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				CADMIUM	2E+01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	3E-05	mg/kg-day	2E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	4E-07	mg/kg-day	2E-05	mg/kg-day	2E-02
				ACENAPHTHYLENE	2E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	3E-02	mg/kg-day	2E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	3E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	1E-06	mg/kg-day	7E+00	1/(mg/kg-day)	8E-06	2E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	3E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	3E-02	mg/kg-day	3E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	5E-07	mg/kg-day	7E-02	1/(mg/kg-day)	4E-08	1E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	1E-06	mg/kg-day	7E-03	1/(mg/kg-day)	1E-08	3E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	9E-08	mg/kg-day	7E+00	1/(mg/kg-day)	7E-07	2E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	1E-03	mg/kg-day	5E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	3E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	7E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				Exp. Route Total							1E-05					3E-02

TABLE 7.11a. RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	1E-12	mg/kg-day	2E+05	1/(mg/kg-day)	2E-07	3E-12	mg/kg-day	1E-09	mg/kg-day	3E-03
				ALUMINUM	5E+03	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	1E+00	mg/kg-day	4E-04
				ARSENIC	6E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	5E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				CADMIUM	2E+01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03
				CHROMIUM	1E+02	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-03	mg/kg-day	3E-03
				COPPER	1E+02	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-02	mg/kg-day	2E-04
				IRON	1E+04	mg/kg	5E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	7E-01	mg/kg-day	2E-03
				MANGANESE	3E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	1E-01	mg/kg-day	2E-04
				MERCURY	2E+00	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-04	mg/kg-day	5E-04
				VANADIUM	1E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	9E-03	mg/kg-day	1E-04
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	7E-08	8E-08	mg/kg-day	2E-05	mg/kg-day	4E-03
				ACENAPHTHYLENE	2E+00	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	6E-06
				BENZ(A)ANTHRACENE	9E+00	mg/kg	3E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	8E-07	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	2E-06	5E-07	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	3E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	8E-07	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	8E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	6E-06
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	1E-07	mg/kg-day	7E-02	1/(mg/kg-day)	8E-09	3E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	3E-07	mg/kg-day	7E-03	1/(mg/kg-day)	2E-09	8E-07	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	2E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	5E-08	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	1E-03	mg/kg-day	1E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	6E-08	mg/kg-day	7E-01	1/(mg/kg-day)	5E-08	1E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	4E-05
				BENZENE	1E-03	mg/kg	3E-11	mg/kg-day	6E-02	1/(mg/kg-day)	2E-12	8E-11	mg/kg-day	4E-03	mg/kg-day	2E-08
						Exp. Route Total								3E-06		
				Exp. Point Total								1E-05				4E-02
		Exp. Medium Total									1E-05				4E-02	
Medium Total										1E-05				4E-02		
Total of Receptor Risks Across All Media											1E-05	Total of Receptor Hazards Across All Media				4E-02

TABLE 7.12 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations								
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient				
							Value	Units	Value	Units		Value	Units	Value	Units					
Ground Water	Potable Water	Exposure Unit 8	Ingestion	TETRACHLOROETHENE	3E-01	ug/l	2E-06	mg/kg-day	5E-01	1/(mg/kg-day)	9E-07	2E-05	mg/kg-day	1E-02	mg/kg-day	2E-03				
				TOLUENE	1E+03	ug/l	7E-03	mg/kg-day		1/(mg/kg-day)		8E-02	mg/kg-day	8E-02	mg/kg-day	1E+00				
				VINYL CHLORIDE	1E+00	ug/l	6E-06	mg/kg-day	8E-01	1/(mg/kg-day)	5E-06	7E-05	mg/kg-day	3E-03	mg/kg-day	2E-02				
				XYLENES, TOTAL	1E+03	ug/l	5E-03	mg/kg-day		1/(mg/kg-day)		6E-02	mg/kg-day	2E-01	mg/kg-day	3E-01				
		Exp. Point Total	Exp. Route Total								3E-03					2E+02				
	Exp. Medium Total										7E-01					2E+02				
											7E-01					2E+02				
	Shower Vapor	Exposure Unit 8	Inhalation	1,2,3-TRICHLOROBENZENE	3E-01	mg/m3	6E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day		mg/kg-day					
				1,2,4-TRICHLOROBENZENE	3E-01	mg/m3	7E-04	mg/kg-day		1/(mg/kg-day)		8E-03	mg/kg-day		mg/kg-day					
				1,2,4-TRIMETHYLBENZENE	7E+00	mg/m3	2E-02	mg/kg-day		1/(mg/kg-day)		2E-01	mg/kg-day	2E-03	mg/kg-day	1E+02				
				1,2-DICHLOROBENZENE	1E+01	mg/m3	3E-02	mg/kg-day		1/(mg/kg-day)		3E-01	mg/kg-day	4E-02	mg/kg-day	8E+00				
				1,3,5-TRIMETHYLBENZENE	5E+00	mg/m3	1E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day		mg/kg-day					
				1,3-DICHLOROBENZENE	1E-01	mg/m3	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day		mg/kg-day					
				1,4-DICHLOROBENZENE	1E+01	mg/m3	2E-02	mg/kg-day	4E-02	1/(mg/kg-day)	9E-04	3E-01	mg/kg-day	2E-01	mg/kg-day	1E+00				
				2-HEXANONE	4E-02	mg/m3	1E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	6E-02	mg/kg-day	2E-02				
				ACETONE	2E+00	mg/m3	4E-03	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	9E+00	mg/kg-day	5E-03				
				BENZENE	1E+02	mg/m3	3E-01	mg/kg-day	3E-02	1/(mg/kg-day)	8E-03	3E+00	mg/kg-day	9E-03	mg/kg-day	4E+02				
				BROMODICHLOROMETHANE	7E-02	mg/m3	2E-04	mg/kg-day	1E-01	1/(mg/kg-day)	2E-05	2E-03	mg/kg-day		mg/kg-day					
				CARBON DISULFIDE	3E-01	mg/m3	6E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	2E-01	mg/kg-day	4E-02				
				CHLOROETHANE	4E+00	mg/m3	9E-03	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day		mg/kg-day					
				CHLOROFORM	1E-01	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E+00	mg/kg-day	1E-03				
				ETHYLBENZENE	3E+00	mg/m3	6E-04	mg/kg-day	8E-02	1/(mg/kg-day)	5E-05	7E-03	mg/kg-day	3E-02	mg/kg-day	2E-01				
				ISOPROPYLBENZENE	9E-02	mg/m3	7E-03	mg/kg-day		1/(mg/kg-day)		9E-02	mg/kg-day	3E-01	mg/kg-day	3E-01				
				METHYLENE CHLORIDE	2E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E-01	mg/kg-day	2E-02				
				P-ISOPROPYLTOLUENE	7E-02	mg/m3	4E-05	mg/kg-day	2E-03	1/(mg/kg-day)	6E-08	4E-04	mg/kg-day	3E-01	mg/kg-day	1E-03				
				SEC-BUTYLBENZENE	3E-01	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day		mg/kg-day					
				STYRENE	2E+01	mg/m3	6E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day		mg/kg-day					
				TETRACHLOROETHENE	2E+01	mg/m3	4E-02	mg/kg-day		1/(mg/kg-day)		5E-01	mg/kg-day	3E+00	mg/kg-day	2E-01				
				TOLUENE	6E-03	mg/m3	1E-05	mg/kg-day	2E-05	1/(mg/kg-day)	3E-10	2E-04	mg/kg-day	8E-02	mg/kg-day	2E-03				
				VINYL CHLORIDE	3E+01	mg/m3	6E-02	mg/kg-day		1/(mg/kg-day)		7E-01	mg/kg-day	1E+00	mg/kg-day	5E-01				
				XYLENES, TOTAL	2E-02	mg/m3	6E-05	mg/kg-day	3E-02	1/(mg/kg-day)	2E-06	6E-04	mg/kg-day	3E-02	mg/kg-day	2E-02				
					2E+01	mg/m3	4E-02	mg/kg-day		1/(mg/kg-day)		4E-01	mg/kg-day	3E-02	mg/kg-day	1E+01				
		Exp. Point Total	Exp. Route Total								9E-03					5E+02				
	Exp. Medium Total										9E-03					5E+02				
											9E-03					5E+02				
Medium Total											7E-01					7E+02				
							Total of Receptor Risks Across All Media					7E-01	Total of Receptor Hazards Across All Media			8E+02				

Notes:
(a) See Table 7.12 RME Supplement A for the intake and toxicity values for COPCs with an MMOA

TABLE 7.12 RME Supplement A
CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Resident
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	EU-6	Ingestion	Benz(a)anthracene	7.5E+00	mg/kg	4.1E-06	5.1E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	4.E-05
				Benzo(a)pyrene	9.0E+00	mg/kg	4.9E-06	6.2E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	5.E-04
				Benzo(b)fluoranthene	6.6E+00	mg/kg	3.6E-06	4.5E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	4.E-05
				Benzo(k)fluoranthene	5.7E+00	mg/kg	3.1E-06	3.9E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	3.E-06
				Chrysene	8.0E+00	mg/kg	4.4E-06	5.5E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	4.E-07
				Dibenz(a,h)anthracene	1.6E+00	mg/kg	8.9E-07	1.1E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	9.E-05
				Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	2.5E-06	3.1E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
			Dermal	Benz(a)anthracene	7.5E+00	mg/kg	1.4E-06	1.9E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-05
				Benzo(a)pyrene	9.0E+00	mg/kg	1.7E-06	2.3E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-04
				Benzo(b)fluoranthene	6.6E+00	mg/kg	1.2E-06	1.7E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-05
				Benzo(k)fluoranthene	5.7E+00	mg/kg	1.0E-06	1.5E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	1.E-06
				Chrysene	8.0E+00	mg/kg	1.5E-06	2.1E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	2.E-07
				Dibenz(a,h)anthracene	1.6E+00	mg/kg	3.0E-07	4.2E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	3.E-05
				Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	8.4E-07	1.2E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	9.E-06
	Fugitive Dust	EU-6	Inhalation	Benz(a)anthracene	1.9E-09	mg/m ³	2.9E-11	5.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	2.3E-09	mg/m ³	3.4E-11	6.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	1.7E-09	mg/m ³	2.5E-11	4.4E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	1.4E-09	mg/m ³	2.2E-11	3.8E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.0E-09	mg/m ³	3.1E-11	5.4E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	4.1E-10	mg/m ³	6.3E-12	1.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	1.2E-09	mg/m ³	1.7E-11	3.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
Ground Water	Potable Water	EU-8	Ingestion	Benz(a)anthracene	5.5E+01	µg/L	2.0E-05	3.3E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-04
				Benzo(a)pyrene	2.0E+01	µg/L	7.2E-06	1.2E-05	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	8.E-04
				Benzo(b)fluoranthene	2.1E+01	µg/L	7.7E-06	1.3E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	8.E-05
				Benzo(k)fluoranthene	1.8E+01	µg/L	6.5E-06	1.1E-05	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	7.E-06
				Chrysene	3.5E+01	µg/L	1.3E-05	2.1E-05	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	1.E-06
				Dibenz(a,h)anthracene	2.8E+00	µg/L	1.0E-06	1.7E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-04
				Indeno(1,2,3-cd)pyrene	8.5E+00	µg/L	3.1E-06	5.1E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
			Dermal	Benz(a)anthracene	5.5E+01	µg/L	1.6E-03	2.3E-03	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-02
				Benzo(a)pyrene	2.0E+01	µg/L	5.0E-03	7.2E-03	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	5.E-01
				Benzo(b)fluoranthene	2.1E+01	µg/L	5.4E-03	7.8E-03	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	6.E-02
				Benzo(k)fluoranthene	1.8E+01	µg/L			mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	
				Chrysene	3.5E+01	µg/L	5.1E-03	7.4E-03	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	5.E-04
				Dibenz(a,h)anthracene	2.8E+00	µg/L	1.1E-03	1.6E-03	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-01
				Indeno(1,2,3-cd)pyrene	8.5E+00	µg/L	2.1E-03	3.1E-03	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-02

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include Carcinogens Described in the Supplemental Guidance as having a Mutagenic Mode of Action.

TABLE 7.12a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		9E-09	mg/kg-day		mg/kg-day			
				ALUMINUM	2E-06	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	1E-03		
				ARSENIC	3E-09	mg/m3	1E-10	mg/kg-day	2E+01	1/(mg/kg-day)	2E-09	2E-09	mg/kg-day	1E-05	mg/kg-day	1E-04		
				CADMIUM	8E-09	mg/m3	4E-10	mg/kg-day	6E+00	1/(mg/kg-day)	3E-09	5E-09	mg/kg-day		mg/kg-day			
				CHROMIUM	5E-08	mg/m3	3E-09	mg/kg-day	4E+01	1/(mg/kg-day)	1E-07	3E-08	mg/kg-day	3E-05	mg/kg-day	1E-03		
				COPPER	5E-08	mg/m3	3E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day			
				IRON	6E-06	mg/m3	3E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day		mg/kg-day			
				MANGANESE	1E-07	mg/m3	8E-09	mg/kg-day		1/(mg/kg-day)		9E-08	mg/kg-day	1E-05	mg/kg-day	7E-03		
				MERCURY	8E-10	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day	9E-05	mg/kg-day	6E-06		
				VANADIUM	6E-09	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day			
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	2E-11	mg/kg-day	2E+00	1/(mg/kg-day)	4E-11	3E-10	mg/kg-day		mg/kg-day			
				ACENAPHTHYLENE	9E-10	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day		mg/kg-day			
				BENZ(A)ANTHRACENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	3E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day			
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				CHRYSENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	7E-10	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day			
				PHENANTHRENE	6E-09	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day			
				BENZENE	5E-07	mg/m3	3E-08	mg/kg-day	3E-02	1/(mg/kg-day)	8E-10	3E-07	mg/kg-day	9E-03	mg/kg-day	4E-05		
				Exp. Route Total										1E-07				9E-03
				Exp. Point Total										1E-07				9E-03
				Exp. Medium Total										1E-07				9E-03
Medium Total										1E-07				9E-03				
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	4E-11	mg/kg-day	2E+05	1/(mg/kg-day)	6E-06	5E-10	mg/kg-day	1E-09	mg/kg-day	5E-01		
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day			
				ARSENIC	6E+00	mg/kg	9E-06	mg/kg-day	2E+00	1/(mg/kg-day)	1E-05	1E-04	mg/kg-day	3E-04	mg/kg-day	3E-01		
				CADMIUM	2E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-05	mg/kg-day	4E-01		
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day			
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day			
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day			
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day			
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	6E-06	mg/kg-day	2E+00	1/(mg/kg-day)	1E-05	7E-05	mg/kg-day	2E-05	mg/kg-day	4E+00		
				ACENAPHTHYLENE	2E+00	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	5E-03		
				BENZ(A)ANTHRACENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-04	(a)	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-02	mg/kg-day	5E-03		
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-07	(a)	mg/kg-day		mg/kg-day			
				CHRYSENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-07	(a)	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-05	(a)	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	2E+00	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	1E-03	mg/kg-day	9E-02		
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-06	(a)	mg/kg-day		mg/kg-day			
				PHENANTHRENE	1E+01	mg/kg	9E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	3E-02	mg/kg-day	3E-02		
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day			
				Exp. Route Total										2E-04				5E+00

TABLE 7.12a.RME Supplement A
CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Resident
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	EU-9	Ingestion	Benz(a)anthracene	9.3E+00	mg/kg	5.1E-06	6.3E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	5.E-05
				Benzo(a)pyrene	6.6E+00	mg/kg	3.6E-06	4.5E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	4.E-04
				Benzo(b)fluoranthene	9.6E+00	mg/kg	5.2E-06	6.5E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	5.E-05
				Benzo(k)fluoranthene	3.3E+00	mg/kg	1.8E-06	2.2E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	2.E-06
				Chrysene	9.5E+00	mg/kg	5.2E-06	6.5E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	5.E-07
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	3.2E-07	4.0E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	3.E-05
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	9.8E-07	1.2E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-05
			Dermal	Benz(a)anthracene	9.3E+00	mg/kg	1.7E-06	2.4E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-05
				Benzo(a)pyrene	6.6E+00	mg/kg	1.2E-06	1.7E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-04
				Benzo(b)fluoranthene	9.6E+00	mg/kg	1.8E-06	2.4E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-05
				Benzo(k)fluoranthene	3.3E+00	mg/kg	6.0E-07	8.4E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	6.E-07
				Chrysene	9.5E+00	mg/kg	1.7E-06	2.4E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	2.E-07
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	1.1E-07	1.5E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-05
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	3.3E-07	4.6E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-06
	Fugitive Dust	EU-9	Inhalation	Benz(a)anthracene	2.7E-08	mg/m ³	4.1E-10	7.2E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	1.9E-08	mg/m ³	2.9E-10	5.1E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	2.8E-08	mg/m ³	4.2E-10	7.3E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	9.4E-09	mg/m ³	1.4E-10	2.5E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.7E-08	mg/m ³	4.2E-10	7.3E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	1.7E-09	mg/m ³	2.6E-11	4.5E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	5.2E-09	mg/m ³	7.9E-11	1.4E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include Carcinogens Described in the Supplemental Guidance as having a Mutagenic Mode of Action.

TABLE 7.13 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Surface Soil	Outdoor Air	Exposure Unit 6	Inhalation	2,3,7,8-TCDD Equivalent	1E-07	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day		mg/kg-day			
				ALUMINUM	2E-06	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	1E-03	mg/kg-day	2E-04		
				ARSENIC	2E-09	mg/m3	2E-10	mg/kg-day	2E+01	1/(mg/kg-day)	2E-09	4E-10	mg/kg-day	1E-05	mg/kg-day	3E-05		
				BARIUM	9E-08	mg/m3	7E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-04	mg/kg-day	1E-04		
				CADMIUM	9E-09	mg/m3	7E-10	mg/kg-day	6E+00	1/(mg/kg-day)	4E-09	2E-09	mg/kg-day		mg/kg-day			
				CHROMIUM	3E-08	mg/m3	2E-09	mg/kg-day	4E+01	1/(mg/kg-day)	9E-08	5E-09	mg/kg-day	3E-05	mg/kg-day	2E-04		
				COPPER	6E-08	mg/m3	5E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day			
				IRON	3E-06	mg/m3	2E-07	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day		mg/kg-day			
				LEAD	2E-07	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day			
				MANGANESE	8E-08	mg/m3	6E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	1E-05	mg/kg-day	9E-04		
				MERCURY	3E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day	9E-05	mg/kg-day	6E-06		
				SILVER	4E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day			
				THALLIUM	2E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day			
				VANADIUM	5E-09	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day			
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	3E-11	mg/kg-day	2E+00	1/(mg/kg-day)	6E-11	7E-11	mg/kg-day		mg/kg-day			
				LESS CHLORINATED PCBs	2E-10	mg/m3	1E-11	mg/kg-day	2E+00	1/(mg/kg-day)	3E-11	3E-11	mg/kg-day		mg/kg-day			
				DIELDRIN	3E-11	mg/m3	2E-12	mg/kg-day	2E+01	1/(mg/kg-day)	3E-11	5E-12	mg/kg-day		mg/kg-day			
				2-METHYLNAPHTHALENE	3E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day		mg/kg-day			
				ACENAPHTHYLENE	1E-09	mg/m3	7E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				BENZ(A)ANTHRACENE	2E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	2E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	2E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				CHRYSENE	2E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	4E-10	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	1E-09	mg/m3	8E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				HEXACHLOROBENZENE	2E-10	mg/m3	2E-11	mg/kg-day	2E+00	1/(mg/kg-day)	3E-11	4E-11	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	1E-09	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				NAPHTHALENE	6E-09	mg/m3	4E-10	mg/kg-day	1E-01	1/(mg/kg-day)	5E-11	1E-09	mg/kg-day	9E-04	mg/kg-day	1E-06		
				PHENANTHRENE	5E-09	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day			
				1,2,3-TRICHLOROBENZENE	1E-04	mg/m3	1E-05	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	1E-04	mg/m3	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day		mg/kg-day			
				1,2-DICHLOROBENZENE	9E-04	mg/m3	7E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	4E-03		
				1,4-DICHLOROBENZENE	4E-03	mg/m3	3E-04	mg/kg-day	4E-02	1/(mg/kg-day)	1E-05	6E-04	mg/kg-day	2E-01	mg/kg-day	3E-03		
				BENZENE	3E-04	mg/m3	2E-05	mg/kg-day	3E-02	1/(mg/kg-day)	6E-07	5E-05	mg/kg-day	9E-03	mg/kg-day	6E-03		
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				DODECANE	2E-07	mg/m3	2E-08	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day			
				Exp. Route Total										1E-05				1E-02
				Exp. Point Total										1E-05				1E-02
				Exp. Medium Total										1E-05				1E-02
Medium Total										1E-05				1E-02				
Soil	Surface Soil	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	4E-11	mg/kg-day	2E+05	1/(mg/kg-day)	5E-06	8E-11	mg/kg-day	1E-09	mg/kg-day	8E-02		
				ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day			
				ARSENIC	8E+00	mg/kg	6E-07	mg/kg-day	2E+00	1/(mg/kg-day)	9E-07	1E-06	mg/kg-day	3E-04	mg/kg-day	5E-03		
				BARIUM	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day			
				CADMIUM	4E+01	mg/kg	8E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-05	mg/kg-day	8E-03		
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				COPPER	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day			
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day			
				LEAD	7E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day			
				MERCURY	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day			
				SILVER	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				THALLIUM	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	1E-06	mg/kg-day	2E-05	mg/kg-day	6E-02		
				LESS CHLORINATED PCBs	7E-01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	6E-07	mg/kg-day	7E-05	mg/kg-day	8E-03		

TABLE 7.13 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Dermal	DIELDRIN	1E-01	mg/kg										
				2-METHYLNAPHTHALENE	1E+01	mg/kg	4E-06	mg/kg-day	2E+01	1/(mg/kg-day)		9E-06	mg/kg-day	5E-05	mg/kg-day	2E-03
				ACENAPHTHYLENE	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	9E-05
				BENZ(A)ANTHRACENE	8E+00	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	5E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	3E-06	mg/kg-day	7E+00	1/(mg/kg-day)	2E-05	6E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	5E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	2E-06	mg/kg-day	7E-02	1/(mg/kg-day)	1E-07	4E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	2E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	6E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	5E-07	mg/kg-day	7E+00	1/(mg/kg-day)	4E-06	1E-06	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	1E-03	mg/kg-day	2E-03
				HEXACHLOROBENZENE	1E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	4E-07	5E-07	mg/kg-day	8E-04	mg/kg-day	7E-04
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	3E-06	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-02	mg/kg-day	8E-04
				PHENANTHRENE	2E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	5E-04
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBENZENE	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	3E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				BENZENE	5E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
			Exp. Route Total								4E-05					2E-01
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	2E-10	mg/kg-day	2E+05	1/(mg/kg-day)	2E-05	4E-10	mg/kg-day	1E-09	mg/kg-day	4E-01
				ALUMINUM	7E+03	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	1E+00	mg/kg-day	5E-03
				ARSENIC	8E+00	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	4E-06	6E-06	mg/kg-day	3E-04	mg/kg-day	2E-02
				BARIUM	4E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	2E-01	mg/kg-day	1E-03
				CADMIUM	4E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	1E-03	mg/kg-day	2E-02
				CHROMIUM	1E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	3E-03	mg/kg-day	3E-02
				COPPER	2E+02	mg/kg	7E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	4E-03
				IRON	1E+04	mg/kg	4E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day	7E-01	mg/kg-day	1E-02
				LEAD	7E+02	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	9E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	1E-03
				MERCURY	1E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-04	mg/kg-day	3E-02
				SILVER	2E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	5E-03	mg/kg-day	2E-03
				THALLIUM	8E-01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	8E-05	mg/kg-day	7E-03
				VANADIUM	2E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	2E-03
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	9E-07	1E-06	mg/kg-day	2E-05	mg/kg-day	5E-02
				LESS CHLORINATED PCBs	7E-01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	4E-07	5E-07	mg/kg-day	7E-05	mg/kg-day	7E-03
				DIELDRIN	1E-01	mg/kg	3E-08	mg/kg-day	2E+01	1/(mg/kg-day)	5E-07	8E-08	mg/kg-day	5E-05	mg/kg-day	2E-03
				2-METHYLNAPHTHALENE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	4E-03	mg/kg-day	2E-03
				ACENAPHTHYLENE	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	9E-05
				BENZ(A)ANTHRACENE	8E+00	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	5E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	3E-06	mg/kg-day	7E+00	1/(mg/kg-day)	2E-05	6E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	5E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	2E-06	mg/kg-day	7E-02	1/(mg/kg-day)	1E-07	4E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	2E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	5E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	5E-07	mg/kg-day	7E+00	1/(mg/kg-day)	4E-06	1E-06	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	1E-03	mg/kg-day	3E-03
				HEXACHLOROBENZENE	1E+00	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	4E-07	7E-07	mg/kg-day	8E-04	mg/kg-day	8E-04
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	3E-06	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-02	mg/kg-day	8E-04
				PHENANTHRENE	2E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	4E-04
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg	1E-06	mg/kg-day	4E-03	1/(mg/kg-day)	4E-09	3E-06	mg/kg-day	1E-02	mg/kg-day	3E-04
				1,2-DICHLOROBENZENE	8E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	9E-02	mg/kg-day	6E-05
				1,4-DICHLOROBENZENE	3E+01	mg/kg	9E-06	mg/kg-day	5E-03	1/(mg/kg-day)	5E-08	2E-05	mg/kg-day	7E-02	mg/kg-day	3E-04

TABLE 7.13 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Soil	Surface Soil	Exposure Unit 6	Ingestion	BENZENE	5E-01	mg/kg	2E-07	mg/kg-day	6E-02	1/(mg/kg-day)	9E-09	4E-07	mg/kg-day	4E-03	mg/kg-day	9E-05	
				P-ISOPROPYLTOLUENE	4E-01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day		
				DODECANE	8E+02	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day		mg/kg-day		
				Exp. Route Total													
	Exp. Point Total																
Exp. Medium Total																	
Medium Total																	
Ground Water	Potable Water	Exposure Unit 8	Dermal	ALUMINUM	2E+04	ug/l	2E-03	mg/kg-day	2E+00	1/(mg/kg-day)	9E-07	4E-03	mg/kg-day	1E+00	mg/kg-day	4E-03	
				ANTIMONY	2E+00	ug/l	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	6E-05	mg/kg-day	5E-03	
				ARSENIC	9E+00	ug/l	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-04	mg/kg-day	4E-03	
				BARIUM	1E+03	ug/l	9E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-02	mg/kg-day	2E-02	
				BERYLLIUM	8E-01	ug/l	5E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	1E-05	mg/kg-day	8E-03	
				CADMIUM	2E+00	ug/l	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-05	mg/kg-day	1E-02	
				CHROMIUM	7E+01	ug/l	9E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	8E-05	mg/kg-day	3E-01	
				COBALT	1E+01	ug/l	3E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day		mg/kg-day		
				COPPER	9E+01	ug/l	6E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-02	mg/kg-day	3E-04	
				CYANIDE	3E+01	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	2E-02	mg/kg-day	2E-04	
				IRON	4E+04	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	7E-01	mg/kg-day	8E-03	
				LEAD	6E+01	ug/l	4E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day		mg/kg-day		
				MANGANESE	2E+03	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	6E-03	mg/kg-day	5E-02	
				MERCURY	2E+00	ug/l	1E-07	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	2E-05	mg/kg-day	1E-02	
				NICKEL	5E+01	ug/l	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	8E-04	mg/kg-day	2E-03	
				SELENIUM	4E+00	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	5E-03	mg/kg-day	1E-04	
				SILVER	2E+00	ug/l	8E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	2E-04	mg/kg-day	9E-04	
				THALLIUM	7E+00	ug/l	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	8E-05	mg/kg-day	1E-02	
				VANADIUM	4E+01	ug/l	3E-06	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	2E-04	mg/kg-day	3E-02	
				ZINC	1E+02	ug/l	4E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-01	mg/kg-day	3E-05	
				HIGHLY CHLORINATED PCBs	7E-02	ug/l		mg/kg-day		2E+00		1/(mg/kg-day)		mg/kg-day	2E-05	mg/kg-day	
				4,4'-DDD	9E-02	ug/l	7E-06	mg/kg-day		2E-01		1/(mg/kg-day)	2E-06	mg/kg-day		mg/kg-day	
				4,4'-DDT	1E+00	ug/l	1E-04	mg/kg-day		3E-01		1/(mg/kg-day)	5E-05	mg/kg-day		mg/kg-day	
				ALDRIN	3E-02	ug/l	4E-08	mg/kg-day		2E+01		1/(mg/kg-day)	6E-07	mg/kg-day	3E-05	mg/kg-day	7E-01
				ALPHA-BHC	2E-01	ug/l		mg/kg-day		6E+00		1/(mg/kg-day)		mg/kg-day		mg/kg-day	
				ENDOSULFAN II	6E-02	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day	6E-03	mg/kg-day	
				ENDOSULFAN SULFATE	2E-02	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day	6E-03	mg/kg-day	
				HEPTACHLOR EPOXIDE	1E-02	ug/l		mg/kg-day		9E+00		1/(mg/kg-day)		mg/kg-day	1E-05	mg/kg-day	
				1,1'-BIPHENYL	1E+01	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day	5E-02	mg/kg-day	
				2,4-DICHLOROPHENOL	1E+01	ug/l	4E-05	mg/kg-day				1/(mg/kg-day)		mg/kg-day	3E-03	mg/kg-day	
				2,4-DIMETHYLPHENOL	4E+03	ug/l	7E-03	mg/kg-day				1/(mg/kg-day)		mg/kg-day	2E-02	mg/kg-day	
				2-METHYLNAPHTHALENE	6E+02	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day	4E-03	mg/kg-day	
				2-METHYLPHENOL	1E+03	ug/l	1E-03	mg/kg-day				1/(mg/kg-day)		mg/kg-day	5E-02	mg/kg-day	
				2-NITROPHENOL	6E+00	ug/l	4E-06	mg/kg-day				1/(mg/kg-day)		mg/kg-day		mg/kg-day	
				3&4-METHYLPHENOL	4E+03	ug/l	5E-03	mg/kg-day				1/(mg/kg-day)		mg/kg-day	1E-02	mg/kg-day	
				4-CHLORO-3-METHYLPHENOL	1E+00	ug/l	5E-06	mg/kg-day				1/(mg/kg-day)		mg/kg-day	5E-02	mg/kg-day	
				4-METHYLPHENOL	8E+03	ug/l	9E-03	mg/kg-day				1/(mg/kg-day)		mg/kg-day	1E-05	mg/kg-day	
				4-NITROPHENOL	1E+01	ug/l	8E-06	mg/kg-day				1/(mg/kg-day)		mg/kg-day	2E-02	mg/kg-day	
				ACENAPHTHENE	1E+02	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day	2E-05	mg/kg-day	
				ACENAPHTHYLENE	2E+02	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day		mg/kg-day	
				ANTHRACENE	1E+02	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day	6E-02	mg/kg-day	
				ATRAZINE	5E+01	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day	3E-02	mg/kg-day	
				BENZ(A)ANTHRACENE	5E+01	ug/l	8E-03	mg/kg-day		7E-01		1/(mg/kg-day)	6E-03	mg/kg-day	3E-01	mg/kg-day	
				BENZO(A)PYRENE	2E+01	ug/l	5E-03	mg/kg-day		7E+00		1/(mg/kg-day)	4E-02	mg/kg-day	4E-02	mg/kg-day	
				BENZO(B)FLUORANTHENE	2E+01	ug/l	6E-03	mg/kg-day		7E-01		1/(mg/kg-day)	4E-03	mg/kg-day	1E-02	mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	2E+01	ug/l		mg/kg-day		7E-02		1/(mg/kg-day)		mg/kg-day	3E-02	mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	2E-04	mg/kg-day		1E-02		1/(mg/kg-day)	3E-06	mg/kg-day	2E-02	mg/kg-day	
				CARBAZOLE	1E+02	ug/l		mg/kg-day				1/(mg/kg-day)		mg/kg-day		mg/kg-day	
				CHRYSENE	4E+01	ug/l	5E-03	mg/kg-day		7E-03		1/(mg/kg-day)	4E-05	mg/kg-day	1E-02	mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	1E-03	mg/kg-day		7E+00		1/(mg/kg-day)	8E-03	mg/kg-day	3E-03	mg/kg-day	

TABLE 7.13 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Potable Water	Exposure Unit 8	Dermal	DIBENZOFURAN	2E+02	ug/l										
				FLUORANTHENE	2E+02	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	1E-03	mg/kg-day	6E-01
				FLUORENE	2E+02	ug/l				1/(mg/kg-day)				4E-02	mg/kg-day	
				HEXACHLOROBUTADIENE	1E+00	ug/l	3E-05	mg/kg-day	8E-02	1/(mg/kg-day)	2E-06	7E-05	mg/kg-day	4E-02	mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	2E-03	mg/kg-day	7E-01	1/(mg/kg-day)	2E-03	5E-03	mg/kg-day		mg/kg-day	
				NAPHTHALENE	4E+03	ug/l	3E-02	mg/kg-day		1/(mg/kg-day)		7E-02	mg/kg-day	2E-02	mg/kg-day	4E+00
				NITROBENZENE	3E+00	ug/l				1/(mg/kg-day)				5E-04	mg/kg-day	
				PHENANTHRENE	4E+02	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	3E-02	mg/kg-day	1E+00
				PHENOL	2E+03	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-01	mg/kg-day	9E-03
				PYRENE	1E+02	ug/l				1/(mg/kg-day)				3E-02	mg/kg-day	
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l				1/(mg/kg-day)					mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	2E-04	mg/kg-day	4E-03	1/(mg/kg-day)	8E-07	5E-04	mg/kg-day	1E-02	mg/kg-day	5E-02
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l				1/(mg/kg-day)					mg/kg-day	
				1,2-DICHLOROBENZENE	5E+02	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	9E-02	mg/kg-day	1E-01
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l				1/(mg/kg-day)					mg/kg-day	
				1,3-DICHLOROBENZENE	5E+00	ug/l	6E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day	
				1,4-DICHLOROBENZENE	5E+02	ug/l	4E-03	mg/kg-day	5E-03	1/(mg/kg-day)	2E-05	9E-03	mg/kg-day	7E-02	mg/kg-day	1E-01
				2-HEXANONE	2E+00	ug/l				1/(mg/kg-day)				2E-01	mg/kg-day	
				ACETONE	8E+01	ug/l				1/(mg/kg-day)				9E-01	mg/kg-day	
				BENZENE	6E+03	ug/l	1E-02	mg/kg-day	6E-02	1/(mg/kg-day)	6E-04	2E-02	mg/kg-day	4E-03	mg/kg-day	6E+00
				BROMODICHLOROMETHANE	3E+00	ug/l	3E-06	mg/kg-day	6E-02	1/(mg/kg-day)	2E-07	7E-06	mg/kg-day	2E-02	mg/kg-day	3E-04
				CARBON DISULFIDE	1E+01	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	1E-01	mg/kg-day	6E-04
				CHLOROBENZENE	2E+02	ug/l	8E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-02	mg/kg-day	9E-02
				CHLOROETHANE	5E+00	ug/l	3E-06	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day		mg/kg-day	
				ETHYLBENZENE	1E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E-01	mg/kg-day	2E-02
				ISOPROPYLBENZENE	4E+00	ug/l				1/(mg/kg-day)				1E-01	mg/kg-day	
				METHYLENE CHLORIDE	7E-01	ug/l	3E-07	mg/kg-day	8E-03	1/(mg/kg-day)	2E-09	8E-07	mg/kg-day	6E-02	mg/kg-day	1E-05
				P-ISOPROPYLTOLUENE	3E+00	ug/l				1/(mg/kg-day)					mg/kg-day	
				SEC-BUTYLBENZENE	1E+01	ug/l				1/(mg/kg-day)					mg/kg-day	
				STYRENE	8E+02	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	2E-01	mg/kg-day	5E-02
				TETRACHLOROETHENE	3E-01	ug/l	2E-06	mg/kg-day	5E-01	1/(mg/kg-day)	1E-06	5E-06	mg/kg-day	1E-02	mg/kg-day	5E-04
				TOLUENE	1E+03	ug/l	5E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	8E-02	mg/kg-day	2E-01
				VINYL CHLORIDE	1E+00	ug/l	7E-07	mg/kg-day	8E-01	1/(mg/kg-day)	5E-07	2E-06	mg/kg-day	3E-03	mg/kg-day	5E-04
				XYLENES, TOTAL	1E+03	ug/l				1/(mg/kg-day)				2E-01	mg/kg-day	
			Exp. Route Total								6E-02					1E+01
			Ingestion	ALUMINUM	2E+04	ug/l	3E-01	mg/kg-day		1/(mg/kg-day)		7E-01	mg/kg-day	1E+00	mg/kg-day	7E-01
				ANTIMONY	2E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	4E-04	mg/kg-day	2E-01
				ARSENIC	9E+00	ug/l	1E-04	mg/kg-day	2E+00	1/(mg/kg-day)	2E-04	3E-04	mg/kg-day	3E-04	mg/kg-day	9E-01
				BARIIUM	1E+03	ug/l	2E-02	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	2E-01	mg/kg-day	2E-01
				BERYLLIUM	8E-01	ug/l	9E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-03	mg/kg-day	1E-02
				CADMIUM	2E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	1E-03	mg/kg-day	5E-02
				CHROMIUM	7E+01	ug/l	8E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-03	mg/kg-day	6E-01
				COBALT	1E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day	
				COPPER	9E+01	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	4E-02	mg/kg-day	6E-02
				CYANIDE	3E+01	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	2E-02	mg/kg-day	4E-02
				IRON	4E+04	ug/l	5E-01	mg/kg-day		1/(mg/kg-day)		1E+00	mg/kg-day	7E-01	mg/kg-day	2E+00
				LEAD	6E+01	ug/l	8E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day		mg/kg-day	
				MANGANESE	2E+03	ug/l	2E-02	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	1E-01	mg/kg-day	4E-01
				MERCURY	2E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	3E-04	mg/kg-day	2E-01
				NICKEL	5E+01	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-02	mg/kg-day	7E-02
				SELENIUM	4E+00	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	5E-03	mg/kg-day	2E-02
				SILVER	2E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	5E-03	mg/kg-day	1E-02
				THALLIUM	7E+00	ug/l	8E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	8E-05	mg/kg-day	2E+00
				VANADIUM	4E+01	ug/l	5E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	9E-03	mg/kg-day	1E-01
				ZINC	1E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-01	mg/kg-day	9E-03
				HIGHLY CHLORINATED PCBs	7E-02	ug/l	8E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	2E-06	mg/kg-day	2E-05	mg/kg-day	1E-01
				4,4'-DDD	9E-02	ug/l	1E-06	mg/kg-day	2E-01	1/(mg/kg-day)	2E-07	2E-06	mg/kg-day		mg/kg-day	
				4,4'-DDT	1E+00	ug/l	1E-05	mg/kg-day	3E-01	1/(mg/kg-day)	4E-06	3E-05	mg/kg-day	5E-04	mg/kg-day	6E-02

TABLE 7.13 RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Potable Water	Exposure Unit 8	Ingestion	ALDRIN	3E-02	ug/l	4E-07	mg/kg-day	2E+01	1/(mg/kg-day)	7E-06	9E-07	mg/kg-day	3E-05	mg/kg-day	3E-02
				ALPHA-BHC	2E-01	ug/l	2E-06	mg/kg-day	6E+00	1/(mg/kg-day)	1E-05	5E-06	mg/kg-day		mg/kg-day	
				ENDOSULFAN II	6E-02	ug/l	7E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	6E-03	mg/kg-day	3E-04
				ENDOSULFAN SULFATE	2E-02	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	6E-03	mg/kg-day	1E-04
				HEPTACHLOR EPOXIDE	1E-02	ug/l	1E-07	mg/kg-day	9E+00	1/(mg/kg-day)	1E-06	3E-07	mg/kg-day	1E-05	mg/kg-day	2E-02
				1,1'-BIPHENYL	1E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	5E-02	mg/kg-day	7E-03
				2,4-DICHLOROPHENOL	1E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	3E-03	mg/kg-day	9E-02
				2,4-DIMETHYLPHENOL	4E+03	ug/l	5E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	2E-02	mg/kg-day	6E+00
				2-METHYLNAPHTHALENE	6E+02	ug/l	7E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	4E-03	mg/kg-day	4E+00
				2-METHYLPHENOL	1E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	5E-02	mg/kg-day	5E-01
				2-NITROPHENOL	6E+00	ug/l	7E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day			
				3&4-METHYLPHENOL	4E+03	ug/l	5E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	5E-02	mg/kg-day	2E+00
				4-CHLORO-3-METHYLPHENOL	1E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day			
				4-METHYLPHENOL	8E+03	ug/l	1E-01	mg/kg-day		1/(mg/kg-day)		2E-01	mg/kg-day	5E-02	mg/kg-day	5E+00
				4-NITROPHENOL	1E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day			
				ACENAPHTHENE	1E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	6E-02	mg/kg-day	5E-02
				ACENAPHTHYLENE	2E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	3E-02	mg/kg-day	2E-01
				ANTHRACENE	1E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-01	mg/kg-day	1E-02
				ATRAZINE	5E+01	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	4E-02	mg/kg-day	4E-02
				BENZ(A)ANTHRACENE	5E+01	ug/l	6E-04	mg/kg-day	7E-01	1/(mg/kg-day)	5E-04	1E-03	mg/kg-day			
				BENZO(A)PYRENE	2E+01	ug/l	2E-04	mg/kg-day	7E+00	1/(mg/kg-day)	2E-03	5E-04	mg/kg-day			
				BENZO(B)FLUORANTHENE	2E+01	ug/l	3E-04	mg/kg-day	7E-01	1/(mg/kg-day)	2E-04	6E-04	mg/kg-day			
				BENZO(G,H,I)PERYLENE	5E+00	ug/l	6E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	5E-03
				BENZO(K)FLUORANTHENE	2E+01	ug/l	2E-04	mg/kg-day	7E-02	1/(mg/kg-day)	2E-05	5E-04	mg/kg-day			
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	1E-04	mg/kg-day	1E-02	1/(mg/kg-day)	2E-06	3E-04	mg/kg-day	2E-02	mg/kg-day	1E-02
				CARBAZOLE	1E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day			
				CHRYSENE	4E+01	ug/l	4E-04	mg/kg-day	7E-03	1/(mg/kg-day)	3E-06	1E-03	mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	3E-05	mg/kg-day	7E+00	1/(mg/kg-day)	2E-04	8E-05	mg/kg-day			
				DIBENZOFURAN	2E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	1E-03	mg/kg-day	5E+00
				FLUORANTHENE	2E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	4E-02	mg/kg-day	1E-01
				FLUORENE	2E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	4E-02	mg/kg-day	1E-01
				HEXACHLOROBUTADIENE	1E+00	ug/l	1E-05	mg/kg-day	8E-02	1/(mg/kg-day)	9E-07	3E-05	mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	1E-04	mg/kg-day	7E-01	1/(mg/kg-day)	7E-05	2E-04	mg/kg-day			
				NAPHTHALENE	4E+03	ug/l	5E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	2E-02	mg/kg-day	5E+00
				NITROBENZENE	3E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	5E-04	mg/kg-day	1E-01
				PHENANTHRENE	4E+02	ug/l	5E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	3E-02	mg/kg-day	4E-01
				PHENOL	2E+03	ug/l	2E-02	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	3E-01	mg/kg-day	2E-01
				PYRENE	1E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-02	mg/kg-day	9E-02
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day			
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	2E-04	mg/kg-day	4E-03	1/(mg/kg-day)	6E-07	4E-04	mg/kg-day	1E-02	mg/kg-day	4E-02
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day			
				1,2-DICHLOROBENZENE	5E+02	ug/l	6E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	9E-02	mg/kg-day	2E-01
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day			
				1,3-DICHLOROBENZENE	5E+00	ug/l	6E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day			
				1,4-DICHLOROBENZENE	5E+02	ug/l	5E-03	mg/kg-day	5E-03	1/(mg/kg-day)	3E-05	1E-02	mg/kg-day	7E-02	mg/kg-day	2E-01
				2-HEXANONE	2E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	2E-01	mg/kg-day	3E-04
				ACETONE	8E+01	ug/l	9E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	9E-01	mg/kg-day	2E-03
				BENZENE	6E+03	ug/l	7E-02	mg/kg-day	6E-02	1/(mg/kg-day)	4E-03	2E-01	mg/kg-day	4E-03	mg/kg-day	4E+01
				BROMODICHLOROMETHANE	3E+00	ug/l	4E-05	mg/kg-day	6E-02	1/(mg/kg-day)	2E-06	8E-05	mg/kg-day	2E-02	mg/kg-day	4E-03
				CARBON DISULFIDE	1E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-01	mg/kg-day	3E-03
				CHLOROBENZENE	2E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	2E-02	mg/kg-day	2E-01
				CHLOROETHANE	5E+00	ug/l	5E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day			
				ETHYLBENZENE	1E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	1E-01	mg/kg-day	4E-02
				ISOPROPYLBENZENE	4E+00	ug/l	5E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-01	mg/kg-day	1E-03
				METHYLENE CHLORIDE	7E-01	ug/l	9E-06	mg/kg-day	8E-03	1/(mg/kg-day)	7E-08	2E-05	mg/kg-day	6E-02	mg/kg-day	3E-04
				P-ISOPROPYLTOLUENE	3E+00	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day			
				SEC-BUTYLBENZENE	1E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day			
				STYRENE	8E+02	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	2E-01	mg/kg-day	1E-01

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REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations							
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient			
							Value	Units	Value	Units		Value	Units	Value	Units				
Ground Water	Potable Water	Exposure Unit 8	Ingestion	TETRACHLOROETHENE	3E-01	ug/l	3E-06	mg/kg-day	5E-01	1/(mg/kg-day)	2E-06	8E-06	mg/kg-day	1E-02	mg/kg-day	8E-04			
				TOLUENE	1E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	8E-02	mg/kg-day	4E-01			
				VINYL CHLORIDE	1E+00	ug/l	1E-05	mg/kg-day	8E-01	1/(mg/kg-day)	1E-05	3E-05	mg/kg-day	3E-03	mg/kg-day	1E-02			
				XYLENES, TOTAL	1E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	2E-01	mg/kg-day	1E-01			
			Exp. Route Total								7E-03					8E+01			
		Exp. Point Total									6E-02					9E+01			
	Exp. Medium Total										6E-02					9E+01			
	Shower Vapor	Exposure Unit 8	Inhalation	1,2,3-TRICHLOROBENZENE	1E-01	mg/m3	4E-04	mg/kg-day		1/(mg/kg-day)		9E-04	mg/kg-day		mg/kg-day				
				1,2,4-TRICHLOROBENZENE	2E-01	mg/m3	5E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day		mg/kg-day				
				1,2,4-TRIMETHYLBENZENE	4E+00	mg/m3	1E-02	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	2E-03	mg/kg-day	1E+01			
				1,2-DICHLOROBENZENE	7E+00	mg/m3	2E-02	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	4E-02	mg/kg-day	1E+00			
				1,3,5-TRIMETHYLBENZENE	3E+00	mg/m3	7E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day		mg/kg-day				
				1,3-DICHLOROBENZENE	7E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day		mg/kg-day				
				1,4-DICHLOROBENZENE	6E+00	mg/m3	2E-02	mg/kg-day	4E-02	1/(mg/kg-day)	6E-04	4E-02	mg/kg-day	2E-01	mg/kg-day	2E-01			
				2-HEXANONE	2E-02	mg/m3	7E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	6E-02	mg/kg-day	3E-03			
				ACETONE	1E+00	mg/m3	3E-03	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	9E+00	mg/kg-day	7E-04			
				BENZENE	7E+01	mg/m3	2E-01	mg/kg-day	3E-02	1/(mg/kg-day)	5E-03	5E-01	mg/kg-day	9E-03	mg/kg-day	5E+01			
				BROMODICHLOROMETHANE	4E-02	mg/m3	1E-04	mg/kg-day	1E-01	1/(mg/kg-day)	1E-05	2E-04	mg/kg-day		mg/kg-day				
				CARBON DISULFIDE	2E-01	mg/m3	4E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-01	mg/kg-day	5E-03			
				CHLOROETHANE	2E+00	mg/m3	6E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day		mg/kg-day				
				CHLOROFORM	6E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	3E+00	mg/kg-day	1E-04			
				ETHYLBENZENE	1E-01	mg/m3	4E-04	mg/kg-day	8E-02	1/(mg/kg-day)	3E-05	9E-04	mg/kg-day	3E-02	mg/kg-day	3E-02			
				ISOPROPYLBENZENE	2E+00	mg/m3	5E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	3E-01	mg/kg-day	4E-02			
				METHYLENE CHLORIDE	5E-02	mg/m3	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-01	mg/kg-day	3E-03			
				P-ISOPROPYLTOLUENE	9E-03	mg/m3	2E-05	mg/kg-day	2E-03	1/(mg/kg-day)	4E-08	6E-05	mg/kg-day	3E-01	mg/kg-day	2E-04			
				SEC-BUTYLBENZENE	4E-02	mg/m3	1E-04	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day				
				STYRENE	1E-01	mg/m3	4E-04	mg/kg-day		1/(mg/kg-day)		9E-04	mg/kg-day		mg/kg-day				
				TETRACHLOROETHENE	1E+01	mg/m3	3E-02	mg/kg-day		1/(mg/kg-day)		6E-02	mg/kg-day	3E+00	mg/kg-day	2E-02			
				TOLUENE	4E-03	mg/m3	1E-05	mg/kg-day	2E-05	1/(mg/kg-day)	2E-10	2E-05	mg/kg-day	8E-02	mg/kg-day	3E-04			
				VINYL CHLORIDE	2E+01	mg/m3	4E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	1E+00	mg/kg-day	7E-02			
				XYLENES, TOTAL	1E-02	mg/m3	4E-05	mg/kg-day	2E-02	1/(mg/kg-day)	6E-07	9E-05	mg/kg-day	3E-02	mg/kg-day	3E-03			
					9E+00	mg/m3	2E-02	mg/kg-day		1/(mg/kg-day)		6E-02	mg/kg-day	3E-02	mg/kg-day	2E+00			
Exp. Route Total										6E-03					7E+01				
Exp. Point Total									6E-03					7E+01					
Exp. Medium Total										6E-03					7E+01				
		Medium Total											7E-02					2E+02	
		Total of Receptor Risks Across All Media											7E-02	Total of Receptor Hazards Across All Media					2E+02

TABLE 7.13a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	2E-07	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day		mg/kg-day	3E-04
				ARSENIC	3E-09	mg/m3	2E-10	mg/kg-day	2E+01	1/(mg/kg-day)	3E-09	5E-10	mg/kg-day	1E-03	mg/kg-day	3E-05
				CADMIUM	8E-09	mg/m3	6E-10	mg/kg-day	6E+00	1/(mg/kg-day)	4E-09	1E-09	mg/kg-day		mg/kg-day	
				CHROMIUM	5E-08	mg/m3	4E-09	mg/kg-day	4E+01	1/(mg/kg-day)	2E-07	9E-09	mg/kg-day	3E-05	mg/kg-day	3E-04
				COPPER	5E-08	mg/m3	4E-09	mg/kg-day		1/(mg/kg-day)		9E-09	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	4E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	1E-05	mg/kg-day	2E-03
				MERCURY	8E-10	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day	9E-05	mg/kg-day	2E-06
				VANADIUM	6E-09	mg/m3	5E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	3E-11	mg/kg-day	2E+00	1/(mg/kg-day)	6E-11	7E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	7E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	8E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	5E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	4E-08	mg/kg-day	3E-02	1/(mg/kg-day)	1E-09	9E-08	mg/kg-day	9E-03	mg/kg-day	1E-05
			Exp. Route Total								2E-07					2E-03
		Exp. Point Total									2E-07					2E-03
	Exp. Medium Total										2E-07					2E-03
Medium Total											2E-07					2E-03
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	3E-07	5E-12	mg/kg-day	1E-09	mg/kg-day	5E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	4E-07	mg/kg-day	2E+00	1/(mg/kg-day)	7E-07	1E-06	mg/kg-day	3E-04	mg/kg-day	3E-03
				CADMIUM	2E+01	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-05	mg/kg-day	4E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	6E-07	7E-07	mg/kg-day	2E-05	mg/kg-day	4E-02
				ACENAPHTHYLENE	2E+00	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	7E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	5E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	7E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	6E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	1E-06	mg/kg-day	7E-02	1/(mg/kg-day)	7E-08	2E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	7E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	4E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	1E-03	mg/kg-day	9E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	4E-07	1E-06	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	3E-04
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								2E-05					5E-02

TABLE 7.13a RME
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	9E-12	mg/kg-day	2E+05	1/(mg/kg-day)	1E-06	2E-11	mg/kg-day	1E-09	mg/kg-day	2E-02
				ALUMINUM	5E+03	mg/kg	2E-03	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	1E+00	mg/kg-day	4E-03
				ARSENIC	6E+00	mg/kg	2E-06	mg/kg-day	2E+00	1/(mg/kg-day)	3E-06	4E-06	mg/kg-day	3E-04	mg/kg-day	1E-02
				CADMIUM	2E+01	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02
				CHROMIUM	1E+02	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	3E-03	mg/kg-day	3E-02
				COPPER	1E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	4E-02	mg/kg-day	2E-03
				IRON	1E+04	mg/kg	4E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day	7E-01	mg/kg-day	1E-02
				MANGANESE	3E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	2E-03
				MERCURY	2E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-04	mg/kg-day	4E-03
				VANADIUM	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	1E-03
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	6E-07	mg/kg-day	2E-05	mg/kg-day	3E-02
				ACENAPHTHYLENE	2E+00	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	6E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	5E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	7E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	1E-06	mg/kg-day	7E-02	1/(mg/kg-day)	7E-08	2E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	3E-06	mg/kg-day	7E-03	1/(mg/kg-day)	2E-08	7E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	4E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	4E-07	1E-06	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	3E-04
				BENZENE	1E-03	mg/kg	3E-10	mg/kg-day	6E-02	1/(mg/kg-day)	2E-11	7E-10	mg/kg-day	4E-03	mg/kg-day	2E-07
			Exp. Route Total								2E-05					1E-01
		Exp. Point Total									5E-05					2E-01
	Exp. Medium Total										5E-05					2E-01
Medium Total											5E-05					2E-01
Total of Receptor Risks Across All Media											5E-05	Total of Receptor Hazards Across All Media				2E-01

RAGS Table 7 CT Series

TABLE 7.1 CT Supplement A
 CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
 CENTRAL TENDENCY
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Older Child Trespasser
Receptor Age:	12 to < 18 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations							
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk	
							Value		Units	Value		Units		
							12-16 yrs	16-18 yrs		12-16 yrs (ADAF=3)	16-18 yrs (ADAF=1)			
Soil	Surface Soil	EU-1	Ingestion	Benz(a)anthracene	1.5E+01	mg/kg	3.6E-08	1.5E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	9.E-08	
				Benzo(a)pyrene	1.5E+01	mg/kg	3.5E-08	1.5E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	9.E-07	
				Benzo(b)fluoranthene	1.3E+01	mg/kg	3.1E-08	1.3E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	8.E-08	
				Benzo(k)fluoranthene	1.2E+01	mg/kg	2.8E-08	1.2E-08	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	7.E-09	
				Chrysene	1.5E+01	mg/kg	3.6E-08	1.5E-08	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	9.E-10	
				Dibenz(a,h)anthracene	3.3E+00	mg/kg	7.8E-09	3.3E-09	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	2.E-07	
				Indeno(1,2,3-cd)pyrene	9.1E+00	mg/kg	2.2E-08	9.1E-09	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	5.E-08	
			Dermal	Benz(a)anthracene	1.5E+01	mg/kg	1.9E-07	8.8E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	5.E-07	
				Benzo(a)pyrene	1.5E+01	mg/kg	1.8E-07	8.6E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	5.E-06	
				Benzo(b)fluoranthene	1.3E+01	mg/kg	1.6E-07	7.4E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	4.E-07	
				Benzo(k)fluoranthene	1.2E+01	mg/kg	1.5E-07	6.9E-08	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	4.E-08	
				Chrysene	1.5E+01	mg/kg	1.9E-07	8.7E-08	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	5.E-09	
				Dibenz(a,h)anthracene	3.3E+00	mg/kg	4.0E-08	1.9E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	1.E-06	
				Indeno(1,2,3-cd)pyrene	9.1E+00	mg/kg	1.1E-07	5.2E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	3.E-07	
	Fugitive Dust	EU-1	Inhalation	Benz(a)anthracene	1.1E-08	mg/m ³	1.2E-12	5.3E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA	
				Benzo(a)pyrene	1.1E-08	mg/m ³	1.2E-12	5.2E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA	
				Benzo(b)fluoranthene	9.4E-09	mg/m ³	1.0E-12	4.5E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA	
				Benzo(k)fluoranthene	8.8E-09	mg/m ³	9.6E-13	4.2E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA	
				Chrysene	1.1E-08	mg/m ³	1.2E-12	5.2E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA	
				Dibenz(a,h)anthracene	2.4E-09	mg/m ³	2.6E-13	1.1E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA	
				Indeno(1,2,3-cd)pyrene	6.6E-09	mg/m ³	7.3E-13	3.2E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA	
Sediment	Surface Sediment	EU-1	Ingestion	Benz(a)anthracene	2.8E+02	mg/kg	6.6E-07	2.8E-07	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	2.E-06	
				Benzo(a)pyrene	6.3E+01	mg/kg	1.5E-07	6.3E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	4.E-06	
				Benzo(b)fluoranthene	9.4E+01	mg/kg	2.2E-07	9.5E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	6.E-07	
				Benzo(k)fluoranthene	3.5E+01	mg/kg	8.4E-08	3.5E-08	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	2.E-08	
				Chrysene	8.7E+01	mg/kg	2.1E-07	8.7E-08	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	5.E-09	
				Dibenz(a,h)anthracene	1.1E+01	mg/kg	2.6E-08	1.1E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	6.E-07	
				Indeno(1,2,3-cd)pyrene	3.3E+01	mg/kg	7.8E-08	3.3E-08	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	2.E-07	
			Dermal	Benz(a)anthracene	2.8E+02	mg/kg	3.4E-06	1.6E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	9.E-06	
				Benzo(a)pyrene	6.3E+01	mg/kg	7.8E-07	3.6E-07	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	2.E-05	
				Benzo(b)fluoranthene	9.4E+01	mg/kg	1.2E-06	5.5E-07	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	3.E-06	
				Benzo(k)fluoranthene	3.5E+01	mg/kg	4.4E-07	2.0E-07	mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	1.E-07	
				Chrysene	8.7E+01	mg/kg	1.1E-06	5.0E-07	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	3.E-08	
				Dibenz(a,h)anthracene	1.1E+01	mg/kg	1.3E-07	6.3E-08	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	3.E-06	
				Indeno(1,2,3-cd)pyrene	3.3E+01	mg/kg	4.1E-07	1.9E-07	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	1.E-06	

TABLE 7.1 CT Supplement A
 CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
 CENTRAL TENDENCY
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Older Child Trespasser
Receptor Age:	12 to < 18 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							12-16 yrs	16-18 yrs		12-16 yrs (ADAF=3)	16-18 yrs (ADAF=1)		
Water	Surface Water	EU-1	Dermal	Benz(a)anthracene	3.8E+00	µg/L	4.7E-06	2.2E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	1.E-05
				Benzo(a)pyrene	2.2E+00	µg/L	4.7E-06	2.2E-06	mg/kg/day	2.2E+01	7.3E+00	1/(mg/kg-day)	1.E-04
				Benzo(b)fluoranthene	2.9E+00	µg/L	6.2E-06	2.9E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	2.E-05
				Benzo(k)fluoranthene	1.6E+00	µg/L			mg/kg/day	2.2E-01	7.3E-02	1/(mg/kg-day)	
				Chrysene	2.9E+00	µg/L	3.6E-06	1.7E-06	mg/kg/day	2.2E-02	7.3E-03	1/(mg/kg-day)	9.E-08
				Indeno(1,2,3-cd)pyrene	1.4E+00	µg/L	3.0E-06	1.4E-06	mg/kg/day	2.2E+00	7.3E-01	1/(mg/kg-day)	8.E-06

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup

TABLE 7.2 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 1	Inhalation	1,2-DICHLOROBENZENE	6E-04	mg/m3	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	4E-02	mg/kg-day	4E-05
				1,4-DICHLOROBENZENE	2E-03	mg/m3	8E-07	mg/kg-day	4E-02	1/(mg/kg-day)	3E-08	6E-06	mg/kg-day	2E-01	mg/kg-day	3E-05
				BENZENE	2E-04	mg/m3	7E-08	mg/kg-day	3E-02	1/(mg/kg-day)	2E-09	5E-07	mg/kg-day	9E-03	mg/kg-day	6E-05
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
		DODECANE	6E-07	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day			
				Exp. Route Total							3E-08				2E-04	
				Exp. Point Total							3E-08				2E-04	
				Exp. Medium Total							3E-08				2E-04	
Medium Total												3E-08				2E-04
Surface Water	Surface water	Exposure Unit 1	Dermal	ANTIMONY	2E+00	ug/l	3E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	6E-05	mg/kg-day	4E-04
				ARSENIC	3E+00	ug/l	6E-09	mg/kg-day	2E+00	1/(mg/kg-day)	9E-09	5E-08	mg/kg-day	3E-04	mg/kg-day	2E-04
				CHROMIUM	6E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	8E-05	mg/kg-day	2E-03
				IRON	6E+03	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	7E-01	mg/kg-day	1E-04
				LEAD	1E+01	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day	
				MANGANESE	4E+02	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	6E-03	mg/kg-day	1E-03
				MERCURY	1E-01	ug/l	2E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day	2E-05	mg/kg-day	6E-05
				THALLIUM	4E+00	ug/l	7E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	8E-05	mg/kg-day	7E-04
				VANADIUM	2E+00	ug/l	3E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	2E-04	mg/kg-day	1E-04
				ZINC	3E+02	ug/l	4E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-01	mg/kg-day	1E-05
				2,4-DIMETHYLPHENOL	5E+01	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	2E-02	mg/kg-day	6E-04
				2-METHYLNAPHTHALENE	6E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				3&4-METHYLPHENOL	6E+01	ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	5E-02	mg/kg-day	2E-04
				ACENAPHTHENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day	
				ACENAPHTHYLENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				BENZ(A)ANTHRACENE	4E+00	ug/l	9E-06	mg/kg-day	7E-01	1/(mg/kg-day)	7E-06	7E-05	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+00	ug/l	8E-06	mg/kg-day	7E+00	1/(mg/kg-day)	6E-05	6E-05	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	3E+00	ug/l	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	9E-06	9E-05	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				BENZO(K)FLUORANTHENE	2E+00	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+00	ug/l	2E-06	mg/kg-day	1E-02	1/(mg/kg-day)	2E-08	1E-05	mg/kg-day	2E-02	mg/kg-day	7E-04
				CARBAZOLE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				CHRYSENE	3E+00	ug/l	7E-06	mg/kg-day	7E-03	1/(mg/kg-day)	5E-08	5E-05	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day	
				FLUORENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	1E+00	ug/l	6E-06	mg/kg-day	7E-01	1/(mg/kg-day)	4E-06	5E-05	mg/kg-day		mg/kg-day	
				NAPHTHALENE	1E+03	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-02	mg/kg-day	6E-02
				PHENANTHRENE	2E+01	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	3E-02	mg/kg-day	2E-03
				PYRENE	6E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				1,2,4-TRIMETHYLBENZENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,3,5-TRIMETHYLBENZENE	9E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,4-DICHLORO BENZENE	8E+00	ug/l	1E-06	mg/kg-day	5E-03	1/(mg/kg-day)	6E-09	8E-06	mg/kg-day	7E-02	mg/kg-day	1E-04
				BENZENE	4E+01	ug/l	2E-06	mg/kg-day	6E-02	1/(mg/kg-day)	8E-08	1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				TOLUENE	2E+02	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	8E-02	mg/kg-day	2E-03
				XYLENES, TOTAL	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
				Exp. Route Total							8E-05				7E-02	
				Exp. Point Total							8E-05				7E-02	
				Exp. Medium Total							8E-05				7E-02	
Medium Total												8E-05				7E-02
Total of Receptor Risks Across All Media										2E-04	Total of Receptor Hazards Across All Media					6E+00

TABLE 7.3 CT
 CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
 CENTRAL TENDENCY
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Water	Surface Water	Exposure Unit 1	Dermal	1,4-DICHLOROBENZENE	8E+00	ug/l	2E-07	mg/kg-day	5E-03	1/(mg/kg-day)	1E-09	2E-06	mg/kg-day	7E-02	mg/kg-day	3E-05
				BENZENE	4E+01	ug/l	4E-07	mg/kg-day	6E-02	1/(mg/kg-day)	2E-08	3E-06	mg/kg-day	4E-03	mg/kg-day	9E-04
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				TOLUENE	2E+02	ug/l	5E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	8E-02	mg/kg-day	4E-04
				XYLENES, TOTAL	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
		Exp. Route Total									1E-05					2E-02
		Exp. Point Total									1E-05					2E-02
	Exp. Medium Total										1E-05					2E-02
Medium Total											1E-05					2E-02
Total of Receptor Risks Across All Media											2E-05	Total of Receptor Hazards Across All Media				2E-01

TABLE 7.3a CT
 CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
 CENTRAL TENDENCY
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Shallow Ground Water	Shallow Ground Water	Exposure Unit 9	Dermal	PYRENE	7E+00	ug/l										
				1,4-DICHLOROBENZENE	3E-01	ug/l										
				BENZENE	9E-01	ug/l										
			Exp. Route Total													
		Exp. Point Total														
	Exp. Medium Total															
Medium Total																
Total of Receptor Risks Across All Media												Total of Receptor Hazards Across All Media				

TABLE 7.4 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Surface Water	Surface Water	Exposure Unit 1	Dermal	ANTIMONY	2E+00	ug/l	3E-09	mg/kg-day		1/(mg/kg-day)			2E-07	mg/kg-day	6E-05	mg/kg-day	4E-03
				ARSENIC	3E+00	ug/l	6E-09	mg/kg-day	2E+00	1/(mg/kg-day)	9E-09	4E-07	mg/kg-day	3E-04	mg/kg-day	1E-03	
				CHROMIUM	6E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	8E-05	mg/kg-day	2E-02	
				IRON	6E+03	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day	7E-01	mg/kg-day	1E-03	
				LEAD	1E+01	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day		
				MANGANESE	4E+02	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	6E-03	mg/kg-day	9E-03	
				MERCURY	1E-01	ug/l	2E-10	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	2E-05	mg/kg-day	6E-04	
				THALLIUM	4E+00	ug/l	7E-09	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	8E-05	mg/kg-day	6E-03	
				VANADIUM	2E+00	ug/l	3E-09	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	2E-04	mg/kg-day	9E-04	
				ZINC	3E+02	ug/l	4E-07	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-01	mg/kg-day	9E-05	
				2,4-DIMETHYLPHENOL	5E+01	ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	2E-02	mg/kg-day	4E-03	
				2-METHYLNAPHTHALENE	6E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day		
				3&4-METHYLPHENOL	6E+01	ug/l	9E-07	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	5E-02	mg/kg-day	1E-03	
				ACENAPHTHENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day		
				ACENAPHTHYLENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				BENZ(A)ANTHRACENE	4E+00	ug/l	5E-06	mg/kg-day	7E-01	1/(mg/kg-day)	3E-06	3E-04	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	2E+00	ug/l	4E-06	mg/kg-day	7E+00	1/(mg/kg-day)	3E-05	3E-04	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	3E+00	ug/l	6E-06	mg/kg-day	7E-01	1/(mg/kg-day)	4E-06	4E-04	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	2E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				BENZO(K)FLUORANTHENE	2E+00	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+00	ug/l	9E-07	mg/kg-day	1E-02	1/(mg/kg-day)	1E-08	6E-05	mg/kg-day	2E-02	mg/kg-day	3E-03	
				CARBAZOLE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				CHRYSENE	3E+00	ug/l	3E-06	mg/kg-day	7E-03	1/(mg/kg-day)	3E-08	2E-04	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day		
				FLUORENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day		
				INDENO(1,2,3-CD)PYRENE	1E+00	ug/l	3E-06	mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	2E-04	mg/kg-day		mg/kg-day		
				NAPHTHALENE	1E+03	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	2E-02	mg/kg-day	3E-01	
				PHENANTHRENE	2E+01	ug/l	5E-06	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	3E-02	mg/kg-day	1E-02	
				PYRENE	6E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				1,2,4-TRIMETHYLBENZENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				1,3,5-TRIMETHYLBENZENE	9E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				1,4-DICHLOROENZENE	8E+00	ug/l	6E-07	mg/kg-day	5E-03	1/(mg/kg-day)	3E-09	5E-05	mg/kg-day	7E-02	mg/kg-day	6E-04	
				BENZENE	4E+01	ug/l	1E-06	mg/kg-day	6E-02	1/(mg/kg-day)	7E-08	9E-05	mg/kg-day	4E-03	mg/kg-day	2E-02	
				DICHLOROENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day		
				TOLUENE	2E+02	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		9E-04	mg/kg-day	8E-02	mg/kg-day	1E-02	
				XYLENES, TOTAL	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day		
				Exp. Route Total				4E-05							4E-01		
				Exp. Point Total				4E-05							4E-01		
				Exp. Medium Total				4E-05							4E-01		
				Medium Total				4E-05							4E-01		
				Total of Receptor Risks Across All Media							1E-04		Total of Receptor Hazards Across All Media				

TABLE 7.4a CT
 CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
 CENTRAL TENDENCY
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Shallow Ground Water	Exposure Unit 9	Dermal	PHENANTHRENE	7E+00	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	4E-03
				PYRENE	7E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	3E-01	ug/l	3E-08	mg/kg-day	5E-03	1/(mg/kg-day)	1E-10	2E-06	mg/kg-day	7E-02	mg/kg-day	3E-05
				BENZENE	9E-01	ug/l	2E-08	mg/kg-day	6E-02	1/(mg/kg-day)	1E-09	2E-06	mg/kg-day	4E-03	mg/kg-day	4E-04
				Exp. Route Total							1E-04					8E-01
		Exp. Point Total									1E-04					8E-01
	Exp. Medium Total										1E-04					8E-01
Medium Total											1E-04					8E-01
Total of Receptor Risks Across All Media											1E-04	Total of Receptor Hazards Across All Media				1E+00

TABLE 7.6 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Ditch Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations								
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient				
							Value	Units	Value	Units		Value	Units	Value	Units					
Surface Sediment	Outdoor Air	Exposure Unit 3	Inhalation	BENZENE	2E-04	mg/m3	7E-08	mg/kg-day	3E-02	1/(mg/kg-day)	2E-09	5E-07	mg/kg-day	9E-03	mg/kg-day	6E-05				
			Exp. Route Total								2E-09					6E-05				
		Exp. Point Total									2E-09					6E-05				
	Exp. Medium Total									2E-09					6E-05					
Medium Total																2E-09				6E-05
Sediment	Surface Sediment	Exposure Unit 3	Dermal	2,3,7,8-TCDD Equivalent	9E-06	mg/kg	5E-15	mg/kg-day	2E+05	1/(mg/kg-day)	7E-10	4E-14	mg/kg-day	1E-09	mg/kg-day	4E-05				
				ARSENIC	4E+00	mg/kg	2E-09	mg/kg-day	2E+00	1/(mg/kg-day)	3E-09	1E-08	mg/kg-day	3E-04	mg/kg-day	5E-05				
				CHROMIUM	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day					
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day					
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day					
				MERCURY	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day					
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day					
				2-METHYLNAPHTHALENE	2E+01	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	4E-03	mg/kg-day	7E-05				
				ACENAPHTHYLENE	4E+00	mg/kg	8E-09	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	3E-02	mg/kg-day	2E-06				
				BENZ(A)ANTHRACENE	1E+00	mg/kg	3E-09	mg/kg-day	7E-01	1/(mg/kg-day)	2E-09	2E-08	mg/kg-day		mg/kg-day					
				BENZO(A)PYRENE	1E+00	mg/kg	3E-09	mg/kg-day	7E+00	1/(mg/kg-day)	2E-08	2E-08	mg/kg-day		mg/kg-day					
				BENZO(B)FLUORANTHENE	1E+00	mg/kg	3E-09	mg/kg-day	7E-01	1/(mg/kg-day)	2E-09	2E-08	mg/kg-day		mg/kg-day					
				BENZO(G,H,I)PERYLENE	1E+00	mg/kg	3E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	3E-02	mg/kg-day	7E-07				
				DIBENZ(A,H)ANTHRACENE	1E-01	mg/kg	2E-10	mg/kg-day	7E+00	1/(mg/kg-day)	2E-09	2E-09	mg/kg-day		mg/kg-day					
				DIBENZOFURAN	4E+00	mg/kg	6E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	1E-03	mg/kg-day	5E-05				
				INDENO(1,2,3-CD)PYRENE	1E+00	mg/kg	2E-09	mg/kg-day	7E-01	1/(mg/kg-day)	2E-09	2E-08	mg/kg-day		mg/kg-day					
				NAPHTHALENE	6E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	2E-02	mg/kg-day	5E-05				
				PHENANTHRENE	1E+01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	6E-06				
				BENZENE	9E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day					
				P-ISOPROPYLTOLUENE	2E-02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day					
				Exp. Route Total							3E-08						3E-04			
		Ingestion	2,3,7,8-TCDD Equivalent	9E-06	mg/kg	8E-14	mg/kg-day	2E+05	1/(mg/kg-day)	1E-08	6E-13	mg/kg-day	1E-09	mg/kg-day	6E-04					
			ARSENIC	4E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	2E-07	mg/kg-day	3E-04	mg/kg-day	8E-04					
			CHROMIUM	2E+02	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-03	mg/kg-day	3E-03					
			IRON	1E+04	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		9E-04	mg/kg-day	7E-01	mg/kg-day	1E-03					
			MANGANESE	3E+02	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	1E-01	mg/kg-day	1E-04					
			MERCURY	8E-01	mg/kg	6E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	3E-04	mg/kg-day	2E-04					
			VANADIUM	2E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	9E-03	mg/kg-day	1E-04					
			2-METHYLNAPHTHALENE	2E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	4E-03	mg/kg-day	3E-04					
			ACENAPHTHYLENE	4E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	8E-06					
			BENZ(A)ANTHRACENE	1E+00	mg/kg	1E-08	mg/kg-day	7E-01	1/(mg/kg-day)	8E-09	9E-08	mg/kg-day		mg/kg-day						
			BENZO(A)PYRENE	1E+00	mg/kg	1E-08	mg/kg-day	7E+00	1/(mg/kg-day)	8E-08	9E-08	mg/kg-day		mg/kg-day						
			BENZO(B)FLUORANTHENE	1E+00	mg/kg	1E-08	mg/kg-day	7E-01	1/(mg/kg-day)	9E-09	9E-08	mg/kg-day		mg/kg-day						
			BENZO(G,H,I)PERYLENE	1E+00	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day	3E-02	mg/kg-day	3E-06					
			DIBENZ(A,H)ANTHRACENE	1E-01	mg/kg	8E-10	mg/kg-day	7E+00	1/(mg/kg-day)	6E-09	6E-09	mg/kg-day		mg/kg-day						
			DIBENZOFURAN	4E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	1E-03	mg/kg-day	2E-04					
			INDENO(1,2,3-CD)PYRENE	1E+00	mg/kg	8E-09	mg/kg-day	7E-01	1/(mg/kg-day)	6E-09	6E-08	mg/kg-day		mg/kg-day						
			NAPHTHALENE	6E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	2E-02	mg/kg-day	2E-04					
			PHENANTHRENE	1E+01	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	3E-02	mg/kg-day	2E-05					
			BENZENE	9E-01	mg/kg	7E-09	mg/kg-day	6E-02	1/(mg/kg-day)	4E-10	6E-08	mg/kg-day	4E-03	mg/kg-day	1E-05					
			P-ISOPROPYLTOLUENE	2E-02	mg/kg	1E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day						
			Exp. Route Total							2E-07						7E-03				
			Exp. Point Total							2E-07						8E-03				
			Exp. Medium Total							2E-07						8E-03				
Medium Total																2E-07				8E-03
Surface Water	Surface Water	Exposure Unit 3	Dermal	CHROMIUM	1E+01	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	8E-05	mg/kg-day	2E-03				
				IRON	2E+03	ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	7E-01	mg/kg-day	1E-05				
				LEAD	2E+01	ug/l	1E-09	mg/kg-day		1/(mg/kg-day)		8E-09	mg/kg-day		mg/kg-day					
				MANGANESE	1E-01	ug/l	7E-11	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day	6E-03	mg/kg-day	1E-07				
				MERCURY	2E-01	ug/l	2E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day	2E-05	mg/kg-day	6E-05				
				VANADIUM	4E+00	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	2E-04	mg/kg-day	8E-05				
				ZINC	1E+03	ug/l	4E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-01	mg/kg-day	1E-05				

TABLE 7.6 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Ditch Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Surface Water	Surface Water	Exposure Unit 3	Dermal	2-METHYLNAPHTHALENE	9E+01	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	4E-03	mg/kg-day	1E-04	
				3&4-METHYLPHENOL	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	5E-02	mg/kg-day		
				ACENAPHTHYLENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				CARBAZOLE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				DIBENZOFURAN	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day		
				FLUORENE	2E+01	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	1E-02	
				NAPHTHALENE	8E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-02	mg/kg-day		
				PHENANTHRENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day		
				1,2,4-TRIMETHYLBENZENE	7E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				1,3,5-TRIMETHYLBENZENE	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				BENZENE	7E+01	ug/l	7E-07	mg/kg-day		1/(mg/kg-day)	4E-08	6E-06	mg/kg-day	4E-03	mg/kg-day	1E-03	
				TOLUENE	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-02	mg/kg-day		
				XYLENES, TOTAL	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day		
					Exp. Route Total								4E-08				
				Exp. Point Total								4E-08					1E-02
			Exp. Medium Total								4E-08					1E-02	
Medium Total										4E-08					1E-02		
Total of Receptor Risks Across All Media											2E-07	Total of Receptor Hazards Across All Media					2E-02

TABLE 7.7 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 4	Inhalation	ALUMINUM	1E-05	mg/m3	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	1E-03	mg/kg-day	2E-04
				ARSENIC	2E-08	mg/m3	4E-11	mg/kg-day	2E+01	1/(mg/kg-day)	6E-10	3E-10	mg/kg-day	1E-05	mg/kg-day	2E-05
				BARIUM	3E-07	mg/m3	9E-10	mg/kg-day		1/(mg/kg-day)		7E-09	mg/kg-day	1E-04	mg/kg-day	5E-05
				CHROMIUM	2E-08	mg/m3	6E-11	mg/kg-day	4E+01	1/(mg/kg-day)	2E-09	5E-10	mg/kg-day	3E-05	mg/kg-day	2E-05
				IRON	2E-05	mg/m3	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day		mg/kg-day	
				LEAD	6E-07	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day	
				MANGANESE	5E-07	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		9E-09	mg/kg-day	1E-05	mg/kg-day	6E-04
				MERCURY	1E-09	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day	9E-05	mg/kg-day	3E-07
				VANADIUM	3E-08	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	7E-11	mg/m3	2E-13	mg/kg-day	2E+00	1/(mg/kg-day)	4E-13	1E-12	mg/kg-day		mg/kg-day	
				LESS CHLORINATED PCBs	4E-12	mg/m3	9E-15	mg/kg-day	2E+00	1/(mg/kg-day)	2E-14	7E-14	mg/kg-day		mg/kg-day	
				DIELDRIN	6E-11	mg/m3	2E-13	mg/kg-day	2E+01	1/(mg/kg-day)	2E-12	1E-12	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	2E-10	mg/m3	5E-13	mg/kg-day		1/(mg/kg-day)		4E-12	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	5E-10	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	5E-10	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		9E-12	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	6E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	3E-10	mg/m3	7E-13	mg/kg-day		1/(mg/kg-day)		6E-12	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	4E-10	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		7E-12	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E-10	mg/m3	4E-13	mg/kg-day		1/(mg/kg-day)		3E-12	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	3E-10	mg/m3	8E-13	mg/kg-day		1/(mg/kg-day)		6E-12	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-10	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZENE	4E-07	mg/m3	1E-09	mg/kg-day	3E-02	1/(mg/kg-day)	3E-11	9E-09	mg/kg-day	9E-03	mg/kg-day	1E-06
				P-ISOPROPYLTOLUENE	5E-12	mg/m3	1E-14	mg/kg-day		1/(mg/kg-day)		1E-13	mg/kg-day		mg/kg-day	
			Exp. Route Total								3E-09					9E-04
		Exp. Point Total									3E-09					9E-04
	Exp. Medium Total										3E-09					9E-04
Medium Total											3E-09					9E-04
Soil	Surface Soil	Exposure Unit 4	Dermal	ALUMINUM	9E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	1E+01	mg/kg		mg/kg-day	8E-08	1/(mg/kg-day)	1E-07	6E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				BARIUM	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				CHROMIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				IRON	2E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	5E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	1E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	6E-02	mg/kg	2E-09	mg/kg-day	2E+00	1/(mg/kg-day)	3E-09	1E-08	mg/kg-day	2E-05	mg/kg-day	6E-04
				LESS CHLORINATED PCBs	3E-03	mg/kg	8E-11	mg/kg-day	2E+00	1/(mg/kg-day)	2E-10	6E-10	mg/kg-day	7E-05	mg/kg-day	9E-06
				DIELDRIN	5E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				ACENAPHTHYLENE	2E-01	mg/kg	4E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	3E-02	mg/kg-day	1E-06
				BENZ(A)ANTHRACENE	4E-01	mg/kg	1E-08	mg/kg-day	7E-01	1/(mg/kg-day)	7E-09	8E-08	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	4E-01	mg/kg	1E-08	mg/kg-day	7E+00	1/(mg/kg-day)	7E-08	8E-08	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	5E-01	mg/kg	1E-08	mg/kg-day	7E-01	1/(mg/kg-day)	9E-09	1E-07	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E-01	mg/kg	6E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	3E-02	mg/kg-day	2E-06
				BENZO(K)FLUORANTHENE	3E-01	mg/kg	8E-09	mg/kg-day	7E-02	1/(mg/kg-day)	6E-10	6E-08	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E-01	mg/kg	3E-09	mg/kg-day	7E+00	1/(mg/kg-day)	2E-08	3E-08	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	2E-01	mg/kg	6E-09	mg/kg-day	7E-01	1/(mg/kg-day)	5E-09	5E-08	mg/kg-day		mg/kg-day	
				PHENANTHRENE	5E-01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		9E-08	mg/kg-day	3E-02	mg/kg-day	3E-06
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
			Exp. Route Total								2E-07					3E-03

TABLE 7.7 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Soil	Surface Soil	Exposure Unit 4	Ingestion	ALUMINUM	9E+03	mg/kg	8E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	1E+00	mg/kg-day	6E-03	
				ARSENIC	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-04	mg/kg-day	3E-02	
				BARIUM	3E+02	mg/kg	2E-05	mg/kg-day	2E+00	1/(mg/kg-day)		2E-04	mg/kg-day	2E-01	mg/kg-day	9E-04	
				CHROMIUM	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-03	mg/kg-day	4E-03	
				IRON	2E+04	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	7E-01	mg/kg-day	2E-02	
				LEAD	5E+02	mg/kg	4E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day		
				MANGANESE	4E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	2E-03	
				MERCURY	1E+00	mg/kg	8E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	3E-04	mg/kg-day	2E-03	
				VANADIUM	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	1E-03	
				HIGHLY CHLORINATED PCBs	6E-02	mg/kg	5E-09	mg/kg-day	2E+00	1/(mg/kg-day)	1E-08	4E-08	mg/kg-day	2E-05	mg/kg-day	2E-03	
				LESS CHLORINATED PCBs	3E-03	mg/kg	2E-10	mg/kg-day	2E+00	1/(mg/kg-day)	5E-10	2E-09	mg/kg-day	7E-05	mg/kg-day	3E-05	
				DIELDRIN	5E-02	mg/kg	4E-09	mg/kg-day	2E+01	1/(mg/kg-day)	7E-08	3E-08	mg/kg-day	5E-05	mg/kg-day	6E-04	
				ACENAPHTHYLENE	2E-01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-02	mg/kg-day	4E-06	
				BENZ(A)ANTHRACENE	4E-01	mg/kg	3E-08	mg/kg-day	7E-01	1/(mg/kg-day)	2E-08	3E-07	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	4E-01	mg/kg	3E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	3E-07	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	5E-01	mg/kg	4E-08	mg/kg-day	7E-01	1/(mg/kg-day)	3E-08	3E-07	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	2E-01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	5E-06	
				BENZO(K)FLUORANTHENE	3E-01	mg/kg	3E-08	mg/kg-day	7E-02	1/(mg/kg-day)	2E-09	2E-07	mg/kg-day		mg/kg-day		
				DIBENZ(A,H)ANTHRACENE	1E-01	mg/kg	1E-08	mg/kg-day	7E+00	1/(mg/kg-day)	8E-08	9E-08	mg/kg-day		mg/kg-day		
				INDENO(1,2,3-CD)PYRENE	2E-01	mg/kg	2E-08	mg/kg-day	7E-01	1/(mg/kg-day)	2E-08	2E-07	mg/kg-day		mg/kg-day		
				PHENANTHRENE	5E-01	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-02	mg/kg-day	1E-05	
				BENZENE	1E-03	mg/kg	8E-11	mg/kg-day	6E-02	1/(mg/kg-day)	5E-12	6E-10	mg/kg-day	4E-03	mg/kg-day	2E-07	
				P-ISOPROPYLTOLUENE	4E-03	mg/kg	3E-10	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day		
							Exp. Route Total							2E-06			
					Exp. Point Total							2E-06				7E-02	
				Exp. Medium Total							2E-06				7E-02		
Medium Total											2E-06				7E-02		
Total of Receptor Risks Across All Media											2E-06	Total of Receptor Hazards Across All Media					7E-02

TABLE 7.7a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	6E-09	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	7E-12	mg/kg-day	2E+01	1/(mg/kg-day)	1E-10	5E-11	mg/kg-day	1E-05	mg/kg-day	4E-06
				CADMIUM	8E-09	mg/m3	2E-11	mg/kg-day	6E+00	1/(mg/kg-day)	1E-10	1E-10	mg/kg-day		mg/kg-day	
				CHROMIUM	5E-08	mg/m3	1E-10	mg/kg-day	4E+01	1/(mg/kg-day)	6E-09	1E-09	mg/kg-day	3E-05	mg/kg-day	4E-05
				COPPER	5E-08	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day	1E-05	mg/kg-day	2E-04
				MERCURY	8E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day	9E-05	mg/kg-day	2E-07
				VANADIUM	6E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED	4E-10	mg/m3	1E-12	mg/kg-day	2E+00	1/(mg/kg-day)	2E-12	8E-12	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		8E-11	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	7E-12	mg/kg-day		1/(mg/kg-day)		6E-11	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		8E-11	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		8E-11	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	6E-13	mg/kg-day		1/(mg/kg-day)		5E-12	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	1E-09	mg/kg-day	3E-02	1/(mg/kg-day)	3E-11	1E-08	mg/kg-day	9E-03	mg/kg-day	1E-06
			Exp. Route Total								6E-09					3E-04
		Exp. Point Total									6E-09					3E-04
	Exp. Medium Total										6E-09					3E-04
Medium Total											6E-09					3E-04
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	2E-13	mg/kg-day	2E+05	1/(mg/kg-day)	3E-08	1E-12	mg/kg-day	1E-09	mg/kg-day	1E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	4E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	3E-07	mg/kg-day	3E-04	mg/kg-day	9E-04
				CADMIUM	2E+01	mg/kg	3E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	3E-05	mg/kg-day	1E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	2E-07	mg/kg-day	2E-05	mg/kg-day	1E-02
				ACENAPHTHYLENE	2E+00	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	3E-02	mg/kg-day	1E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	2E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	1E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	2E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	3E-02	mg/kg-day	1E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	8E-08	mg/kg-day	7E-02	1/(mg/kg-day)	6E-09	6E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	2E-07	mg/kg-day	7E-03	1/(mg/kg-day)	2E-09	2E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	1E-08	mg/kg-day	7E+00	1/(mg/kg-day)	1E-07	1E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	1E-03	mg/kg-day	2E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	4E-08	mg/kg-day	7E-01	1/(mg/kg-day)	3E-08	3E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	9E-05
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								2E-06					1E-02

TABLE 7.7a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Soil	Surface Soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	3E-12	mg/kg-day	2E+05	1/(mg/kg-day)	4E-07	2E-11	mg/kg-day	1E-09	mg/kg-day	2E-02	
				ALUMINIUM	5E+03	mg/kg	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	1E+00	mg/kg-day	3E-03	
				ARSENIC	6E+00	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	4E-06	mg/kg-day	3E-04	mg/kg-day	1E-02	
				CADMIUM	2E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02	
				CHROMIUM	1E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	3E-03	mg/kg-day	3E-02	
				COPPER	1E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	4E-02	mg/kg-day	2E-03	
				IRON	1E+04	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day	7E-01	mg/kg-day	1E-02	
				MANGANESE	3E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	2E-03	
				MERCURY	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-04	mg/kg-day	4E-03	
				VANADIUM	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	9E-03	mg/kg-day	1E-03	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	8E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	6E-07	mg/kg-day	2E-05	mg/kg-day	3E-02	
				ACENAPHTHYLENE	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	4E-05	
				BENZ(A)ANTHRACENE	9E+00	mg/kg	8E-07	mg/kg-day	7E-01	1/(mg/kg-day)	6E-07	6E-06	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	7E+00	mg/kg	5E-07	mg/kg-day	7E+00	1/(mg/kg-day)	4E-06	4E-06	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	8E-07	mg/kg-day	7E-01	1/(mg/kg-day)	6E-07	6E-06	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05	
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	3E-07	mg/kg-day	7E-02	1/(mg/kg-day)	2E-08	2E-06	mg/kg-day		mg/kg-day		
				CHRYSENE	1E+01	mg/kg	8E-07	mg/kg-day	7E-03	1/(mg/kg-day)	6E-09	6E-06	mg/kg-day		mg/kg-day		
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	5E-08	mg/kg-day	7E+00	1/(mg/kg-day)	4E-07	4E-07	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03	
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	1E-06	mg/kg-day		mg/kg-day		
				PHENANTHRENE	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-02	mg/kg-day	3E-04	
				BENZENE	1E-03	mg/kg	8E-11	mg/kg-day	6E-02	1/(mg/kg-day)	4E-12	6E-10	mg/kg-day	4E-03	mg/kg-day	2E-07	
						Exp. Point Total	Exp. Route Total							7E-06			
													9E-06				1E-01
		Exp. Medium Total										9E-06				1E-01	
	Medium Total											9E-06				1E-01	
	Total of Receptor Risks Across All Media											9E-06	Total of Receptor Hazards Across All Media				1E-01

TABLE 7.8 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 5	Dermal	ALUMINUM	7E+03	mg/kg										
				ANTIMONY	2E+00	mg/kg										
				ARSENIC	2E+01	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	1E-06	mg/kg-day	6E-05	mg/kg-day	4E-03
				CHROMIUM	4E+01	mg/kg										
				IRON	2E+04	mg/kg										
				LEAD	1E+02	mg/kg										
				MANGANESE	3E+02	mg/kg										
				MERCURY	2E+00	mg/kg										
				THALLIUM	1E+00	mg/kg										
				VANADIUM	2E+01	mg/kg										
				HIGHLY CHLORINATED PCBs	6E+00	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	6E-07	2E-06	mg/kg-day	2E-05	mg/kg-day	1E-01
				ENDOSULFAN SULFATE	1E-01	mg/kg										
				ENDRIN ALDEHYDE	7E-02	mg/kg										
				3&4-METHYLPHENOL	4E-02	mg/kg	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	5E-02	mg/kg-day	2E-07
				ACENAPHTHYLENE	1E+01	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZ(A)ANTHRACENE	4E+01	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	1E-05	mg/kg-day			
				BENZO(A)PYRENE	3E+01	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	1E-05	mg/kg-day			
				BENZO(B)FLUORANTHENE	3E+01	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	1E-05	mg/kg-day			
				BENZO(G,H,I)PERYLENE	3E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	4E-04
				BENZO(K)FLUORANTHENE	4E+01	mg/kg	2E-06	mg/kg-day	7E-02	1/(mg/kg-day)	1E-07	1E-05	mg/kg-day			
				CHRYSENE	3E+01	mg/kg	2E-06	mg/kg-day	7E-03	1/(mg/kg-day)	1E-08	1E-05	mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	5E-07	mg/kg-day	7E+00	1/(mg/kg-day)	3E-06	4E-06	mg/kg-day			
				DIBENZOFURAN	6E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	1E-03	mg/kg-day	2E-03
				FLUORANTHENE	8E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	4E-02	mg/kg-day	7E-04
				INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	1E-05	mg/kg-day			
				NAPHTHALENE	7E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	2E-02	mg/kg-day	1E-04
				PHENANTHRENE	4E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	5E-04
				BENZENE	8E-03	mg/kg			6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-03	mg/kg				1/(mg/kg-day)			mg/kg-day			
				Exp. Route Total							2E-05					1E-01
			Ingestion	ALUMINUM	7E+03	mg/kg	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	1E+00	mg/kg-day	3E-03
				ANTIMONY	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	4E-04	mg/kg-day	2E-03
				ARSENIC	2E+01	mg/kg	8E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	6E-06	mg/kg-day	3E-04	mg/kg-day	2E-02
				CHROMIUM	4E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-03	mg/kg-day	5E-03
				IRON	2E+04	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		8E-03	mg/kg-day	7E-01	mg/kg-day	1E-02
				LEAD	1E+02	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day			
				MANGANESE	3E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-01	mg/kg-day	1E-03
				MERCURY	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-04	mg/kg-day	3E-03
				THALLIUM	1E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	8E-05	mg/kg-day	5E-03
				VANADIUM	2E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	1E-03
				HIGHLY CHLORINATED PCBs	6E+00	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	7E-07	3E-06	mg/kg-day	2E-05	mg/kg-day	1E-01
				ENDOSULFAN SULFATE	1E-01	mg/kg	7E-09	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	6E-03	mg/kg-day	9E-06
				ENDRIN ALDEHYDE	7E-02	mg/kg	4E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	3E-04	mg/kg-day	1E-04
				3&4-METHYLPHENOL	4E-02	mg/kg	2E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	5E-02	mg/kg-day	4E-07
				ACENAPHTHYLENE	1E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZ(A)ANTHRACENE	4E+01	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	2E-05	mg/kg-day			
				BENZO(A)PYRENE	3E+01	mg/kg	2E-06	mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	1E-05	mg/kg-day			
				BENZO(B)FLUORANTHENE	3E+01	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	1E-05	mg/kg-day			
				BENZO(G,H,I)PERYLENE	3E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	4E-04
				BENZO(K)FLUORANTHENE	4E+01	mg/kg	2E-06	mg/kg-day	7E-02	1/(mg/kg-day)	2E-07	2E-05	mg/kg-day			
				CHRYSENE	3E+01	mg/kg	2E-06	mg/kg-day	7E-03	1/(mg/kg-day)	1E-08	1E-05	mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	5E-07	mg/kg-day	7E+00	1/(mg/kg-day)	4E-06	4E-06	mg/kg-day			
				DIBENZOFURAN	6E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	1E-03	mg/kg-day	3E-03
				FLUORANTHENE	8E+01	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	4E-02	mg/kg-day	8E-04

TABLE 7.8 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Soil	Surface Soil	Exposure Unit 5	Ingestion	INDENO(1,2,3-CD)PYRENE	3E+01	mg/kg	2E-06	mg/kg-day	7E-01	1/(mg/kg-day)	1E-06	1E-05	mg/kg-day	2E-02	mg/kg-day	2E-04		
				NAPHTHALENE	7E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	6E-04		
				PHENANTHRENE	4E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	9E-07		
				BENZENE	8E-03	mg/kg	4E-10	mg/kg-day	6E-02	1/(mg/kg-day)	2E-11	3E-09	mg/kg-day	4E-03	mg/kg-day			
				P-ISOPROPYLTOLUENE	4E-03	mg/kg	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day			
				Exp. Route Total							2E-05					2E-01		
				Exp. Point Total							4E-05					3E-01		
				Exp. Medium Total							4E-05					3E-01		
Medium Total											4E-05					3E-01		
Surface Soil	Outdoor Air	Exposure Unit 5	Inhalation	ALUMINUM	1E-05	mg/m3	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	9E-04		
				ANTIMONY	3E-09	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				ARSENIC	3E-08	mg/m3	4E-10	mg/kg-day	2E+01	1/(mg/kg-day)	5E-09	3E-09	mg/kg-day	1E-05	mg/kg-day	2E-04		
				CHROMIUM	6E-08	mg/m3	8E-10	mg/kg-day	4E+01	1/(mg/kg-day)	4E-08	7E-09	mg/kg-day	3E-05	mg/kg-day	2E-04		
				IRON	3E-05	mg/m3	4E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day			
				LEAD	2E-07	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day		mg/kg-day			
				MANGANESE	5E-07	mg/m3	8E-09	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	1E-05	mg/kg-day	4E-03		
				MERCURY	4E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day	9E-05	mg/kg-day	5E-06		
				THALLIUM	2E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				VANADIUM	4E-08	mg/m3	6E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day			
				HIGHLY CHLORINATED PCBs	1E-08	mg/m3	1E-10	mg/kg-day	2E+00	1/(mg/kg-day)	3E-10	1E-09	mg/kg-day		mg/kg-day			
				ENDOSULFAN SULFATE	2E-10	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day			
				ENDRIN ALDEHYDE	1E-10	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day			
				3&4-METHYLPHENOL	7E-11	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		8E-12	mg/kg-day		mg/kg-day			
				ACENAPHTHYLENE	2E-08	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day			
				BENZ(A)ANTHRACENE	6E-08	mg/m3	9E-10	mg/kg-day		1/(mg/kg-day)		7E-09	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	6E-08	mg/m3	8E-10	mg/kg-day		1/(mg/kg-day)		7E-09	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	5E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		6E-09	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	5E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		6E-09	mg/kg-day		mg/kg-day			
				BENZO(K)FLUORANTHENE	6E-08	mg/m3	9E-10	mg/kg-day		1/(mg/kg-day)		7E-09	mg/kg-day		mg/kg-day			
				CHRYSENE	6E-08	mg/m3	8E-10	mg/kg-day		1/(mg/kg-day)		6E-09	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	2E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	1E-08	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day			
				FLUORANTHENE	1E-07	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	5E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		5E-09	mg/kg-day		mg/kg-day			
				NAPHTHALENE	1E-08	mg/m3	2E-10	mg/kg-day	1E-01	1/(mg/kg-day)	2E-11	1E-09	mg/kg-day	9E-04	mg/kg-day	2E-06		
				PHENANTHRENE	7E-08	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		8E-09	mg/kg-day		mg/kg-day			
				BENZENE	4E-06	mg/m3	5E-08	mg/kg-day	3E-02	1/(mg/kg-day)	1E-09	4E-07	mg/kg-day	9E-03	mg/kg-day	5E-05		
				P-ISOPROPYLTOLUENE	6E-12	mg/m3	9E-14	mg/kg-day		1/(mg/kg-day)		7E-13	mg/kg-day		mg/kg-day			
								Exp. Route Total							4E-08			
				Exp. Point Total							4E-08					5E-03		
				Exp. Medium Total							4E-08					5E-03		
Medium Total											4E-08					5E-03		
							Total of Receptor Risks Across All Media					4E-05	Total of Receptor Hazards Across All Media					3E-01

TABLE 7.9 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Ground Water	Potable Water	Exposure Unit 8	Ingestion	ANTHRACENE	1E+02	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-01	mg/kg-day	6E-03		
				ATRAZINE	5E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		9E-04	mg/kg-day	4E-02	mg/kg-day	3E-02		
				BENZ(A)ANTHRACENE	5E+01	ug/l	1E-04	mg/kg-day	7E-01	1/(mg/kg-day)	9E-05	9E-04	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	2E+01	ug/l	4E-05	mg/kg-day	7E+00	1/(mg/kg-day)	3E-04	3E-04	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	2E+01	ug/l	5E-05	mg/kg-day	7E-01	1/(mg/kg-day)	3E-05	4E-04	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	5E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	3E-02	mg/kg-day	3E-03		
				BENZO(K)FLUORANTHENE	2E+01	ug/l	4E-05	mg/kg-day	7E-02	1/(mg/kg-day)	3E-06	3E-04	mg/kg-day		mg/kg-day			
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	2E-05	mg/kg-day	1E-02	1/(mg/kg-day)	3E-07	2E-04	mg/kg-day	2E-02	mg/kg-day	9E-03		
				CARBAZOLE	1E+02	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day		mg/kg-day			
				CHRYSENE	4E+01	ug/l	8E-05	mg/kg-day	7E-03	1/(mg/kg-day)	6E-07	6E-04	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	6E-06	mg/kg-day	7E+00	1/(mg/kg-day)	5E-05	5E-05	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	2E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	1E-03	mg/kg-day	3E+00		
				FLUORANTHENE	2E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	4E-02	mg/kg-day	7E-02		
				FLUORENE	2E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	4E-02	mg/kg-day	7E-02		
				HEXACHLOROBUTADIENE	1E+00	ug/l	2E-06	mg/kg-day	8E-02	1/(mg/kg-day)	2E-07	2E-05	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	2E-05	mg/kg-day	7E-01	1/(mg/kg-day)	1E-05	1E-04	mg/kg-day		mg/kg-day			
				NAPHTHALENE	4E+03	ug/l	9E-03	mg/kg-day		1/(mg/kg-day)		7E-02	mg/kg-day	2E-02	mg/kg-day	3E+00		
				NITROBENZENE	3E+00	ug/l	6E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	5E-04	mg/kg-day	9E-02		
				PHENANTHRENE	4E+02	ug/l	9E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	3E-02	mg/kg-day	2E-01		
				PHENOL	2E+03	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	3E-01	mg/kg-day	1E-01		
				PYRENE	1E+02	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-02	mg/kg-day	6E-02		
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	3E-05	mg/kg-day	4E-03	1/(mg/kg-day)	1E-07	2E-04	mg/kg-day	1E-02	mg/kg-day	2E-02		
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l	7E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day		mg/kg-day			
				1,2-DICHLOROBENZENE	5E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day	9E-02	mg/kg-day	1E-01		
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l	5E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day		mg/kg-day			
				1,3-DICHLOROBENZENE	5E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day		mg/kg-day			
				1,4-DICHLOROBENZENE	5E+02	ug/l	1E-03	mg/kg-day	5E-03	1/(mg/kg-day)	6E-06	8E-03	mg/kg-day	7E-02	mg/kg-day	1E-01		
				2-HEXANONE	2E+00	ug/l	4E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	2E-01	mg/kg-day	2E-04		
				ACETONE	8E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	9E-01	mg/kg-day	1E-03		
				BENZENE	6E+03	ug/l	1E-02	mg/kg-day	6E-02	1/(mg/kg-day)	7E-04	1E-01	mg/kg-day	4E-03	mg/kg-day	2E+01		
				BROMODICHLOROMETHANE	3E+00	ug/l	7E-06	mg/kg-day	6E-02	1/(mg/kg-day)	4E-07	5E-05	mg/kg-day	2E-02	mg/kg-day	3E-03		
				CARBON DISULFIDE	1E+01	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	2E-03		
				CHLOROBENZENE	2E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	2E-02	mg/kg-day	2E-01		
				CHLOROETHANE	5E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day		mg/kg-day			
				ETHYLBENZENE	1E+02	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	1E-01	mg/kg-day	3E-02		
				ISOPROPYLBENZENE	4E+00	ug/l	9E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	1E-01	mg/kg-day	7E-04		
				METHYLENE CHLORIDE	7E-01	ug/l	2E-06	mg/kg-day	8E-03	1/(mg/kg-day)	1E-08	1E-05	mg/kg-day	6E-02	mg/kg-day	2E-04		
				P-ISOPROPYLTOLUENE	3E+00	ug/l	7E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day		mg/kg-day			
				SEC-BUTYLBENZENE	1E+01	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day			
				STYRENE	8E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	2E-01	mg/kg-day	7E-02		
				TETRACHLOROETHENE	3E-01	ug/l	7E-07	mg/kg-day	5E-01	1/(mg/kg-day)	4E-07	5E-06	mg/kg-day	1E-02	mg/kg-day	5E-04		
				TOLUENE	1E+03	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	8E-02	mg/kg-day	3E-01		
				VINYL CHLORIDE	1E+00	ug/l	2E-06	mg/kg-day	8E-01	1/(mg/kg-day)	2E-06	2E-05	mg/kg-day	3E-03	mg/kg-day	6E-03		
				XYLENES, TOTAL	1E+03	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	2E-01	mg/kg-day	9E-02		
				Exp. Route Total											1E-03			5E+01
		Exp. Point Total											1E-03			5E+01		
		Exp. Medium Total											1E-03			5E+01		
		Medium Total											1E-03			5E+01		
									Total of Receptor Risks Across All Media					1E-03	Total of Receptor Hazards Across All Media			

TABLE 7.9a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	3E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day	2E-04
				ARSENIC	3E-09	mg/m3	4E-11	mg/kg-day	2E+01	1/(mg/kg-day)	6E-10	3E-10	mg/kg-day	1E-05	mg/kg-day	2E-05
				CADMIUM	8E-09	mg/m3	1E-10	mg/kg-day	6E+00	1/(mg/kg-day)	7E-10	8E-10	mg/kg-day		mg/kg-day	
				CHROMIUM	5E-08	mg/m3	8E-10	mg/kg-day	4E+01	1/(mg/kg-day)	3E-08	6E-09	mg/kg-day	3E-05	mg/kg-day	2E-04
				COPPER	5E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		6E-09	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	8E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-05	mg/kg-day	1E-03
				MERCURY	8E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		9E-11	mg/kg-day	9E-05	mg/kg-day	1E-06
				VANADIUM	6E-09	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	6E-12	mg/kg-day	2E+00	1/(mg/kg-day)	1E-11	4E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		8E-11	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		9E-11	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	7E-09	mg/kg-day	3E-02	1/(mg/kg-day)	2E-10	6E-08	mg/kg-day	9E-03	mg/kg-day	7E-06
			Exp. Route Total								3E-08					2E-03
		Exp. Point Total									3E-08					2E-03
	Exp. Medium Total										3E-08					2E-03
Medium Total											3E-08					2E-03
Soil	Surface soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	3E-13	mg/kg-day	2E+05	1/(mg/kg-day)	5E-08	3E-12	mg/kg-day	1E-09	mg/kg-day	3E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	7E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	5E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				CADMIUM	2E+01	mg/kg	6E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	3E-05	mg/kg-day	2E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	5E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	4E-07	mg/kg-day	2E-05	mg/kg-day	2E-02
				ACENAPHTHYLENE	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	3E-02	mg/kg-day	2E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	4E-07	mg/kg-day	7E-01	1/(mg/kg-day)	3E-07	3E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	3E-07	mg/kg-day	7E+00	1/(mg/kg-day)	2E-06	2E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	3E-07	4E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	3E-02	mg/kg-day	3E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	2E-07	mg/kg-day	7E-02	1/(mg/kg-day)	1E-08	1E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	4E-07	mg/kg-day	7E-03	1/(mg/kg-day)	3E-09	3E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	3E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	2E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	1E-03	mg/kg-day	5E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	9E-08	mg/kg-day	7E-01	1/(mg/kg-day)	6E-08	7E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								3E-06					3E-02

TABLE 7.9a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	3E-07	1E-11	mg/kg-day	1E-09	mg/kg-day	1E-02
				ALUMINUM	5E+03	mg/kg	3E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E+00	mg/kg-day	2E-03
				ARSENIC	6E+00	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	3E-06	mg/kg-day	3E-04	mg/kg-day	9E-03
				CADMIUM	2E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day	1E-03	mg/kg-day	7E-03
				CHROMIUM	1E+02	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	3E-03	mg/kg-day	2E-02
				COPPER	1E+02	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	4E-02	mg/kg-day	1E-03
				IRON	1E+04	mg/kg	7E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	7E-01	mg/kg-day	8E-03
				MANGANESE	3E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-01	mg/kg-day	1E-03
				MERCURY	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	3E-04	mg/kg-day	3E-03
				VANADIUM	1E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	9E-03	mg/kg-day	7E-04
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	5E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	4E-07	mg/kg-day	2E-05	mg/kg-day	2E-02
				ACENAPHTHYLENE	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	3E-02	mg/kg-day	3E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	4E-07	4E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	4E-07	mg/kg-day	7E+00	1/(mg/kg-day)	3E-06	3E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	5E-07	mg/kg-day	7E-01	1/(mg/kg-day)	4E-07	4E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	3E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	2E-07	mg/kg-day	7E-02	1/(mg/kg-day)	1E-08	1E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	5E-07	mg/kg-day	7E-03	1/(mg/kg-day)	4E-09	4E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	3E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	3E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	1E-03	mg/kg-day	7E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	7E-08	8E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZENE	1E-03	mg/kg	5E-11	mg/kg-day	6E-02	1/(mg/kg-day)	3E-12	4E-10	mg/kg-day	4E-03	mg/kg-day	1E-07
			Exp. Route Total								5E-06					8E-02
		Exp. Point Total									8E-06					1E-01
	Exp. Medium Total										8E-06					1E-01
Medium Total											8E-06					1E-01
							Total of Receptor Risks Across All Media				8E-06	Total of Receptor Hazards Across All Media				1E-01

TABLE 7.10 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	Ingestion	2,3,7,8-TCDD Equivalent	2E-05	mg/kg	2E-10	mg/kg-day	2E+05	1/(mg/kg-day)	3E-05	2E-09	mg/kg-day	1E-09	mg/kg-day	2E+00	
				ANTIMONY	1E+00	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-04	mg/kg-day	4E-01	
				ARSENIC	8E-02	mg/kg	1E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	1E-05	mg/kg-day	3E-04	mg/kg-day	5E-02	
				CHROMIUM	6E-01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-03	mg/kg-day	3E-02	
				CYANIDE	6E+00	mg/kg	9E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-02	mg/kg-day	5E-02	
				MANGANESE	3E+00	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	1E-01	mg/kg-day	4E-03	
				MERCURY (AS METHYLMERCURY)	1E+00	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-04	mg/kg-day	2E+00	
				SELENIUM	2E+00	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	5E-03	mg/kg-day	5E-02	
				VANADIUM	6E-01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	9E-03	mg/kg-day	1E-02	
				ZINC	4E+01	mg/kg	7E-04	mg/kg-day		1/(mg/kg-day)		8E-03	mg/kg-day	3E-01	mg/kg-day	3E-02	
				HIGHLY CHLORINATED PCBs	6E-01	mg/kg	6E-06	mg/kg-day	2E+00	1/(mg/kg-day)	1E-05	7E-05	mg/kg-day	2E-05	mg/kg-day	3E+00	
				LESS CHLORINATED PCBs	5E-01	mg/kg	5E-06	mg/kg-day	2E+00	1/(mg/kg-day)	1E-05	6E-05	mg/kg-day	7E-05	mg/kg-day	8E-01	
				4,4-DDD	1E-02	mg/kg	2E-07	mg/kg-day	2E-01	1/(mg/kg-day)	5E-08	2E-06	mg/kg-day		mg/kg-day		
				4,4'-DDT	1E-02	mg/kg	1E-07	mg/kg-day	3E-01	1/(mg/kg-day)	5E-08	2E-06	mg/kg-day	5E-04	mg/kg-day	3E-03	
				ALDRIN	3E-03	mg/kg	4E-08	mg/kg-day	2E+01	1/(mg/kg-day)	7E-07	5E-07	mg/kg-day	3E-05	mg/kg-day	2E-02	
				DELTA-BHC	3E-03	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day		mg/kg-day		
				DIELDRIN	4E-03	mg/kg	6E-08	mg/kg-day	2E+01	1/(mg/kg-day)	9E-07	7E-07	mg/kg-day	5E-05	mg/kg-day	1E-02	
				HEPTACHLOR EPOXIDE	4E-03	mg/kg	6E-08	mg/kg-day	9E+00	1/(mg/kg-day)	6E-07	7E-07	mg/kg-day	1E-05	mg/kg-day	6E-02	
				BIS(2-ETHYLHEXYL)PHTHALATE	2E+00	mg/kg	4E-05	mg/kg-day	1E-02	1/(mg/kg-day)	5E-07	4E-04	mg/kg-day	2E-02	mg/kg-day	2E-02	
				HEXACHLOROENZENE	1E-02	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	2E-06	mg/kg-day	8E-04	mg/kg-day	3E-03	
				Exp. Route Total										6E-05			
		Exp. Point Total											6E-05				1E+01
		Exp. Medium Total											6E-05				1E+01
Medium Total												6E-05				1E+01	
Sediment	Surface Sediment	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	1E-04	mg/kg	9E-13	mg/kg-day	2E+05	1/(mg/kg-day)	1E-07	1E-11	mg/kg-day	1E-09	mg/kg-day	1E-02	
				ARSENIC	1E+01	mg/kg	9E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	1E-06	mg/kg-day	3E-04	mg/kg-day	3E-03	
				CADMIUM	5E+00	mg/kg	1E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	3E-05	mg/kg-day	6E-04	
				CHROMIUM	7E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day		
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day		
				LEAD	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				MANGANESE	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day		
				MERCURY	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day		
				THALLIUM	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day		
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day		
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	6E-08	4E-07	mg/kg-day	2E-05	mg/kg-day	2E-02	
				DIELDRIN	2E-02	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day		
				ENDRIN KETONE	5E-02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-04	mg/kg-day		
				HEPTACHLOR EPOXIDE	1E-02	mg/kg		mg/kg-day	9E+00	1/(mg/kg-day)			mg/kg-day	1E-05	mg/kg-day		
				2-METHYLNAPHTHALENE	4E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	4E-03	mg/kg-day	4E-03	
				ACENAPHTHYLENE	6E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	8E-05	
				BENZ(A)ANTHRACENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	1E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-05	(a)	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	7E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-02	mg/kg-day	9E-04	
				BENZO(K)FLUORANTHENE	6E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day		
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+01	mg/kg	2E-06	mg/kg-day	1E-02	1/(mg/kg-day)	2E-08	2E-05	mg/kg-day	2E-02	mg/kg-day	1E-03	
				CARBAZOLE	2E+01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day		mg/kg-day		
				CHRYSENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-07	(a)	mg/kg-day		mg/kg-day		
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	3E+01	mg/kg	8E-07	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	1E-03	mg/kg-day	9E-03	
				FLUORANTHENE	1E+02	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	4E-02	mg/kg-day	1E-03	
				HEXACHLOROENZENE	1E-01	mg/kg	4E-09	mg/kg-day	2E+00	1/(mg/kg-day)	6E-09	4E-08	mg/kg-day	8E-04	mg/kg-day	5E-05	
				INDENO(1,2,3-CD)PYRENE	5E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-06	(a)	mg/kg-day		mg/kg-day		
				NAPHTHALENE	7E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	2E-02	mg/kg-day	1E-03	
				PHENANTHRENE	2E+02	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	4E-03	
				PYRENE	4E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	3E-02	mg/kg-day	6E-03	
				1,2,4-TRICHLOROENZENE	8E-01	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day		
				1,4-DICHLOROENZENE	5E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day		

TABLE 7.10 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Sediment	Surface Sediment	Exposure Unit 6	Dermal	BENZENE	8E+00	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day			
				CHLOROBENZENE	5E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-02	mg/kg-day			
				METHYLENE CHLORIDE	7E-01	mg/kg		mg/kg-day	8E-03	1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day			
				TOLUENE	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-02	mg/kg-day			
				XYLENES, TOTAL	7E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day			
			Exp. Route Total								3E-04					6E-02		
			Ingestion	2,3,7,8-TCDD Equivalent	1E-04	mg/kg	3E-12	mg/kg-day	2E+05	1/(mg/kg-day)	4E-07	3E-11	mg/kg-day	1E-09	mg/kg-day	3E-02		
				ARSENIC	1E+01	mg/kg	3E-07	mg/kg-day	2E+00	1/(mg/kg-day)	4E-07	3E-06	mg/kg-day	3E-04	mg/kg-day	1E-02		
				CADMIUM	5E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-05	mg/kg-day	5E-02		
				CHROMIUM	7E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	8E-05	mg/kg-day	3E-01		
				IRON	1E+04	mg/kg	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	7E-01	mg/kg-day	5E-03		
				LEAD	1E+02	mg/kg	4E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day		mg/kg-day			
				MANGANESE	2E+02	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	6E-03	mg/kg-day	1E-02		
				MERCURY	8E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	2E-05	mg/kg-day	1E-01		
				THALLIUM	8E-01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	8E-05	mg/kg-day	3E-03		
				VANADIUM	1E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	2E-04	mg/kg-day	2E-02		
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	2E-07	mg/kg-day	2E-05	mg/kg-day	1E-02		
				DIELDRIN	2E-02	mg/kg	5E-10	mg/kg-day	2E+01	1/(mg/kg-day)	8E-09	6E-09	mg/kg-day	5E-05	mg/kg-day	1E-04		
				ENDRIN KETONE	5E-02	mg/kg	1E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	3E-04	mg/kg-day	5E-05		
				HEPTACHLOR EPOXIDE	1E-02	mg/kg	2E-10	mg/kg-day	9E+00	1/(mg/kg-day)	2E-09	3E-09	mg/kg-day	1E-05	mg/kg-day	2E-04		
				2-METHYLNAPHTHALENE	4E+01	mg/kg	9E-07	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03		
				ACENAPHTHYLENE	6E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	5E-05		
				BENZ(A)ANTHRACENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-04	(a)	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	1E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-04	(a)	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	7E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	6E-04		
				BENZO(K)FLUORANTHENE	6E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-06	(a)	mg/kg-day		mg/kg-day			
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+01	mg/kg	2E-06	mg/kg-day	1E-02	1/(mg/kg-day)	2E-08	2E-05	mg/kg-day	2E-02	mg/kg-day	9E-04		
				CARBAZOLE	2E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day		mg/kg-day			
				CHRYSENE	2E+02	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	8E-05	(a)	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	3E+01	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	1E-03	mg/kg-day	8E-03		
				FLUORANTHENE	1E+02	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	4E-02	mg/kg-day	1E-03		
				HEXACHLOROBENZENE	1E-01	mg/kg	3E-09	mg/kg-day	2E+00	1/(mg/kg-day)	5E-09	4E-08	mg/kg-day	8E-04	mg/kg-day	5E-05		
				INDENO(1,2,3-CD)PYRENE	5E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-05	(a)	mg/kg-day		mg/kg-day			
				NAPHTHALENE	7E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-02	mg/kg-day	1E-03		
				PHENANTHRENE	2E+02	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	3E-02	mg/kg-day	2E-03		
				PYRENE	4E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	4E-03		
				1,2,4-TRICHLOROBENZENE	8E-01	mg/kg	2E-08	mg/kg-day	4E-03	1/(mg/kg-day)	7E-11	2E-07	mg/kg-day	1E-02	mg/kg-day	2E-05		
				1,4-DICHLOROBENZENE	5E+01	mg/kg	1E-06	mg/kg-day	5E-03	1/(mg/kg-day)	7E-09	2E-05	mg/kg-day	7E-02	mg/kg-day	2E-04		
				BENZENE	8E+00	mg/kg	2E-07	mg/kg-day	6E-02	1/(mg/kg-day)	1E-08	2E-06	mg/kg-day	4E-03	mg/kg-day	6E-04		
				CHLOROBENZENE	5E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	2E-02	mg/kg-day	7E-04		
				METHYLENE CHLORIDE	7E-01	mg/kg	2E-08	mg/kg-day	8E-03	1/(mg/kg-day)	1E-10	2E-07	mg/kg-day	6E-02	mg/kg-day	4E-06		
				TOLUENE	2E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	8E-02	mg/kg-day	8E-05		
				XYLENES, TOTAL	7E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-01	mg/kg-day	1E-04		
				Exp. Route Total								1E-03					5E-01	
				Exp. Point Total								1E-03					6E-01	
				Exp. Medium Total								1E-03					6E-01	
				Medium Total										1E-03				6E-01
				Soil	Surface Soil	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	4E-12	mg/kg-day	2E+05	1/(mg/kg-day)	7E-07	5E-11	mg/kg-day	1E-09
ALUMINUM	7E+03	mg/kg							mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day		
ARSENIC	8E+00	mg/kg	7E-08					mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	8E-07	mg/kg-day	3E-04	mg/kg-day	3E-03		
BARIUM	4E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day			
CADMIUM	4E+01	mg/kg	1E-08					mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-05	mg/kg-day	5E-03		
CHROMIUM	1E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
COPPER	2E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day			
IRON	1E+04	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day			
LEAD	7E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			

TABLE 7.10 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations							
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient			
							Value	Units	Value	Units		Value	Units	Value	Units				
Soil	Surface Soil	Exposure Unit 6	Dermal	MANGANESE	3E+02	mg/kg				1/(mg/kg-day)					mg/kg-day	6E-03	mg/kg-day		
				MERCURY	1E+01	mg/kg					1/(mg/kg-day)					mg/kg-day	2E-05	mg/kg-day	
				SILVER	2E+01	mg/kg					1/(mg/kg-day)					mg/kg-day	2E-04	mg/kg-day	
				THALLIUM	8E-01	mg/kg					1/(mg/kg-day)					mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg					1/(mg/kg-day)					mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	6E-08		mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	7E-07	mg/kg-day	2E-05	mg/kg-day	4E-02		
				LESS CHLORINATED PCBs	7E-01	mg/kg	3E-08		mg/kg-day	2E+00	1/(mg/kg-day)	6E-08	3E-07	mg/kg-day	7E-05	mg/kg-day	5E-03		
				DIELDRIN	1E-01	mg/kg			mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day			
				2-METHYLNAPHTHALENE	1E+01	mg/kg	5E-07		mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	4E-03	mg/kg-day	1E-03		
				ACENAPHTHYLENE	4E+00	mg/kg	1E-07		mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	6E-05		
				BENZ(A)ANTHRACENE	8E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	9E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	2E-07		mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	7E-05		
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	1E-07	(a)	mg/kg-day		mg/kg-day			
				CHRYSENE	8E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	1E-08	(a)	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	3E-06	(a)	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	4E+00	mg/kg	1E-07		mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03		
				HEXACHLOROBENZENE	1E+00	mg/kg	3E-08		mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	3E-07	mg/kg-day	8E-04	mg/kg-day	4E-04		
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	8E-07	(a)	mg/kg-day		mg/kg-day			
				NAPHTHALENE	2E+01	mg/kg	9E-07		mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	2E-02	mg/kg-day	5E-04		
				PHENANTHRENE	2E+01	mg/kg	7E-07		mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-02	mg/kg-day	3E-04		
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg			mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg			mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day			
				1,2-DICHLOROBENZENE	8E+00	mg/kg			mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day			
				1,4-DICHLOROBENZENE	3E+01	mg/kg			mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day			
				BENZENE	5E-01	mg/kg			mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day			
				P-ISOPROPYLTOLUENE	4E-01	mg/kg			mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				DODECANE	8E+02	mg/kg			mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				Exp. Route Total									2E-05						1E-01
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	1E-11		mg/kg-day	2E+05	1/(mg/kg-day)	2E-06	2E-10	mg/kg-day	1E-09	mg/kg-day	2E-01		
				ALUMINUM	7E+03	mg/kg	2E-04		mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E+00	mg/kg-day	2E-03		
				ARSENIC	8E+00	mg/kg	2E-07		mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	2E-06	mg/kg-day	3E-04	mg/kg-day	8E-03		
				BARIUM	4E+02	mg/kg	9E-06		mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	2E-01	mg/kg-day	5E-04		
				CADMIUM	4E+01	mg/kg	9E-07		mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02		
				CHROMIUM	1E+02	mg/kg	3E-06		mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-03	mg/kg-day	1E-02		
				COPPER	2E+02	mg/kg	6E-06		mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	4E-02	mg/kg-day	2E-03		
				IRON	1E+04	mg/kg	3E-04		mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	7E-01	mg/kg-day	5E-03		
				LEAD	7E+02	mg/kg	2E-05		mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day			
				MANGANESE	3E+02	mg/kg	7E-06		mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	1E-01	mg/kg-day	6E-04		
				MERCURY	1E+01	mg/kg	3E-07		mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-04	mg/kg-day	1E-02		
				SILVER	2E+01	mg/kg	4E-07		mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	5E-03	mg/kg-day	9E-04		
				THALLIUM	8E-01	mg/kg	2E-08		mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	8E-05	mg/kg-day	3E-03		
				VANADIUM	2E+01	mg/kg	5E-07		mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	9E-03	mg/kg-day	7E-04		
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	4E-08		mg/kg-day	2E+00	1/(mg/kg-day)	8E-08	4E-07	mg/kg-day	2E-05	mg/kg-day	2E-02		
				LESS CHLORINATED PCBs	7E-01	mg/kg	2E-08		mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	2E-07	mg/kg-day	7E-05	mg/kg-day	3E-03		
				DIELDRIN	1E-01	mg/kg	3E-09		mg/kg-day	2E+01	1/(mg/kg-day)	5E-08	3E-08	mg/kg-day	5E-05	mg/kg-day	7E-04		
				2-METHYLNAPHTHALENE	1E+01	mg/kg	3E-07		mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	4E-03	mg/kg-day	9E-04		
				ACENAPHTHYLENE	4E+00	mg/kg	1E-07		mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	4E-05		
				BENZ(A)ANTHRACENE	8E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	9E-07	(a)	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	9E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	1E-05	(a)	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	8E-07	(a)	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	1E-07		mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	5E-05		
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	7E-08	(a)	mg/kg-day		mg/kg-day			
				CHRYSENE	8E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	1E-08	(a)	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	(a)		mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	4E+00	mg/kg	1E-07		mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03		

TABLE 7.10 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Ingestion	HEXACHLOROBENZENE	1E+00	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	3E-07	mg/kg-day	8E-04	mg/kg-day	3E-04
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-07	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		7E-06	mg/kg-day	2E-02	mg/kg-day	3E-04
				PHENANTHRENE	2E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg	1E-07	mg/kg-day	4E-03	1/(mg/kg-day)	4E-10	1E-06	mg/kg-day	1E-02	mg/kg-day	1E-04
				1,2-DICHLOROBENZENE	8E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	9E-02	mg/kg-day	3E-05
				1,4-DICHLOROBENZENE	3E+01	mg/kg	8E-07	mg/kg-day	5E-03	1/(mg/kg-day)	4E-09	9E-06	mg/kg-day	7E-02	mg/kg-day	1E-04
				BENZENE	5E-01	mg/kg	1E-08	mg/kg-day	6E-02	1/(mg/kg-day)	7E-10	2E-07	mg/kg-day	4E-03	mg/kg-day	4E-05
				P-ISOPROPYLTOLUENE	4E-01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day	
				Exp. Route Total							2E-05					2E-01
		Exp. Point Total									4E-05					3E-01
	Exp. Medium Total										4E-05					3E-01
Medium Total											4E-05					3E-01
Surface Soil	Outdoor Air	Exposure Unit 6	Inhalation	2,3,7,8-TCDD Equivalent	1E-07	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-03	mg/kg-day	1E-05
				ARSENIC	2E-09	mg/m3	2E-12	mg/kg-day	2E+01	1/(mg/kg-day)	3E-11	2E-11	mg/kg-day	1E-05	mg/kg-day	2E-06
				BARIIUM	9E-08	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day	1E-04	mg/kg-day	8E-06
				CADMIUM	9E-09	mg/m3	9E-12	mg/kg-day	6E+00	1/(mg/kg-day)	6E-11	1E-10	mg/kg-day		mg/kg-day	
				CHROMIUM	3E-08	mg/m3	3E-11	mg/kg-day	4E+01	1/(mg/kg-day)	1E-09	3E-10	mg/kg-day	3E-05	mg/kg-day	1E-05
				COPPER	6E-08	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				IRON	3E-06	mg/m3	3E-09	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day	
				LEAD	2E-07	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				MANGANESE	8E-08	mg/m3	8E-11	mg/kg-day		1/(mg/kg-day)		9E-10	mg/kg-day	1E-05	mg/kg-day	6E-05
				MERCURY	3E-09	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day	9E-05	mg/kg-day	4E-07
				SILVER	4E-09	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				THALLIUM	2E-10	mg/m3	2E-13	mg/kg-day		1/(mg/kg-day)		2E-12	mg/kg-day		mg/kg-day	
				VANADIUM	5E-09	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		6E-11	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	4E-13	mg/kg-day	2E+00	1/(mg/kg-day)	8E-13	5E-12	mg/kg-day		mg/kg-day	
				LESS CHLORINATED PCBs	2E-10	mg/m3	2E-13	mg/kg-day	2E+00	1/(mg/kg-day)	4E-13	2E-12	mg/kg-day		mg/kg-day	
				DIELDRIN	3E-11	mg/m3	3E-14	mg/kg-day	2E+01	1/(mg/kg-day)	5E-13	3E-13	mg/kg-day		mg/kg-day	
				2-METHYLNAPHTHALENE	3E-09	mg/m3	3E-12	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	1E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	4E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	1E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				HEXACHLOROBENZENE	2E-10	mg/m3	2E-13	mg/kg-day	2E+00	1/(mg/kg-day)	4E-13	3E-12	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)	8E-13	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	6E-09	mg/m3	6E-12	mg/kg-day	1E-01	1/(mg/kg-day)		7E-11	mg/kg-day	9E-04	mg/kg-day	8E-08
				PHENANTHRENE	5E-09	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		6E-11	mg/kg-day		mg/kg-day	
				1,2,3-TRICHLOROBENZENE	1E-04	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E-04	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day		mg/kg-day	
				1,2-DICHLOROBENZENE	9E-04	mg/m3	9E-07	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day		mg/kg-day	3E-04
				1,4-DICHLOROBENZENE	4E-03	mg/m3	4E-06	mg/kg-day	4E-02	1/(mg/kg-day)	1E-07	4E-05	mg/kg-day	2E-01	mg/kg-day	2E-04
				BENZENE	3E-04	mg/m3	3E-07	mg/kg-day	3E-02	1/(mg/kg-day)	8E-09	3E-06	mg/kg-day	9E-03	mg/kg-day	4E-04
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	2E-07	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				Exp. Route Total							2E-07					9E-04
		Exp. Point Total									2E-07					9E-04
	Exp. Medium Total										2E-07					9E-04
Medium Total											2E-07					9E-04

TABLE 7.10 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations								
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RfC		Hazard Quotient				
							Value	Units	Value	Units		Value	Units	Value	Units					
Surface Water	Surface Water	Exposure Unit 6	Dermal	ANTIMONY	2E+00	ug/l	6E-09	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day	6E-05	mg/kg-day	1E-03				
				ARSENIC	2E+00	ug/l	5E-09	mg/kg-day	2E+00	1/(mg/kg-day)	8E-09	6E-08	mg/kg-day	3E-04	mg/kg-day	2E-04				
				CHROMIUM	5E+00	ug/l	3E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	8E-05	mg/kg-day	5E-03				
				IRON	5E+03	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	7E-01	mg/kg-day	2E-04				
				LEAD	8E+00	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day					
				MERCURY	1E-01	ug/l	3E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day	2E-05	mg/kg-day	2E-04				
				THALLIUM	4E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	8E-05	mg/kg-day	2E-03				
				2,4-DIMETHYLPHENOL	1E+02	ug/l	6E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	2E-02	mg/kg-day	3E-03				
				2-METHYLNAPHTHALENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day					
				3&4-METHYLPHENOL	2E+02	ug/l	6E-06	mg/kg-day		1/(mg/kg-day)	7E-05	mg/kg-day	5E-02	mg/kg-day	1E-03					
				ACENAPHTHENE	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)		mg/kg-day	6E-02	mg/kg-day						
				ACENAPHTHYLENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)		mg/kg-day	3E-02	mg/kg-day						
				BENZ(A)ANTHRACENE	4E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-05	(a)	mg/kg-day		mg/kg-day					
				BENZO(A)PYRENE	2E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-04	(a)	mg/kg-day		mg/kg-day					
				BENZO(B)FLUORANTHENE	3E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-05	(a)	mg/kg-day		mg/kg-day					
				BIS(2-ETHYLHEXYL)PHTHALATE	8E+00	ug/l	3E-06	mg/kg-day	1E-02	1/(mg/kg-day)	5E-08	4E-05	mg/kg-day	2E-02	mg/kg-day	2E-03				
				CARBAZOLE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day					
				CHRYSENE	4E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-07	(a)	mg/kg-day		mg/kg-day					
				DIBENZOFURAN	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day					
				FLUORENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day					
				NAPHTHALENE	2E+03	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)	4E-03	mg/kg-day	2E-02	mg/kg-day	2E-01					
				PHENANTHRENE	3E+01	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)	3E-04	mg/kg-day	3E-02	mg/kg-day	9E-03					
				PYRENE	8E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day					
				1,4-DICHLOROBENZENE	8E+00	ug/l	2E-06	mg/kg-day	5E-03	1/(mg/kg-day)	9E-09	2E-05	mg/kg-day	7E-02	mg/kg-day	3E-04				
				BENZENE	7E+01	ug/l	4E-06	mg/kg-day	6E-02	1/(mg/kg-day)	2E-07	4E-05	mg/kg-day	4E-03	mg/kg-day	1E-02				
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day					
				TOLUENE	4E+02	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day	8E-02	mg/kg-day	6E-03				
				XYLENES, TOTAL	5E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day					
								Exp. Route Total												2E-01
								Exp. Point Total												2E-01
								Exp. Medium Total												2E-01
				Medium Total											8E-04					2E-01
															8E-04					2E-01
															8E-04					2E-01
															2E-03					1E+01

Notes:
(a) See Table 7.10 CT Supplement A for the intake and toxicity values for COPCs with an MMOA

TABLE 7.10 CT Supplement A
CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Recreator
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	EU-6	Ingestion	Benz(a)anthracene	7.5E+00	mg/kg	9.3E-08	1.2E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	9.E-07
				Benzo(a)pyrene	9.0E+00	mg/kg	1.1E-07	1.4E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-05
				Benzo(b)fluoranthene	6.6E+00	mg/kg	8.2E-08	1.0E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	8.E-07
				Benzo(k)fluoranthene	5.7E+00	mg/kg	7.0E-08	8.9E-08	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	7.E-08
				Chrysene	8.0E+00	mg/kg	9.9E-08	1.3E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	1.E-08
				Dibenz(a,h)anthracene	1.6E+00	mg/kg	2.0E-08	2.6E-08	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-06
				Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	5.7E-08	7.1E-08	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	6.E-07
			Dermal	Benz(a)anthracene	7.5E+00	mg/kg	1.3E-07	1.8E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-06
				Benzo(a)pyrene	9.0E+00	mg/kg	1.5E-07	2.1E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-05
				Benzo(b)fluoranthene	6.6E+00	mg/kg	1.1E-07	1.5E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-06
				Benzo(k)fluoranthene	5.7E+00	mg/kg	9.5E-08	1.3E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	1.E-07
				Chrysene	8.0E+00	mg/kg	1.3E-07	1.9E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	1.E-08
				Dibenz(a,h)anthracene	1.6E+00	mg/kg	2.8E-08	3.8E-08	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	3.E-06
				Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	7.7E-08	1.1E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	8.E-07
	Fugitive Dust	EU-6	Inhalation	Benz(a)anthracene	1.9E-09	mg/m ³	2.2E-13	3.9E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	2.3E-09	mg/m ³	2.6E-13	4.6E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	1.7E-09	mg/m ³	1.9E-13	3.4E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	1.4E-09	mg/m ³	1.6E-13	2.9E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.0E-09	mg/m ³	2.3E-13	4.1E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	4.1E-10	mg/m ³	4.8E-14	8.4E-14	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
Sediment	Surface Sediment	EU-6	Ingestion	Indeno(1,2,3-cd)pyrene	1.2E-09	mg/m ³	1.3E-13	2.3E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benz(a)anthracene	1.5E+02	mg/kg	1.9E-06	2.4E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-05
				Benzo(a)pyrene	1.1E+02	mg/kg	1.3E-06	1.7E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-04
				Benzo(b)fluoranthene	2.4E+02	mg/kg	3.0E-06	3.7E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
				Benzo(k)fluoranthene	5.9E+01	mg/kg	7.3E-07	9.1E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	7.E-07
				Chrysene	2.2E+02	mg/kg	2.7E-06	3.4E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	3.E-07
				Dibenz(a,h)anthracene	1.3E+01	mg/kg	1.6E-07	2.0E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-05
			Dermal	Indeno(1,2,3-cd)pyrene	5.4E+01	mg/kg	6.7E-07	8.5E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	7.E-06
				Benz(a)anthracene	1.5E+02	mg/kg	2.5E-06	3.6E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
				Benzo(a)pyrene	1.1E+02	mg/kg	1.8E-06	2.5E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-04
				Benzo(b)fluoranthene	2.4E+02	mg/kg	4.0E-06	5.6E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	4.E-05
				Benzo(k)fluoranthene	5.9E+01	mg/kg	9.8E-07	1.4E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	1.E-06
				Chrysene	2.2E+02	mg/kg	3.6E-06	5.1E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	4.E-07
				Dibenz(a,h)anthracene	1.3E+01	mg/kg	2.2E-07	3.0E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-05
				Indeno(1,2,3-cd)pyrene	5.4E+01	mg/kg	9.1E-07	1.3E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	9.E-06
				Water	Surface Water	EU-6	Dermal	Benz(a)anthracene	4.0E+00	µg/L	6.8E-06	9.4E-06	mg/kg/day
Benzo(a)pyrene	2.0E+00	µg/L	5.8E-06					8.1E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	6.E-04
Benzo(b)fluoranthene	3.0E+00	µg/L	8.8E-06					1.2E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	9.E-05
Chrysene	2.9E+00	µg/L	4.8E-06					6.8E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	5.E-07

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include

TABLE 7.10a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	3E-13	mg/kg-day	2E+05	1/(mg/kg-day)	4E-08	3E-12	mg/kg-day	1E-09	mg/kg-day	3E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	5E-08	mg/kg-day	2E+00	1/(mg/kg-day)	8E-08	6E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				CADMIUM	2E+01	mg/kg	5E-09	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	3E-05	mg/kg-day	2E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	4E-08	mg/kg-day	2E+00	1/(mg/kg-day)	7E-08	4E-07	mg/kg-day	2E-05	mg/kg-day	2E-02
				ACENAPHTHYLENE	2E+00	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	3E-02	mg/kg-day	3E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	8E-08	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	3E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-08	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	1E-03	mg/kg-day	6E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-07	(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				Exp. Route Total							2E-05					3E-02
			Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	8E-13	mg/kg-day	2E+05	1/(mg/kg-day)	1E-07	9E-12	mg/kg-day	1E-09	mg/kg-day	9E-03
				ALUMINUM	5E+03	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E+00	mg/kg-day	2E-03
				ARSENIC	6E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	2E-06	mg/kg-day	3E-04	mg/kg-day	6E-03
				CADMIUM	2E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	1E-03	mg/kg-day	5E-03
				CHROMIUM	1E+02	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	3E-03	mg/kg-day	1E-02
				COPPER	1E+02	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	4E-02	mg/kg-day	9E-04
				IRON	1E+04	mg/kg	3E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	7E-01	mg/kg-day	6E-03
				MANGANESE	3E+02	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-01	mg/kg-day	7E-04
				MERCURY	2E+00	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				VANADIUM	1E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	9E-03	mg/kg-day	5E-04
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	3E-07	mg/kg-day	2E-05	mg/kg-day	1E-02
				ACENAPHTHYLENE	2E+00	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	3E-02	mg/kg-day	2E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	8E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	3E-02	mg/kg-day	2E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-08	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-07	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	1E-03	mg/kg-day	5E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-07	(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZENE	1E-03	mg/kg	2E-11	mg/kg-day	6E-02	1/(mg/kg-day)	1E-12	3E-10	mg/kg-day	4E-03	mg/kg-day	7E-08
				Exp. Route Total							1E-05					6E-02
				Exp. Point Total							3E-05					9E-02
				Exp. Medium Total							3E-05					9E-02
				Medium Total							3E-05					9E-02

TABLE 7.10a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations								
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient				
							Value	Units	Value	Units		Value	Units	Value	Units					
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day					
				ALUMINUM	2E-06	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day	2E-05				
				ARSENIC	3E-09	mg/m3	3E-12	mg/kg-day	2E+01	1/(mg/kg-day)	4E-11	3E-11	mg/kg-day	1E-03	mg/kg-day	2E-06				
				CADMIUM	8E-09	mg/m3	8E-12	mg/kg-day	6E+00	1/(mg/kg-day)	5E-11	9E-11	mg/kg-day		mg/kg-day					
				CHROMIUM	5E-08	mg/m3	5E-11	mg/kg-day	4E+01	1/(mg/kg-day)	2E-09	6E-10	mg/kg-day	3E-05	mg/kg-day	2E-05				
				COPPER	5E-08	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day		mg/kg-day					
				IRON	6E-06	mg/m3	6E-09	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day		mg/kg-day					
				MANGANESE	1E-07	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	1E-05	mg/kg-day	1E-04				
				MERCURY	8E-10	mg/m3	8E-13	mg/kg-day		1/(mg/kg-day)		9E-12	mg/kg-day	9E-05	mg/kg-day	1E-07				
				VANADIUM	6E-09	mg/m3	6E-12	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day					
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	4E-13	mg/kg-day	2E+00	1/(mg/kg-day)	8E-13	5E-12	mg/kg-day		mg/kg-day					
				ACENAPHTHYLENE	9E-10	mg/m3	9E-13	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day					
				BENZ(A)ANTHRACENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day					
				BENZO(A)PYRENE	3E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day					
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day					
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day					
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day					
				CHRYSENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day					
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day					
				DIBENZOFURAN	7E-10	mg/m3	7E-13	mg/kg-day		1/(mg/kg-day)		9E-12	mg/kg-day		mg/kg-day					
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day					
				PHENANTHRENE	6E-09	mg/m3	6E-12	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day					
				BENZENE	5E-07	mg/m3	5E-10	mg/kg-day	3E-02	1/(mg/kg-day)	1E-11	6E-09	mg/kg-day	9E-03	mg/kg-day	7E-07				
							Exp. Route Total								2E-09				2E-04	
				Exp. Point Total								2E-09				2E-04				
			Exp. Medium Total										2E-09				2E-04			
		Medium Total										2E-09				2E-04				
									Total of Receptor Risks Across All Media					3E-05	Total of Receptor Hazards Across All Media					9E-02

Notes:
(a) See Table 7.10a CT Supplement A for the intake and toxicity values for COPCs with an MMOA

TABLE 7.10a CT Supplement A
CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Recreator
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	EU-9	Ingestion	Benz(a)anthracene	9.3E+00	mg/kg	1.2E-07	1.5E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-06
				Benzo(a)pyrene	6.6E+00	mg/kg	8.2E-08	1.0E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	8.E-06
				Benzo(b)fluoranthene	9.6E+00	mg/kg	1.2E-07	1.5E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-06
				Benzo(k)fluoranthene	3.3E+00	mg/kg	4.0E-08	5.1E-08	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	4.E-08
				Chrysene	9.5E+00	mg/kg	1.2E-07	1.5E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	1.E-08
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	7.3E-09	9.1E-09	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	7.E-07
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	2.2E-08	2.8E-08	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-07
			Dermal	Benz(a)anthracene	9.3E+00	mg/kg	1.6E-07	2.2E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-06
				Benzo(a)pyrene	6.6E+00	mg/kg	1.1E-07	1.6E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-05
				Benzo(b)fluoranthene	9.6E+00	mg/kg	1.6E-07	2.2E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-06
				Benzo(k)fluoranthene	3.3E+00	mg/kg	5.5E-08	7.6E-08	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	6.E-08
				Chrysene	9.5E+00	mg/kg	1.6E-07	2.2E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	2.E-08
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	9.8E-09	1.4E-08	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-06
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	3.0E-08	4.2E-08	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-07
	Fugitive Dust	EU-9	Inhalation	Benz(a)anthracene	2.7E-08	mg/m ³	3.1E-12	5.5E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	1.9E-08	mg/m ³	2.2E-12	3.9E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	2.8E-08	mg/m ³	3.2E-12	5.6E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	9.4E-09	mg/m ³	1.1E-12	1.9E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.7E-08	mg/m ³	3.2E-12	5.6E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	1.7E-09	mg/m ³	1.9E-13	3.4E-13	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	5.2E-09	mg/m ³	6.0E-13	1.1E-12	mg/kg/day	NA	NA	1/(mg/kg-day)	NA

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include

TABLE 7.11 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations														
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient										
							Value	Units	Value	Units		Value	Units	Value	Units											
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	Ingestion	2,3,7,8-TCDD Equivalent	2E-05	mg/kg	2E-10	mg/kg-day	2E+05	1/(mg/kg-day)	3E-05	2E-09	mg/kg-day	1E-09	mg/kg-day	2E+00										
				ANTIMONY	1E+00	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	4E-04	mg/kg-day	3E-01										
				ARSENIC	8E-02	mg/kg	1E-06	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	9E-06	mg/kg-day	3E-04	mg/kg-day	3E-02										
				CHROMIUM	6E-01	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	3E-03	mg/kg-day	2E-02										
				CYANIDE	6E+00	mg/kg	8E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day	2E-02	mg/kg-day	3E-02										
				MANGANESE	3E+00	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	1E-01	mg/kg-day	3E-03										
				MERCURY (AS METHYLMERCURY)	1E+00	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-04	mg/kg-day	1E+00										
				SELENIUM	2E+00	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	5E-03	mg/kg-day	3E-02										
				VANADIUM	6E-01	mg/kg	9E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	9E-03	mg/kg-day	8E-03										
				ZINC	4E+01	mg/kg	6E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	3E-01	mg/kg-day	2E-02										
				HIGHLY CHLORINATED PCBs	6E-01	mg/kg	6E-06	mg/kg-day	2E+00	1/(mg/kg-day)	1E-05	4E-05	mg/kg-day	2E-05	mg/kg-day	2E+00										
				LESS CHLORINATED PCBs	5E-01	mg/kg	5E-06	mg/kg-day	2E+00	1/(mg/kg-day)	9E-06	4E-05	mg/kg-day	7E-05	mg/kg-day	5E-01										
				4,4-DDD	1E-02	mg/kg	2E-07	mg/kg-day	2E-01	1/(mg/kg-day)	5E-08	1E-06	mg/kg-day		mg/kg-day											
				4,4'-DDT	1E-02	mg/kg	1E-07	mg/kg-day	3E-01	1/(mg/kg-day)	5E-08	1E-06	mg/kg-day	5E-04	mg/kg-day	2E-03										
				ALDRIN	3E-03	mg/kg	4E-08	mg/kg-day	2E+01	1/(mg/kg-day)	6E-07	3E-07	mg/kg-day	3E-05	mg/kg-day	1E-02										
				DELTA-BHC	3E-03	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day											
				DIELDRIN	4E-03	mg/kg	6E-08	mg/kg-day	2E+01	1/(mg/kg-day)	9E-07	4E-07	mg/kg-day	5E-05	mg/kg-day	9E-03										
				HEPTACHLOR EPOXIDE	4E-03	mg/kg	6E-08	mg/kg-day	9E+00	1/(mg/kg-day)	5E-07	5E-07	mg/kg-day	1E-05	mg/kg-day	4E-02										
				BIS(2-ETHYLHEXYL)PHTHALATE	2E+00	mg/kg	3E-05	mg/kg-day	1E-02	1/(mg/kg-day)	5E-07	3E-04	mg/kg-day	2E-02	mg/kg-day	1E-02										
				HEXACHLOROBENZENE	1E-02	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	1E-06	mg/kg-day	8E-04	mg/kg-day	2E-03										
				Exp. Route Total											6E-05				6E+00							
				Exp. Point Total											6E-05				6E+00							
				Exp. Medium Total											6E-05				6E+00							
				Medium Total																	6E-05				6E+00	
				Sediment	Surface Sediment	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	1E-04	mg/kg	5E-13	mg/kg-day	2E+05	1/(mg/kg-day)	7E-08	4E-12	mg/kg-day	1E-09	mg/kg-day	4E-03						
								ARSENIC	1E+01	mg/kg	4E-08	mg/kg-day	2E+00	1/(mg/kg-day)	6E-08	3E-07	mg/kg-day	3E-04	mg/kg-day	1E-03						
								CADMIUM	5E+00	mg/kg	6E-10	mg/kg-day		1/(mg/kg-day)		5E-09	mg/kg-day	3E-05	mg/kg-day	2E-04						
CHROMIUM	7E+01	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day											
IRON	1E+04	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day											
LEAD	1E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day											
MANGANESE	2E+02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day											
MERCURY	8E+00	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day											
THALLIUM	8E-01	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day											
VANADIUM	1E+01	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day											
HIGHLY CHLORINATED PCBs	8E-01	mg/kg	1E-08					mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	1E-07	mg/kg-day	2E-05	mg/kg-day	6E-03										
DIELDRIN	2E-02	mg/kg						mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day											
ENDRIN KETONE	5E-02	mg/kg						mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-04	mg/kg-day											
HEPTACHLOR EPOXIDE	1E-02	mg/kg						mg/kg-day	9E+00	1/(mg/kg-day)			mg/kg-day	1E-05	mg/kg-day											
2-METHYLNAPHTHALENE	4E+01	mg/kg	6E-07					mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	4E-03	mg/kg-day	1E-03										
ACENAPHTHYLENE	6E+00	mg/kg	1E-07					mg/kg-day		1/(mg/kg-day)		8E-07	mg/kg-day	3E-02	mg/kg-day	3E-05										
BENZ(A)ANTHRACENE	2E+02	mg/kg	3E-06					mg/kg-day	7E-01	1/(mg/kg-day)	2E-06	2E-05	mg/kg-day		mg/kg-day											
BENZO(A)PYRENE	1E+02	mg/kg	2E-06					mg/kg-day	7E+00	1/(mg/kg-day)	1E-05	1E-05	mg/kg-day		mg/kg-day											
BENZO(B)FLUORANTHENE	2E+02	mg/kg	4E-06					mg/kg-day	7E-01	1/(mg/kg-day)	3E-06	3E-05	mg/kg-day		mg/kg-day											
BENZO(G,H,I)PERYLENE	7E+01	mg/kg	1E-06					mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	3E-02	mg/kg-day	3E-04										
BENZO(K)FLUORANTHENE	6E+01	mg/kg	1E-06					mg/kg-day	7E-02	1/(mg/kg-day)	8E-08	8E-06	mg/kg-day		mg/kg-day											
BIS(2-ETHYLHEXYL)PHTHALATE	6E+01	mg/kg	9E-07					mg/kg-day	1E-02	1/(mg/kg-day)	1E-08	7E-06	mg/kg-day	2E-02	mg/kg-day	3E-04										
CARBAZOLE	2E+01	mg/kg	3E-07					mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day		mg/kg-day											
CHRYSENE	2E+02	mg/kg	4E-06					mg/kg-day	7E-03	1/(mg/kg-day)	3E-08	3E-05	mg/kg-day		mg/kg-day											
DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	2E-07					mg/kg-day	7E+00	1/(mg/kg-day)	2E-06	2E-06	mg/kg-day		mg/kg-day											
DIBENZOFURAN	3E+01	mg/kg	4E-07					mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	1E-03	mg/kg-day	3E-03										
FLUORANTHENE	1E+02	mg/kg	2E-06					mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	4E-02	mg/kg-day	5E-04										
HEXACHLOROEBENZENE	1E-01	mg/kg	2E-09					mg/kg-day	2E+00	1/(mg/kg-day)	3E-09	1E-08	mg/kg-day	8E-04	mg/kg-day	2E-05										
INDENO(1,2,3-CD)PYRENE	5E+01	mg/kg	1E-06					mg/kg-day	7E-01	1/(mg/kg-day)	7E-07	8E-06	mg/kg-day		mg/kg-day											
NAPHTHALENE	7E+01	mg/kg	1E-06					mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	2E-02	mg/kg-day	5E-04										
PHENANTHRENE	2E+02	mg/kg	4E-06					mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-02	mg/kg-day	1E-03										
PYRENE	4E+02	mg/kg	8E-06					mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	3E-02	mg/kg-day	2E-03										
1,2,4-TRICHLOROBENZENE	8E-01	mg/kg						mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day											
1,4-DICHLOROBENZENE	5E+01	mg/kg						mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day											
BENZENE	8E+00	mg/kg						mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day											

TABLE 7.11 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Sediment	Surface Sediment	Exposure Unit 6	Dermal	CHLOROBENZENE	5E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-02	mg/kg-day	
				METHYLENE CHLORIDE	7E-01	mg/kg		mg/kg-day	8E-03	1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day	
				TOLUENE	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-02	mg/kg-day	
				XYLENES, TOTAL	7E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
			Exp. Route Total								2E-05					2E-02
			Ingestion	2,3,7,8-TCDD Equivalent	1E-04	mg/kg	4E-13	mg/kg-day	2E+05	1/(mg/kg-day)	7E-08	3E-12	mg/kg-day	1E-09	mg/kg-day	3E-03
				ARSENIC	1E+01	mg/kg	4E-08	mg/kg-day	2E+00	1/(mg/kg-day)	6E-08	3E-07	mg/kg-day	3E-04	mg/kg-day	1E-03
				CADMIUM	5E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-05	mg/kg-day	6E-03
				CHROMIUM	7E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	8E-05	mg/kg-day	3E-02
				IRON	1E+04	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	7E-01	mg/kg-day	5E-04
				LEAD	1E+02	mg/kg	6E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day		mg/kg-day	
				MANGANESE	2E+02	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	6E-03	mg/kg-day	1E-03
				MERCURY	8E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	2E-05	mg/kg-day	1E-02
				THALLIUM	8E-01	mg/kg	3E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	8E-05	mg/kg-day	3E-04
				VANADIUM	1E+01	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	2E-04	mg/kg-day	2E-03
				HIGHLY CHLORINATED PCBs	8E-01	mg/kg	3E-09	mg/kg-day	2E+00	1/(mg/kg-day)	6E-09	2E-08	mg/kg-day	2E-05	mg/kg-day	1E-03
				DIELDRIN	2E-02	mg/kg	8E-11	mg/kg-day	2E+01	1/(mg/kg-day)	1E-09	6E-10	mg/kg-day	5E-05	mg/kg-day	1E-05
				ENDRIN KETONE	5E-02	mg/kg	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	3E-04	mg/kg-day	6E-06
				HEPTACHLOR EPOXIDE	1E-02	mg/kg	4E-11	mg/kg-day	9E+00	1/(mg/kg-day)	4E-10	3E-10	mg/kg-day	1E-05	mg/kg-day	2E-05
				2-METHYLNAPHTHALENE	4E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	4E-03	mg/kg-day	3E-04
				ACENAPHTHYLENE	6E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	6E-06
				BENZ(A)ANTHRACENE	2E+02	mg/kg	6E-07	mg/kg-day	7E-01	1/(mg/kg-day)	4E-07	5E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	1E+02	mg/kg	4E-07	mg/kg-day	7E+00	1/(mg/kg-day)	3E-06	3E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E+02	mg/kg	1E-06	mg/kg-day	7E-01	1/(mg/kg-day)	7E-07	7E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	7E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	7E-05
				BENZO(K)FLUORANTHENE	6E+01	mg/kg	2E-07	mg/kg-day	7E-02	1/(mg/kg-day)	2E-08	2E-06	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	6E+01	mg/kg	2E-07	mg/kg-day	1E-02	1/(mg/kg-day)	3E-09	2E-06	mg/kg-day	2E-02	mg/kg-day	1E-04
				CARBAZOLE	2E+01	mg/kg	8E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	2E+02	mg/kg	9E-07	mg/kg-day	7E-03	1/(mg/kg-day)	6E-09	7E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	1E+01	mg/kg	5E-08	mg/kg-day	7E+00	1/(mg/kg-day)	4E-07	4E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	3E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		9E-07	mg/kg-day	1E-03	mg/kg-day	9E-04
				FLUORANTHENE	1E+02	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	4E-02	mg/kg-day	1E-04
				HEXACHLOROBENZENE	1E-01	mg/kg	5E-10	mg/kg-day	2E+00	1/(mg/kg-day)	8E-10	4E-09	mg/kg-day	8E-04	mg/kg-day	5E-06
				INDENO(1,2,3-CD)PYRENE	5E+01	mg/kg	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	2E-07	2E-06	mg/kg-day		mg/kg-day	
				NAPHTHALENE	7E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	2E-02	mg/kg-day	1E-04
				PHENANTHRENE	2E+02	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-02	mg/kg-day	3E-04
				PYRENE	4E+02	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	5E-04
				1,2,4-TRICHLOROBENZENE	8E-01	mg/kg	3E-09	mg/kg-day	4E-03	1/(mg/kg-day)	1E-11	3E-08	mg/kg-day	1E-02	mg/kg-day	3E-06
				1,4-DICHLOROBENZENE	5E+01	mg/kg	2E-07	mg/kg-day	5E-03	1/(mg/kg-day)	1E-09	2E-06	mg/kg-day	7E-02	mg/kg-day	2E-05
				BENZENE	8E+00	mg/kg	3E-08	mg/kg-day	6E-02	1/(mg/kg-day)	2E-09	3E-07	mg/kg-day	4E-03	mg/kg-day	6E-05
				CHLOROBENZENE	5E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	2E-02	mg/kg-day	8E-05
				METHYLENE CHLORIDE	7E-01	mg/kg	3E-09	mg/kg-day	8E-03	1/(mg/kg-day)	2E-11	2E-08	mg/kg-day	6E-02	mg/kg-day	4E-07
TOLUENE	2E+01	mg/kg		8E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	8E-02	mg/kg-day	8E-06			
XYLENES, TOTAL	7E+01	mg/kg		3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	2E-01	mg/kg-day	1E-05			
Exp. Route Total										5E-06				6E-02		
Exp. Point Total										3E-05				8E-02		
Exp. Medium Total										3E-05				8E-02		
Medium Total										3E-05				8E-02		
Soil	Surface Soil	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	3E-07	2E-11	mg/kg-day	1E-09	mg/kg-day	2E-02
				ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	8E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	3E-07	mg/kg-day	3E-04	mg/kg-day	9E-04
				BARIUM	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				CADMIUM	4E+01	mg/kg	5E-09	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day	3E-05	mg/kg-day	2E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				LEAD	7E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	

TABLE 7.11 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Dermal	SILVER	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				THALLIUM	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	6E-08	2E-07	mg/kg-day	2E-05	mg/kg-day	1E-02
				LESS CHLORINATED PCBs	7E-01	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	1E-07	mg/kg-day	7E-05	mg/kg-day	2E-03
				DIELDRIN	1E-01	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				2-METHYLNAPHTHALENE	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	4E-03	mg/kg-day	4E-04
				ACENAPHTHYLENE	4E+00	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	3E-02	mg/kg-day	2E-05
				BENZ(A)ANTHRACENE	8E+00	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	1E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	2E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	1E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	9E-08	9E-07	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	3E-02	mg/kg-day	2E-05
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	1E-07	mg/kg-day	7E-02	1/(mg/kg-day)	7E-09	8E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	1E-07	mg/kg-day	7E-03	1/(mg/kg-day)	1E-09	1E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	3E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	2E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	1E-03	mg/kg-day	4E-04
				HEXACHLOROBENZENE	1E+00	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-08	1E-07	mg/kg-day	8E-04	mg/kg-day	1E-04
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	8E-08	mg/kg-day	7E-01	1/(mg/kg-day)	6E-08	6E-07	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	2E-02	mg/kg-day	2E-04
				PHENANTHRENE	2E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	9E-05
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBENZENE	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	3E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				BENZENE	5E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
			Exp. Route Total								2E-06					3E-02
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	3E-07	2E-11	mg/kg-day	1E-09	mg/kg-day	2E-02
				ALUMINUM	7E+03	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E+00	mg/kg-day	2E-04
				ARSENIC	8E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	5E-08	3E-07	mg/kg-day	3E-04	mg/kg-day	9E-04
				BARIUM	4E+02	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	2E-01	mg/kg-day	6E-05
				CADMIUM	4E+01	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03
				CHROMIUM	1E+02	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-03	mg/kg-day	1E-03
				COPPER	2E+02	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	4E-02	mg/kg-day	2E-04
				IRON	1E+04	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	7E-01	mg/kg-day	6E-04
				LEAD	7E+02	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	1E-01	mg/kg-day	7E-05
				MERCURY	1E+01	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	3E-04	mg/kg-day	1E-03
				SILVER	2E+01	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	5E-03	mg/kg-day	9E-05
				THALLIUM	8E-01	mg/kg	3E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	8E-05	mg/kg-day	3E-04
				VANADIUM	2E+01	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	9E-03	mg/kg-day	8E-05
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	6E-09	mg/kg-day	2E+00	1/(mg/kg-day)	1E-08	5E-08	mg/kg-day	2E-05	mg/kg-day	2E-03
				LESS CHLORINATED PCBs	7E-01	mg/kg	3E-09	mg/kg-day	2E+00	1/(mg/kg-day)	6E-09	2E-08	mg/kg-day	7E-05	mg/kg-day	3E-04
				DIELDRIN	1E-01	mg/kg	5E-10	mg/kg-day	2E+01	1/(mg/kg-day)	7E-09	4E-09	mg/kg-day	5E-05	mg/kg-day	7E-05
				2-METHYLNAPHTHALENE	1E+01	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	4E-03	mg/kg-day	1E-04
				ACENAPHTHYLENE	4E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-02	mg/kg-day	4E-06
				BENZ(A)ANTHRACENE	8E+00	mg/kg	3E-08	mg/kg-day	7E-01	1/(mg/kg-day)	2E-08	2E-07	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	4E-08	mg/kg-day	7E+00	1/(mg/kg-day)	3E-07	3E-07	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	3E-08	mg/kg-day	7E-01	1/(mg/kg-day)	2E-08	2E-07	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	5E-06
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	2E-08	mg/kg-day	7E-02	1/(mg/kg-day)	2E-09	2E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	3E-08	mg/kg-day	7E-03	1/(mg/kg-day)	2E-10	3E-07	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	7E-09	mg/kg-day	7E+00	1/(mg/kg-day)	5E-08	5E-08	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	1E-03	mg/kg-day	1E-04
				HEXACHLOROBENZENE	1E+00	mg/kg	4E-09	mg/kg-day	2E+00	1/(mg/kg-day)	6E-09	3E-08	mg/kg-day	8E-04	mg/kg-day	4E-05
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	2E-08	mg/kg-day	7E-01	1/(mg/kg-day)	1E-08	1E-07	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	9E-08	mg/kg-day		1/(mg/kg-day)		7E-07	mg/kg-day	2E-02	mg/kg-day	4E-05
				PHENANTHRENE	2E+01	mg/kg	8E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	3E-02	mg/kg-day	2E-05

TABLE 7.11 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Soil	Surface Soil	Exposure Unit 6	Dermal	1,2,3-TRICHLOROBENZENE	4E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day		
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day	1E-05	
				1,2-DICHLOROBENZENE	8E+00	mg/kg	3E-08	mg/kg-day	4E-03	1/(mg/kg-day)	6E-11	3E-07	mg/kg-day	1E-02	mg/kg-day	3E-06	
				1,4-DICHLOROBENZENE	3E+01	mg/kg	1E-07	mg/kg-day	5E-03	1/(mg/kg-day)	7E-10	9E-07	mg/kg-day	7E-02	mg/kg-day	1E-05	
				BENZENE	5E-01	mg/kg	2E-09	mg/kg-day	6E-02	1/(mg/kg-day)	1E-10	2E-08	mg/kg-day	4E-03	mg/kg-day	4E-06	
				P-ISOPROPYLTOLUENE	4E-01	mg/kg	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day		
				DODECANE	8E+02	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day		mg/kg-day		
				Exp. Route Total								8E-07				3E-02	
				Exp. Point Total								3E-06				6E-02	
	Exp. Medium Total								3E-06				6E-02				
Medium Total											3E-06				6E-02		
Surface Soil	Outdoor Air	Exposure Unit 6	Inhalation	2,3,7,8-TCDD Equivalent	1E-07	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day		
				ALUMINUM	2E-06	mg/m3	6E-10	mg/kg-day		1/(mg/kg-day)		5E-09	mg/kg-day		mg/kg-day	3E-06	
				ARSENIC	2E-09	mg/m3	7E-13	mg/kg-day	2E+01	1/(mg/kg-day)	1E-11	5E-12	mg/kg-day	1E-05	mg/kg-day	4E-07	
				BARIUM	9E-08	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day	1E-04	mg/kg-day	2E-06	
				CADMIUM	9E-09	mg/m3	3E-12	mg/kg-day	6E+00	1/(mg/kg-day)	2E-11	2E-11	mg/kg-day		mg/kg-day		
				CHROMIUM	3E-08	mg/m3	1E-11	mg/kg-day	4E+01	1/(mg/kg-day)	4E-10	7E-11	mg/kg-day	3E-05	mg/kg-day	3E-06	
				COPPER	6E-08	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day		
				IRON	3E-06	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		8E-09	mg/kg-day		mg/kg-day		
				LEAD	2E-07	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day		mg/kg-day		
				MANGANESE	8E-08	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day	1E-05	mg/kg-day	1E-05	
				MERCURY	3E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		7E-12	mg/kg-day	9E-05	mg/kg-day	9E-08	
				SILVER	4E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day		
				THALLIUM	2E-10	mg/m3	6E-14	mg/kg-day		1/(mg/kg-day)		5E-13	mg/kg-day		mg/kg-day		
				VANADIUM	5E-09	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day		
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	1E-13	mg/kg-day	2E+00	1/(mg/kg-day)	2E-13	1E-12	mg/kg-day		mg/kg-day		
				LESS CHLORINATED PCBs	2E-10	mg/m3	6E-14	mg/kg-day	2E+00	1/(mg/kg-day)	1E-13	5E-13	mg/kg-day		mg/kg-day		
				DIELDRIN	3E-11	mg/m3	9E-15	mg/kg-day	2E+01	1/(mg/kg-day)	1E-13	7E-14	mg/kg-day		mg/kg-day		
				2-METHYLNAPHTHALENE	3E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		8E-12	mg/kg-day		mg/kg-day		
				ACENAPHTHYLENE	1E-09	mg/m3	3E-13	mg/kg-day		1/(mg/kg-day)		2E-12	mg/kg-day		mg/kg-day		
				BENZ(A)ANTHRACENE	2E-09	mg/m3	6E-13	mg/kg-day		1/(mg/kg-day)		5E-12	mg/kg-day		mg/kg-day		
				BENZO(A)PYRENE	2E-09	mg/m3	7E-13	mg/kg-day		1/(mg/kg-day)		6E-12	mg/kg-day		mg/kg-day		
				BENZO(B)FLUORANTHENE	2E-09	mg/m3	5E-13	mg/kg-day		1/(mg/kg-day)		4E-12	mg/kg-day		mg/kg-day		
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	4E-13	mg/kg-day		1/(mg/kg-day)		3E-12	mg/kg-day		mg/kg-day		
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	5E-13	mg/kg-day		1/(mg/kg-day)		4E-12	mg/kg-day		mg/kg-day		
				CHRYSENE	2E-09	mg/m3	7E-13	mg/kg-day		1/(mg/kg-day)		5E-12	mg/kg-day		mg/kg-day		
				DIBENZ(A,H)ANTHRACENE	4E-10	mg/m3	1E-13	mg/kg-day		1/(mg/kg-day)		1E-12	mg/kg-day		mg/kg-day		
				DIBENZOFURAN	1E-09	mg/m3	3E-13	mg/kg-day		1/(mg/kg-day)		3E-12	mg/kg-day		mg/kg-day		
				HEXACHLOROBENZENE	2E-10	mg/m3	8E-14	mg/kg-day	2E+00	1/(mg/kg-day)	1E-13	6E-13	mg/kg-day		mg/kg-day		
				INDENO(1,2,3-CD)PYRENE	1E-09	mg/m3	4E-13	mg/kg-day		1/(mg/kg-day)		3E-12	mg/kg-day		mg/kg-day		
				NAPHTHALENE	6E-09	mg/m3	2E-12	mg/kg-day	1E-01	1/(mg/kg-day)	2E-13	1E-11	mg/kg-day	9E-04	mg/kg-day	2E-08	
				PHENANTHRENE	5E-09	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day		
				1,2,3-TRICHLOROBENZENE	1E-04	mg/m3	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day		
				1,2,4-TRICHLOROBENZENE	1E-04	mg/m3	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day		mg/kg-day		
				1,2-DICHLOROBENZENE	9E-04	mg/m3	3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	4E-02	mg/kg-day	5E-05	
				1,4-DICHLOROBENZENE	4E-03	mg/m3	1E-06	mg/kg-day	4E-02	1/(mg/kg-day)	5E-08	9E-06	mg/kg-day	2E-01	mg/kg-day	4E-05	
				BENZENE	3E-04	mg/m3	1E-07	mg/kg-day	3E-02	1/(mg/kg-day)	3E-09	7E-07	mg/kg-day	9E-03	mg/kg-day	9E-05	
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day		
				DODECANE	2E-07	mg/m3	7E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day		
													5E-08				2E-04
													5E-08				2E-04
													5E-08				2E-04
	Exp. Medium Total								5E-08				2E-04				
Medium Total											5E-08				2E-04		

TABLE 7.11 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations							
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient			
							Value	Units	Value	Units		Value	Units	Value	Units				
Surface Water	Surface Water	Exposure Unit 6	Dermal	ANTIMONY	2E+00	ug/l	4E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	6E-05	mg/kg-day	5E-04			
				ARSENIC	2E+00	ug/l	3E-09	mg/kg-day	2E+00	1/(mg/kg-day)	5E-09	3E-08	mg/kg-day	3E-04	mg/kg-day	9E-05			
				CHROMIUM	5E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	8E-05	mg/kg-day	2E-03			
				IRON	5E+03	ug/l	9E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	7E-01	mg/kg-day	1E-04			
				LEAD	8E+00	ug/l	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day				
				MERCURY	1E-01	ug/l	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	2E-05	mg/kg-day	8E-05			
				THALLIUM	4E+00	ug/l	7E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	8E-05	mg/kg-day	7E-04			
				2,4-DIMETHYLPHENOL	1E+02	ug/l	4E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	2E-02	mg/kg-day	1E-03			
				2-METHYLNAPHTHALENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day				
				3&4-METHYLPHENOL	2E+02	ug/l	4E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	5E-02	mg/kg-day	6E-04			
				ACENAPHTHENE	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day				
				ACENAPHTHYLENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day				
				BENZ(A)ANTHRACENE	4E+00	ug/l	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	7E-06	7E-05	mg/kg-day		mg/kg-day				
				BENZO(A)PYRENE	2E+00	ug/l	8E-06	mg/kg-day	7E+00	1/(mg/kg-day)	6E-05	6E-05	mg/kg-day		mg/kg-day				
				BENZO(B)FLUORANTHENE	3E+00	ug/l	1E-05	mg/kg-day	7E-01	1/(mg/kg-day)	9E-06	1E-04	mg/kg-day		mg/kg-day				
				BIS(2-ETHYLHEXYL)PHTHALATE	8E+00	ug/l	2E-06	mg/kg-day	1E-02	1/(mg/kg-day)	3E-08	2E-05	mg/kg-day	2E-02	mg/kg-day	9E-04			
				CARBAZOLE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day				
				CHRYSENE	4E+00	ug/l	9E-06	mg/kg-day	7E-03	1/(mg/kg-day)	7E-08	7E-05	mg/kg-day		mg/kg-day				
				DIBENZOFURAN	3E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day				
				FLUORENE	2E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day				
				NAPHTHALENE	2E+03	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-02	mg/kg-day	9E-02			
				PHENANTHRENE	3E+01	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	4E-03			
				PYRENE	8E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day				
				1,4-DICHLOROBENZENE	8E+00	ug/l	1E-06	mg/kg-day	5E-03	1/(mg/kg-day)	6E-09	8E-06	mg/kg-day	7E-02	mg/kg-day	1E-04			
				BENZENE	7E+01	ug/l	2E-06	mg/kg-day	6E-02	1/(mg/kg-day)	1E-07	2E-05	mg/kg-day	4E-03	mg/kg-day	5E-03			
				DICHLOROBENZENES	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day				
				TOLUENE	4E+02	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	8E-02	mg/kg-day	3E-03			
				XYLENES, TOTAL	5E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day				
								Exp. Route Total							8E-05				1E-01
								Exp. Point Total							8E-05				1E-01
								Exp. Medium Total							8E-05				1E-01
				Medium Total										8E-05				1E-01	
											Total of Receptor Risks Across All Media				2E-04	Total of Receptor Hazards Across All Media			

TABLE 7.11a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	4E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		6E-09	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	9E-13	mg/kg-day	2E+01	1/(mg/kg-day)	1E-11	7E-12	mg/kg-day	1E-05	mg/kg-day	5E-07
				CADMIUM	8E-09	mg/m3	2E-12	mg/kg-day	6E+00	1/(mg/kg-day)	2E-11	2E-11	mg/kg-day		mg/kg-day	
				CHROMIUM	5E-08	mg/m3	2E-11	mg/kg-day	4E+01	1/(mg/kg-day)	7E-10	1E-10	mg/kg-day	3E-05	mg/kg-day	5E-06
				COPPER	5E-08	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day	1E-05	mg/kg-day	3E-05
				MERCURY	8E-10	mg/m3	3E-13	mg/kg-day		1/(mg/kg-day)		2E-12	mg/kg-day	9E-05	mg/kg-day	2E-08
				VANADIUM	6E-09	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	1E-13	mg/kg-day	2E+00	1/(mg/kg-day)	3E-13	1E-12	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	3E-13	mg/kg-day		1/(mg/kg-day)		2E-12	mg/kg-day		mg/kg-day	
				BENZO(A)ANTHRACENE	4E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	9E-13	mg/kg-day		1/(mg/kg-day)		7E-12	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	3E-13	mg/kg-day		1/(mg/kg-day)		3E-12	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	5E-13	mg/kg-day		1/(mg/kg-day)		4E-12	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	1E-12	mg/kg-day		1/(mg/kg-day)		1E-11	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	8E-14	mg/kg-day		1/(mg/kg-day)		6E-13	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	2E-13	mg/kg-day		1/(mg/kg-day)		2E-12	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	3E-13	mg/kg-day		1/(mg/kg-day)		2E-12	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	2E-12	mg/kg-day		1/(mg/kg-day)		2E-11	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	2E-10	mg/kg-day	3E-02	1/(mg/kg-day)	5E-12	1E-09	mg/kg-day	9E-03	mg/kg-day	2E-07
			Exp. Route Total								8E-10					3E-05
		Exp. Point Total									8E-10					3E-05
	Exp. Medium Total										8E-10					3E-05
Medium Total											8E-10					3E-05
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	1E-13	mg/kg-day	2E+05	1/(mg/kg-day)	2E-08	1E-12	mg/kg-day	1E-09	mg/kg-day	1E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	2E-07	mg/kg-day	3E-04	mg/kg-day	7E-04
				CADMIUM	2E+01	mg/kg	2E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	3E-05	mg/kg-day	7E-04
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg		mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	1E-07	mg/kg-day	2E-05	mg/kg-day	7E-03
				ACENAPHTHYLENE	2E+00	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-02	mg/kg-day	9E-06
				BENZO(A)ANTHRACENE	9E+00	mg/kg	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	1E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	1E-07	mg/kg-day	7E+00	1/(mg/kg-day)	9E-07	9E-07	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	1E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-02	mg/kg-day	1E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	6E-08	mg/kg-day	7E-02	1/(mg/kg-day)	4E-09	5E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	2E-07	mg/kg-day	7E-03	1/(mg/kg-day)	1E-09	1E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	1E-08	mg/kg-day	7E+00	1/(mg/kg-day)	8E-08	8E-08	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	1E-03	mg/kg-day	2E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	3E-08	mg/kg-day	7E-01	1/(mg/kg-day)	2E-08	3E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	7E-05
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								1E-06					1E-02

TABLE 7.11a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units		Quotient	
Soil	Surface Soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	1E-13	mg/kg-day		1/(mg/kg-day)		2E-08	1E-12	mg/kg-day	1E-09	mg/kg-day	1E-03	
				ALUMINUM	5E+03	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E+00	mg/kg-day	2E-04		
				ARSENIC	6E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	2E-07	mg/kg-day	3E-04	mg/kg-day	7E-04		
				CADMIUM	2E+01	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	1E-03	mg/kg-day	5E-04		
				CHROMIUM	1E+02	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-03	mg/kg-day	1E-03		
				COPPER	1E+02	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	4E-02	mg/kg-day	9E-05		
				IRON	1E+04	mg/kg	5E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	7E-01	mg/kg-day	6E-04		
				MANGANESE	3E+02	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-01	mg/kg-day	7E-05		
				MERCURY	2E+00	mg/kg	8E-09	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	3E-04	mg/kg-day	2E-04		
				VANADIUM	1E+01	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	9E-03	mg/kg-day	5E-05		
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	4E-09	mg/kg-day	2E+00	1/(mg/kg-day)	8E-09	3E-08	mg/kg-day	2E-05	mg/kg-day	1E-03		
				ACENAPHTHYLENE	2E+00	mg/kg	8E-09	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	3E-02	mg/kg-day	2E-06		
				BENZ(A)ANTHRACENE	9E+00	mg/kg	4E-08	mg/kg-day	7E-01	1/(mg/kg-day)	3E-08	3E-07	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	7E+00	mg/kg	3E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	2E-07	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	4E-08	mg/kg-day	7E-01	1/(mg/kg-day)	3E-08	3E-07	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	9E-09	mg/kg-day		1/(mg/kg-day)		7E-08	mg/kg-day	3E-02	mg/kg-day	2E-06		
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	1E-08	mg/kg-day	7E-02	1/(mg/kg-day)	1E-09	1E-07	mg/kg-day		mg/kg-day			
				CHRYSENE	1E+01	mg/kg	4E-08	mg/kg-day	7E-03	1/(mg/kg-day)	3E-10	3E-07	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	2E-09	mg/kg-day	7E+00	1/(mg/kg-day)	2E-08	2E-08	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	2E+00	mg/kg	7E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	1E-03	mg/kg-day	5E-05		
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	7E-09	mg/kg-day	7E-01	1/(mg/kg-day)	5E-09	6E-08	mg/kg-day		mg/kg-day			
				PHENANTHRENE	1E+01	mg/kg	6E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	3E-02	mg/kg-day	2E-05		
				BENZENE	1E-03	mg/kg	4E-12	mg/kg-day		1/(mg/kg-day)		3E-11	mg/kg-day	4E-03	mg/kg-day	8E-09		
				Exp. Route Total											3E-07			
		Exp. Point Total											2E-06				2E-02	
	Exp. Medium Total											2E-06				2E-02		
Medium Total											2E-06				2E-02			
							Total of Receptor Risks Across All Media					2E-06	Total of Receptor Hazards Across All Media					2E-02

TABLE 7.12 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Surface Soil	Outdoor Air	Exposure Unit 6	Inhalation	2,3,7,8-TCDD Equivalent	1E-07	mg/m3	7E-09	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day		mg/kg-day			
				ALUMINUM	2E-06	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	8E-04		
				ARSENIC	2E-09	mg/m3	1E-10	mg/kg-day	2E+01	1/(mg/kg-day)	2E-09	1E-09	mg/kg-day	1E-05	mg/kg-day	1E-04		
				BARIIUM	9E-08	mg/m3	5E-09	mg/kg-day		1/(mg/kg-day)		6E-08	mg/kg-day	1E-04	mg/kg-day	4E-04		
				CADMIUM	9E-09	mg/m3	5E-10	mg/kg-day	6E+00	1/(mg/kg-day)	3E-09	6E-09	mg/kg-day		mg/kg-day			
				CHROMIUM	3E-08	mg/m3	2E-09	mg/kg-day	4E+01	1/(mg/kg-day)	7E-08	2E-08	mg/kg-day	3E-05	mg/kg-day	7E-04		
				COPPER	6E-08	mg/m3	3E-09	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day			
				IRON	3E-06	mg/m3	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day		mg/kg-day			
				LEAD	2E-07	mg/m3	9E-09	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day			
				MANGANESE	8E-08	mg/m3	4E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	1E-05	mg/kg-day	3E-03		
				MERCURY	3E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day	9E-05	mg/kg-day	2E-05		
				SILVER	4E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day			
				THALLIUM	2E-10	mg/m3	1E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day			
				VANADIUM	5E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day			
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	2E-11	mg/kg-day	2E+00	1/(mg/kg-day)	4E-11	2E-10	mg/kg-day		mg/kg-day			
				LESS CHLORINATED PCBs	2E-10	mg/m3	1E-11	mg/kg-day	2E+00	1/(mg/kg-day)	2E-11	1E-10	mg/kg-day		mg/kg-day			
				DIELDRIN	3E-11	mg/m3	2E-12	mg/kg-day	2E+01	1/(mg/kg-day)	3E-11	2E-11	mg/kg-day		mg/kg-day			
				2-METHYLNAPHTHALENE	3E-09	mg/m3	2E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day			
				ACENAPHTHYLENE	1E-09	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day		mg/kg-day			
				BENZ(A)ANTHRACENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	7E-11	mg/kg-day		1/(mg/kg-day)		9E-10	mg/kg-day		mg/kg-day			
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				CHRYSENE	2E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	4E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	1E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day			
				HEXACHLOROBENZENE	2E-10	mg/m3	1E-11	mg/kg-day	2E+00	1/(mg/kg-day)	2E-11	2E-10	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day			
				NAPHTHALENE	6E-09	mg/m3	3E-10	mg/kg-day	1E-01	1/(mg/kg-day)	4E-11	4E-09	mg/kg-day	9E-04	mg/kg-day	4E-06		
				PHENANTHRENE	5E-09	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		3E-09	mg/kg-day		mg/kg-day			
				1,2,3-TRICHLOROBENZENE	1E-04	mg/m3	8E-06	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	1E-04	mg/m3	8E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day			
				1,2-DICHLOROBENZENE	9E-04	mg/m3	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	4E-02	mg/kg-day	1E-02		
				1,4-DICHLOROBENZENE	4E-03	mg/m3	2E-04	mg/kg-day	4E-02	1/(mg/kg-day)	8E-06	2E-03	mg/kg-day	2E-01	mg/kg-day	1E-02		
				BENZENE	3E-04	mg/m3	2E-05	mg/kg-day	3E-02	1/(mg/kg-day)	4E-07	2E-04	mg/kg-day	9E-03	mg/kg-day	2E-02		
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				DODECANE	2E-07	mg/m3	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day		mg/kg-day			
				Exp. Route Total										8E-06				5E-02
				Exp. Point Total										8E-06				5E-02
				Exp. Medium Total										8E-06				5E-02
Medium Total										8E-06				5E-02				
Soil	Surface Soil	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	1E-11	mg/kg-day	2E+05	1/(mg/kg-day)	1E-06	1E-10	mg/kg-day	1E-09	mg/kg-day	1E-01		
				ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day			
				ARSENIC	8E+00	mg/kg	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	2E-06	mg/kg-day	3E-04	mg/kg-day	6E-03		
				BARIIUM	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day			
				CADMIUM	4E+01	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	3E-05	mg/kg-day	1E-02		
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				COPPER	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day			
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day			
				LEAD	7E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day			
				MERCURY	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day			
				SILVER	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				THALLIUM	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	2E-06	mg/kg-day	2E-05	mg/kg-day	8E-02		
				LESS CHLORINATED PCBs	7E-01	mg/kg	6E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	7E-07	mg/kg-day	7E-05	mg/kg-day	1E-02		
				DIELDRIN	1E-01	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day			

TABLE 7.12 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Dermal	2-METHYLNAPHTHALENE	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	4E-03	mg/kg-day	3E-03
				ACENAPHTHYLENE	4E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZ(A)ANTHRACENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	3E-02	mg/kg-day	2E-04
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-07	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	1E-03	mg/kg-day	3E-03
				HEXACHLOROBENZENE	1E+00	mg/kg	6E-08	mg/kg-day	2E+00	1/(mg/kg-day)	9E-08	7E-07	mg/kg-day	8E-04	mg/kg-day	9E-04
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-02	mg/kg-day	1E-03
				PHENANTHRENE	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	3E-02	mg/kg-day	6E-04
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day	4E-03				mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBENZENE	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	3E+01	mg/kg		mg/kg-day	5E-03				mg/kg-day	7E-02	mg/kg-day	
				BENZENE	5E-01	mg/kg		mg/kg-day	6E-02				mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				Exp. Route Total							5E-05					2E-01
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	3E-10	mg/kg-day	2E+05	1/(mg/kg-day)	4E-05	3E-09	mg/kg-day	1E-09	mg/kg-day	3E+00
				ALUMINUM	7E+03	mg/kg	4E-03	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	1E+00	mg/kg-day	5E-02
				ARSENIC	8E+00	mg/kg	5E-06	mg/kg-day	2E+00			5E-05	mg/kg-day	3E-04	mg/kg-day	2E-01
				BARIIUM	4E+02	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-01	mg/kg-day	1E-02
				CADMIUM	4E+01	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-03	mg/kg-day	2E-01
				CHROMIUM	1E+02	mg/kg	6E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day	3E-03	mg/kg-day	2E-01
				COPPER	2E+02	mg/kg	1E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	4E-02	mg/kg-day	4E-02
				IRON	1E+04	mg/kg	7E-03	mg/kg-day		1/(mg/kg-day)		8E-02	mg/kg-day	7E-01	mg/kg-day	1E-01
				LEAD	7E+02	mg/kg	4E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E-01	mg/kg-day	1E-02
				MERCURY	1E+01	mg/kg	6E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	3E-04	mg/kg-day	2E-01
				SILVER	2E+01	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	5E-03	mg/kg-day	2E-02
				THALLIUM	8E-01	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day	8E-05	mg/kg-day	6E-02
				VANADIUM	2E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	9E-03	mg/kg-day	2E-02
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	8E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-06	1E-05	mg/kg-day	2E-05	mg/kg-day	5E-01
				LESS CHLORINATED PCBs	7E-01	mg/kg	4E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	5E-06	mg/kg-day	7E-05	mg/kg-day	7E-02
				DIELDRIN	1E-01	mg/kg	6E-08	mg/kg-day	2E+01	1/(mg/kg-day)	1E-06	7E-07	mg/kg-day	5E-05	mg/kg-day	1E-02
				2-METHYLNAPHTHALENE	1E+01	mg/kg	7E-06	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	4E-03	mg/kg-day	2E-02
				ACENAPHTHYLENE	4E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-02	mg/kg-day	8E-04
				BENZ(A)ANTHRACENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	3E-02	mg/kg-day	1E-03
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-07	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	1E-03	mg/kg-day	3E-02
				HEXACHLOROBENZENE	1E+00	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	6E-06	mg/kg-day	8E-04	mg/kg-day	8E-03
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-05	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	2E-02	mg/kg-day	8E-03
				PHENANTHRENE	2E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	4E-03
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day	1E-02	mg/kg-day	3E-03
				1,2-DICHLOROBENZENE	8E+00	mg/kg	5E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	9E-02	mg/kg-day	6E-04
				1,4-DICHLOROBENZENE	3E+01	mg/kg	2E-05	mg/kg-day	5E-03		9E-08	2E-04	mg/kg-day	7E-02	mg/kg-day	3E-03

TABLE 7.12 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Ingestion	BENZENE	5E-01	mg/kg	3E-07	mg/kg-day	6E-02	1/(mg/kg-day)	2E-08	3E-06	mg/kg-day	4E-03	mg/kg-day	9E-04
				P-ISOPROPYLTOLUENE	4E-01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day			
				DODECANE	8E+02	mg/kg	4E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day			
				Exp. Route Total												
	Exp. Point Total												5E+00			
Exp. Medium Total															5E+00	
																5E+00
																5E+00
																5E+00
Medium Total																5E+00
Ground Water	Potable Water	Exposure Unit 8	Dermal	ALUMINUM	2E+04	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	1E+00	mg/kg-day	3E-03
				ANTIMONY	2E+00	ug/l	3E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	6E-05	mg/kg-day	5E-03
				ARSENIC	9E+00	ug/l	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	1E-06	mg/kg-day	3E-04	mg/kg-day	4E-03
				BARIUM	1E+03	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-02	mg/kg-day	1E-02
				BERYLLIUM	8E-01	ug/l	1E-08	mg/kg-day	1/(mg/kg-day)	1E-07	mg/kg-day	1E-05	mg/kg-day	8E-03		
				CADIUM	2E+00	ug/l	2E-08	mg/kg-day	1/(mg/kg-day)	3E-07	mg/kg-day	3E-05	mg/kg-day	1E-02		
				CHROMIUM	7E+01	ug/l	2E-06	mg/kg-day	1/(mg/kg-day)	2E-05	mg/kg-day	8E-05	mg/kg-day	3E-01		
				COBALT	1E+01	ug/l	6E-08	mg/kg-day	1/(mg/kg-day)	6E-07	mg/kg-day		mg/kg-day			
				COPPER	9E+01	ug/l	1E-06	mg/kg-day	1/(mg/kg-day)	1E-05	mg/kg-day	4E-02	mg/kg-day	3E-04		
				CYANIDE	3E+01	ug/l	4E-07	mg/kg-day	1/(mg/kg-day)	4E-06	mg/kg-day	2E-02	mg/kg-day	2E-04		
				IRON	4E+04	ug/l	5E-04	mg/kg-day	1/(mg/kg-day)	5E-03	mg/kg-day	7E-01	mg/kg-day	8E-03		
				LEAD	6E+01	ug/l	8E-08	mg/kg-day	1/(mg/kg-day)	9E-07	mg/kg-day		mg/kg-day			
				MANGANESE	2E+03	ug/l	2E-05	mg/kg-day	1/(mg/kg-day)	3E-04	mg/kg-day	6E-03	mg/kg-day	5E-02		
				MERCURY	2E+00	ug/l	2E-08	mg/kg-day	1/(mg/kg-day)	1E-07	mg/kg-day	2E-05	mg/kg-day	6E-03		
				NICKEL	5E+01	ug/l	1E-07	mg/kg-day	1/(mg/kg-day)	1E-06	mg/kg-day	8E-04	mg/kg-day	2E-03		
				SELENIUM	4E+00	ug/l	4E-08	mg/kg-day	1/(mg/kg-day)	5E-07	mg/kg-day	5E-03	mg/kg-day	1E-04		
				SILVER	2E+00	ug/l	2E-08	mg/kg-day	1/(mg/kg-day)	2E-07	mg/kg-day	2E-04	mg/kg-day	9E-04		
				THALLIUM	7E+00	ug/l	8E-08	mg/kg-day	1/(mg/kg-day)	1E-06	mg/kg-day	8E-05	mg/kg-day	1E-02		
				VANADIUM	4E+01	ug/l	5E-07	mg/kg-day	1/(mg/kg-day)	6E-06	mg/kg-day	2E-04	mg/kg-day	3E-02		
				ZINC	1E+02	ug/l	7E-07	mg/kg-day	1/(mg/kg-day)	8E-06	mg/kg-day	3E-01	mg/kg-day	3E-05		
				HIGHLY CHLORINATED PCBs	7E-02	ug/l		mg/kg-day	2E+00	1/(mg/kg-day)		mg/kg-day	2E-05	mg/kg-day		
				4,4'-DDD	9E-02	ug/l	2E-06	mg/kg-day		2E-01	1/(mg/kg-day)	5E-07	2E-05	mg/kg-day	mg/kg-day	
				4,4'-DDT	1E+00	ug/l	4E-05	mg/kg-day	3E-01	1/(mg/kg-day)	1E-05	4E-04	mg/kg-day	5E-04	mg/kg-day	9E-01
				ALDRIN	3E-02	ug/l	9E-09	mg/kg-day	2E+01	1/(mg/kg-day)	2E-07	1E-07	mg/kg-day	3E-05	mg/kg-day	4E-03
				ALPHA-BHC	2E-01	ug/l		mg/kg-day	6E+00	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				ENDOSULFAN II	6E-02	ug/l		mg/kg-day		1/(mg/kg-day)				mg/kg-day	6E-03	mg/kg-day
				ENDOSULFAN SULFATE	2E-02	ug/l		mg/kg-day	1/(mg/kg-day)				mg/kg-day	6E-03	mg/kg-day	
				HEPTACHLOR EPOXIDE	1E-02	ug/l		mg/kg-day	9E+00	1/(mg/kg-day)			mg/kg-day	1E-05	mg/kg-day	
				1,1'-BIPHENYL	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)				mg/kg-day	5E-02	mg/kg-day
				2,4-DICHLOROPHENOL	1E+01	ug/l	1E-05	mg/kg-day	1E-05	1/(mg/kg-day)		1E-04	mg/kg-day	3E-03	mg/kg-day	4E-02
				2,4-DIMETHYLPHENOL	4E+03	ug/l	2E-03	mg/kg-day	1/(mg/kg-day)	2E-02	mg/kg-day	2E-02	mg/kg-day	2E-02	mg/kg-day	1E+00
				2-METHYLNAPHTHALENE	6E+02	ug/l		mg/kg-day	1/(mg/kg-day)				mg/kg-day	4E-03	mg/kg-day	
				2-METHYLPHENOL	1E+03	ug/l	3E-04	mg/kg-day	1/(mg/kg-day)	3E-03	mg/kg-day	5E-02	mg/kg-day	mg/kg-day	7E-02	
				2-NITROPHENOL	6E+00	ug/l	1E-06	mg/kg-day	1/(mg/kg-day)	1E-05	mg/kg-day		mg/kg-day		mg/kg-day	
				3&4-METHYLPHENOL	4E+03	ug/l	1E-03	mg/kg-day	1/(mg/kg-day)	1E-02	mg/kg-day	5E-02	mg/kg-day		3E-01	
				4-CHLORO-3-METHYLPHENOL	1E+00	ug/l	1E-06	mg/kg-day	1/(mg/kg-day)	2E-05	mg/kg-day		mg/kg-day			
				4-METHYLPHENOL	8E+03	ug/l	2E-03	mg/kg-day	1/(mg/kg-day)	3E-02	mg/kg-day	5E-02	mg/kg-day		6E-01	
				4-NITROPHENOL	1E+01	ug/l	2E-06	mg/kg-day	1/(mg/kg-day)	2E-05	mg/kg-day		mg/kg-day			
				ACENAPHTHENE	1E+02	ug/l		mg/kg-day	1/(mg/kg-day)				mg/kg-day	6E-02	mg/kg-day	
				ACENAPHTHYLENE	2E+02	ug/l		mg/kg-day	1/(mg/kg-day)				mg/kg-day	3E-02	mg/kg-day	
				ANTHRACENE	1E+02	ug/l		mg/kg-day	1/(mg/kg-day)				mg/kg-day	3E-01	mg/kg-day	
				ATRAZINE	5E+01	ug/l		mg/kg-day	1/(mg/kg-day)				mg/kg-day	4E-02	mg/kg-day	
				BENZ(A)ANTHRACENE	5E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-02	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-02	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-03	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				BENZO(K)FLUORANTHENE	2E+01	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	5E-05	mg/kg-day	1E-02	1/(mg/kg-day)	7E-07	6E-04	mg/kg-day	2E-02	mg/kg-day	3E-02
				CARBAZOLE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				CHRYSENE	4E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-02	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-03	mg/kg-day	

TABLE 7.12 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Potable Water	Exposure Unit 8	Dermal	FLUORANTHENE	2E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	4E-02	mg/kg-day	7E-01
				FLUORENE	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				HEXACHLOROBUTADIENE	1E+00	ug/l	7E-06	mg/kg-day	8E-02	1/(mg/kg-day)	6E-07	9E-05	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-03	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	4E+03	ug/l	8E-03	mg/kg-day		1/(mg/kg-day)		9E-02	mg/kg-day	2E-02	mg/kg-day	5E+00
				NITROBENZENE	3E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	5E-04	mg/kg-day	
				PHENANTHRENE	4E+02	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	3E-02	mg/kg-day	1E+00
				PHENOL	2E+03	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-01	mg/kg-day	1E-02
				PYRENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	5E-05	mg/kg-day	4E-03	1/(mg/kg-day)	2E-07	6E-04	mg/kg-day	1E-02	mg/kg-day	6E-02
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2-DICHLOROBENZENE	5E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	9E-02	mg/kg-day	1E-01
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,3-DICHLOROBENZENE	5E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day	
				1,4-DICHLOROBENZENE	5E+02	ug/l	1E-03	mg/kg-day	5E-03	1/(mg/kg-day)	5E-06	1E-02	mg/kg-day	7E-02	mg/kg-day	2E-01
				2-HEXANONE	2E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
				ACETONE	8E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-01	mg/kg-day	
				BENZENE	6E+03	ug/l	3E-03	mg/kg-day	6E-02	1/(mg/kg-day)	1E-04	3E-02	mg/kg-day	4E-03	mg/kg-day	8E+00
				BROMODICHLOROMETHANE	3E+00	ug/l	7E-07	mg/kg-day	6E-02	1/(mg/kg-day)	5E-08	9E-06	mg/kg-day	2E-02	mg/kg-day	4E-04
				CARBON DISULFIDE	1E+01	ug/l	7E-06	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	1E-01	mg/kg-day	8E-04
				CHLOROETHANE	2E+02	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-02	mg/kg-day	1E-01
				CHLOROETHANE	5E+00	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day		mg/kg-day	
				ETHYLBENZENE	1E+02	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	1E-01	mg/kg-day	3E-02
				ISOPROPYLBENZENE	4E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-01	mg/kg-day	
				METHYLENE CHLORIDE	7E-01	ug/l	8E-08	mg/kg-day	8E-03	1/(mg/kg-day)	6E-10	1E-06	mg/kg-day	6E-02	mg/kg-day	2E-05
				P-ISOPROPYLTOLUENE	3E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				SEC-BUTYLBENZENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				STYRENE	8E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	2E-01	mg/kg-day	7E-02
				TETRACHLOROETHENE	3E-01	ug/l	5E-07	mg/kg-day	5E-01	1/(mg/kg-day)	3E-07	6E-06	mg/kg-day	1E-02	mg/kg-day	6E-04
				TOLUENE	1E+03	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	8E-02	mg/kg-day	2E-01
				VINYL CHLORIDE	1E+00	ug/l	2E-07	mg/kg-day	8E-01	1/(mg/kg-day)	1E-07	2E-06	mg/kg-day	3E-03	mg/kg-day	7E-04
				XYLENES, TOTAL	1E+03	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
			Exp. Route Total								9E-02					2E+01
			Ingestion	ALUMINUM	2E+04	ug/l	1E-01	mg/kg-day		1/(mg/kg-day)		2E+00	mg/kg-day	1E+00	mg/kg-day	2E+00
				ANTIMONY	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	4E-04	mg/kg-day	4E-01
				ARSENIC	9E+00	ug/l	5E-05	mg/kg-day	2E+00	1/(mg/kg-day)	8E-05	6E-04	mg/kg-day	3E-04	mg/kg-day	2E+00
				BARIUM	1E+03	ug/l	8E-03	mg/kg-day		1/(mg/kg-day)		9E-02	mg/kg-day	2E-01	mg/kg-day	5E-01
				BERYLLIUM	8E-01	ug/l	4E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	2E-03	mg/kg-day	3E-02
				CADMIUM	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-03	mg/kg-day	1E-01
				CHROMIUM	7E+01	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	3E-03	mg/kg-day	1E+00
				COBALT	1E+01	ug/l	6E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day		mg/kg-day	
				COPPER	9E+01	ug/l	5E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	4E-02	mg/kg-day	1E-01
				CYANIDE	3E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-02	mg/kg-day	1E-01
				IRON	4E+04	ug/l	2E-01	mg/kg-day		1/(mg/kg-day)		3E+00	mg/kg-day	7E-01	mg/kg-day	4E+00
				LEAD	6E+01	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day		mg/kg-day	
				MANGANESE	2E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	1E-01	mg/kg-day	8E-01
				MERCURY	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-04	mg/kg-day	5E-01
				NICKEL	5E+01	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	2E-02	mg/kg-day	2E-01
				SELENIUM	4E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	5E-03	mg/kg-day	5E-02
				SILVER	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	5E-03	mg/kg-day	3E-02
				THALLIUM	7E+00	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	8E-05	mg/kg-day	6E+00
				VANADIUM	4E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	9E-03	mg/kg-day	3E-01
				ZINC	1E+02	ug/l	5E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	3E-01	mg/kg-day	2E-02
				HIGHLY CHLORINATED PCBs	7E-02	ug/l	4E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	4E-06	mg/kg-day	2E-05	mg/kg-day	2E-01
				4,4'-DDD	9E-02	ug/l	5E-07	mg/kg-day	2E-01	1/(mg/kg-day)	1E-07	6E-06	mg/kg-day		mg/kg-day	
				4,4'-DDT	1E+00	ug/l	6E-06	mg/kg-day	3E-01	1/(mg/kg-day)	2E-06	7E-05	mg/kg-day	5E-04	mg/kg-day	1E-01
				ALDRIN	3E-02	ug/l	2E-07	mg/kg-day	2E+01	1/(mg/kg-day)	3E-06	2E-06	mg/kg-day	3E-05	mg/kg-day	7E-02

TABLE 7.12 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Potable Water	Exposure Unit 8	Ingestion	ALPHA-BHC	2E-01	ug/l	1E-06	mg/kg-day	6E+00	1/(mg/kg-day)	7E-06	1E-05	mg/kg-day		mg/kg-day	
				ENDOSULFAN II	6E-02	ug/l	3E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	6E-03	mg/kg-day	7E-04
				ENDOSULFAN SULFATE	2E-02	ug/l	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	6E-03	mg/kg-day	2E-04
				HEPTACHLOR EPOXIDE	1E-02	ug/l	5E-08	mg/kg-day	9E+00	1/(mg/kg-day)	5E-07	6E-07	mg/kg-day	1E-05	mg/kg-day	5E-02
				1,1'-BIPHENYL	1E+01	ug/l	7E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	5E-02	mg/kg-day	2E-02
				2,4-DICHLOROPHENOL	1E+01	ug/l	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day	3E-03	mg/kg-day	2E-01
				2,4-DIMETHYLPHENOL	4E+03	ug/l	2E-02	mg/kg-day		1/(mg/kg-day)		3E-01	mg/kg-day	2E-02	mg/kg-day	1E+01
				2-METHYLNAPHTHALENE	6E+02	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	4E-03	mg/kg-day	1E+01
				2-METHYLPHENOL	1E+03	ug/l	5E-03	mg/kg-day		1/(mg/kg-day)		6E-02	mg/kg-day	5E-02	mg/kg-day	1E+00
				2-NITROPHENOL	6E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day		mg/kg-day	
				3&4-METHYLPHENOL	4E+03	ug/l	2E-02	mg/kg-day		1/(mg/kg-day)		3E-01	mg/kg-day	5E-02	mg/kg-day	6E+00
				4-CHLORO-3-METHYLPHENOL	1E+00	ug/l	5E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day		mg/kg-day	
				4-METHYLPHENOL	8E+03	ug/l	5E-02	mg/kg-day		1/(mg/kg-day)		5E-01	mg/kg-day	5E-02	mg/kg-day	1E+01
				4-NITROPHENOL	1E+01	ug/l	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day		mg/kg-day	
				ACENAPHTHENE	1E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	6E-02	mg/kg-day	1E-01
				ACENAPHTHYLENE	2E+02	ug/l	9E-04	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	3E-02	mg/kg-day	4E-01
				ANTHRACENE	1E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	3E-01	mg/kg-day	2E-02
				ATRAZINE	5E+01	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	4E-02	mg/kg-day	1E-01
				BENZ(A)ANTHRACENE	5E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	6E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	3E-02	mg/kg-day	1E-02
				BENZO(K)FLUORANTHENE	2E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-06	(a)	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	6E-05	mg/kg-day	1E-02	1/(mg/kg-day)	8E-07	7E-04	mg/kg-day	2E-02	mg/kg-day	3E-02
				CARBAZOLE	1E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day		mg/kg-day	
				CHRYSENE	4E+01	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	1E-03	mg/kg-day	1E+01
				FLUORANTHENE	2E+02	ug/l	9E-04	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	4E-02	mg/kg-day	3E-01
				FLUORENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	4E-02	mg/kg-day	3E-01
				HEXACHLOROBUTADIENE	1E+00	ug/l	5E-06	mg/kg-day	8E-02	1/(mg/kg-day)	4E-07	6E-05	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day	
				NAPHTHALENE	4E+03	ug/l	2E-02	mg/kg-day		1/(mg/kg-day)		3E-01	mg/kg-day	2E-02	mg/kg-day	1E+01
				NITROBENZENE	3E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	5E-04	mg/kg-day	3E-01
				PHENANTHRENE	4E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	3E-02	mg/kg-day	9E-01
				PHENOL	2E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	3E-01	mg/kg-day	4E-01
				PYRENE	1E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	3E-02	mg/kg-day	2E-01
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l	7E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	7E-05	mg/kg-day	4E-03	1/(mg/kg-day)	3E-07	9E-04	mg/kg-day	1E-02	mg/kg-day	9E-02
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day		mg/kg-day	
				1,2-DICHLOROBENZENE	5E+02	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	9E-02	mg/kg-day	4E-01
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day		mg/kg-day	
				1,3-DICHLOROBENZENE	5E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day	
				1,4-DICHLOROBENZENE	5E+02	ug/l	3E-03	mg/kg-day	5E-03	1/(mg/kg-day)	1E-05	3E-02	mg/kg-day	7E-02	mg/kg-day	4E-01
				2-HEXANONE	2E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	2E-01	mg/kg-day	6E-04
				ACETONE	8E+01	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	9E-01	mg/kg-day	6E-03
				BENZENE	6E+03	ug/l	3E-02	mg/kg-day	6E-02	1/(mg/kg-day)	2E-03	4E-01	mg/kg-day	4E-03	mg/kg-day	9E+01
				BROMODICHLOROMETHANE	3E+00	ug/l	2E-05	mg/kg-day	6E-02	1/(mg/kg-day)	1E-06	2E-04	mg/kg-day	2E-02	mg/kg-day	1E-02
				CARBON DISULFIDE	1E+01	ug/l	7E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	1E-01	mg/kg-day	8E-03
				CHLOROENZENE	2E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	2E-02	mg/kg-day	6E-01
				CHLOROETHANE	5E+00	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day	
				ETHYLBENZENE	1E+02	ug/l	8E-04	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day	1E-01	mg/kg-day	9E-02
				ISOPROPYLBENZENE	4E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-01	mg/kg-day	3E-03
				METHYLENE CHLORIDE	7E-01	ug/l	4E-06	mg/kg-day	8E-03	1/(mg/kg-day)	3E-08	5E-05	mg/kg-day	6E-02	mg/kg-day	8E-04
				P-ISOPROPYLTOLUENE	3E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day	
				SEC-BUTYLBENZENE	1E+01	ug/l	7E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day		mg/kg-day	
				STYRENE	8E+02	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	2E-01	mg/kg-day	3E-01

TABLE 7.12 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations					
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient	
							Value	Units	Value	Units		Value	Units	Value	Units		
Ground Water	Potable Water	Exposure Unit 8	Ingestion	TETRACHLOROETHENE	3E-01	ug/l	2E-06	mg/kg-day	5E-01	1/(mg/kg-day)	9E-07	2E-05	mg/kg-day	1E-02	mg/kg-day	2E-03	
				TOLUENE	1E+03	ug/l	7E-03	mg/kg-day		1/(mg/kg-day)		8E-02	mg/kg-day	8E-02	mg/kg-day	1E+00	
				VINYL CHLORIDE	1E+00	ug/l	6E-06	mg/kg-day	8E-01	1/(mg/kg-day)	5E-06	7E-05	mg/kg-day	3E-03	mg/kg-day	2E-02	
				XYLENES, TOTAL	1E+03	ug/l	5E-03	mg/kg-day		1/(mg/kg-day)		6E-02	mg/kg-day	2E-01	mg/kg-day	3E-01	
				Exp. Route Total													
		Exp. Point Total									3E-03					2E+02	
		Exp. Medium Total									1E-01						2E+02
	Shower Vapor	Exposure Unit 8	Inhalation	1,2,3-TRICHLOROBENZENE	8E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day		mg/kg-day		
1,2,4-TRICHLOROBENZENE				9E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day		mg/kg-day			
1,2,4-TRIMETHYLBENZENE				2E+00	mg/m3	5E-03	mg/kg-day		1/(mg/kg-day)		6E-02	mg/kg-day	2E-03	mg/kg-day	3E+01		
1,2-DICHLOROBENZENE				4E+00	mg/m3	8E-03	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	4E-02	mg/kg-day	2E+00		
1,3,5-TRIMETHYLBENZENE				1E+00	mg/m3	3E-03	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day		mg/kg-day			
1,3-DICHLOROBENZENE				4E-02	mg/m3	9E-05	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day		mg/kg-day			
1,4-DICHLOROBENZENE				3E+00	mg/m3	7E-03	mg/kg-day	4E-02	1/(mg/kg-day)	3E-04	9E-02	mg/kg-day	2E-01	mg/kg-day	4E-01		
2-HEXANONE				1E-02	mg/m3	3E-05	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	6E-02	mg/kg-day	6E-03		
ACETONE				5E-01	mg/m3	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	9E+00	mg/kg-day	2E-03		
BENZENE				4E+01	mg/m3	9E-02	mg/kg-day	3E-02	1/(mg/kg-day)	3E-03	1E+00	mg/kg-day	9E-03	mg/kg-day	1E+02		
BROMODICHLOROMETHANE				2E-02	mg/m3	5E-05	mg/kg-day	1E-01	1/(mg/kg-day)	6E-06	6E-04	mg/kg-day		mg/kg-day			
CARBON DISULFIDE				9E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	2E-01	mg/kg-day	1E-02		
CHLOROENZENE				1E+00	mg/m3	3E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day		mg/kg-day			
CHLOROETHANE				3E-02	mg/m3	7E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	3E+00	mg/kg-day	3E-04		
CHLOROFORM				8E-02	mg/m3	2E-04	mg/kg-day	8E-02	1/(mg/kg-day)	2E-05	2E-03	mg/kg-day	3E-02	mg/kg-day	8E-02		
ETHYLBENZENE				1E+00	mg/m3	2E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	3E-01	mg/kg-day	9E-02		
ISOPROPYLBENZENE				3E-02	mg/m3	6E-05	mg/kg-day		1/(mg/kg-day)		7E-04	mg/kg-day	1E-01	mg/kg-day	7E-03		
METHYLENE CHLORIDE				5E-03	mg/m3	1E-05	mg/kg-day	2E-03	1/(mg/kg-day)	2E-08	1E-04	mg/kg-day	3E-01	mg/kg-day	5E-04		
P-ISOPROPYLTOLUENE				2E-02	mg/m3	5E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day		mg/kg-day			
SEC-BUTYLBENZENE				8E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day		mg/kg-day			
STYRENE				6E+00	mg/m3	1E-02	mg/kg-day		1/(mg/kg-day)		2E-01	mg/kg-day	3E+00	mg/kg-day	5E-02		
TETRACHLOROETHENE				2E-03	mg/m3	5E-06	mg/kg-day	2E-05	1/(mg/kg-day)	1E-10	5E-05	mg/kg-day	8E-02	mg/kg-day	7E-04		
TOLUENE				9E+00	mg/m3	2E-02	mg/kg-day		1/(mg/kg-day)		2E-01	mg/kg-day	1E+00	mg/kg-day	2E-01		
VINYL CHLORIDE				8E-03	mg/m3	2E-05	mg/kg-day	3E-02	1/(mg/kg-day)	5E-07	2E-04	mg/kg-day	3E-02	mg/kg-day	6E-03		
XYLENES, TOTAL				5E+00	mg/m3	1E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	3E-02	mg/kg-day	5E+00		
				Exp. Route Total								3E-03					2E+02
		Exp. Point Total															
	Exp. Medium Total									3E-03					2E+02		
Medium Total										1E-01		Total of Receptor Hazards Across All Media				4E+02	
												Total of Receptor Risks Across All Media				1E-01	

Notes:
(a) See Table 7.12 CT Supplement A for the intake and toxicity values for COPCs with an MMOA

TABLE 7.12 CT Supplement A
 CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION
 CENTRAL TENDENCY
 HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Resident
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	EU-6	Ingestion	Benz(a)anthracene	7.5E+00	mg/kg	2.0E-06	2.6E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-05
				Benzo(a)pyrene	9.0E+00	mg/kg	2.5E-06	3.1E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-04
				Benzo(b)fluoranthene	6.6E+00	mg/kg	1.8E-06	2.3E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-05
				Benzo(k)fluoranthene	5.7E+00	mg/kg	1.5E-06	1.9E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	2.E-06
				Chrysene	8.0E+00	mg/kg	2.2E-06	2.7E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	2.E-07
				Dibenz(a,h)anthracene	1.6E+00	mg/kg	4.4E-07	5.6E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	4.E-05
				Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	1.2E-06	1.6E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-05
		Dermal	Benz(a)anthracene	7.5E+00	mg/kg	2.8E-07	3.9E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-06	
			Benzo(a)pyrene	9.0E+00	mg/kg	3.4E-07	4.7E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	3.E-05	
			Benzo(b)fluoranthene	6.6E+00	mg/kg	2.5E-07	3.4E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-06	
			Benzo(k)fluoranthene	5.7E+00	mg/kg	2.1E-07	2.9E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	2.E-07	
			Chrysene	8.0E+00	mg/kg	3.0E-07	4.1E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	3.E-08	
			Dibenz(a,h)anthracene	1.6E+00	mg/kg	6.1E-08	8.5E-08	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	6.E-06	
			Indeno(1,2,3-cd)pyrene	4.6E+00	mg/kg	1.7E-07	2.4E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-06	
	Fugitive Dust	EU-6	Inhalation	Benz(a)anthracene	1.9E-09	mg/m ³	2.9E-11	5.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	2.3E-09	mg/m ³	3.4E-11	6.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	1.7E-09	mg/m ³	2.5E-11	4.4E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	1.4E-09	mg/m ³	2.2E-11	3.8E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.0E-09	mg/m ³	3.1E-11	5.4E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	4.1E-10	mg/m ³	6.3E-12	1.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	1.2E-09	mg/m ³	1.7E-11	3.1E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
Ground Water	Potable Water	EU-8	Ingestion	Benz(a)anthracene	5.5E+01	µg/L	1.5E-05	2.5E-05	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	2.E-04
				Benzo(a)pyrene	2.0E+01	µg/L	5.5E-06	9.3E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	6.E-04
				Benzo(b)fluoranthene	2.1E+01	µg/L	5.9E-06	9.9E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	6.E-05
				Benzo(k)fluoranthene	1.8E+01	µg/L	5.0E-06	8.3E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	5.E-06
				Chrysene	3.5E+01	µg/L	9.7E-06	1.6E-05	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	1.E-06
				Dibenz(a,h)anthracene	2.8E+00	µg/L	7.8E-07	1.3E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	9.E-05
				Indeno(1,2,3-cd)pyrene	8.5E+00	µg/L	2.3E-06	3.9E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
		Dermal	Benz(a)anthracene	5.5E+01	µg/L	9.3E-04	1.3E-03	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	1.E-02	
			Benzo(a)pyrene	2.0E+01	µg/L	5.8E-04	8.4E-04	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	6.E-02	
			Benzo(b)fluoranthene	2.1E+01	µg/L	6.3E-04	9.1E-04	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	7.E-03	
			Benzo(k)fluoranthene	1.8E+01	µg/L			mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)		
			Chrysene	3.5E+01	µg/L	6.0E-04	8.7E-04	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	6.E-05	
			Dibenz(a,h)anthracene	2.8E+00	µg/L	1.3E-04	1.8E-04	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	1.E-02	
			Indeno(1,2,3-cd)pyrene	8.5E+00	µg/L	2.5E-04	3.6E-04	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-03	

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include

TABLE 7.12a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	7E-10	mg/kg-day		1/(mg/kg-day)		9E-09	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	1E-10	mg/kg-day	2E+01	1/(mg/kg-day)	2E-09	2E-09	mg/kg-day	1E-05	mg/kg-day	1E-04
				CADMIUM	8E-09	mg/m3	4E-10	mg/kg-day	6E+00	1/(mg/kg-day)	3E-09	5E-09	mg/kg-day		mg/kg-day	
				CHROMIUM	5E-08	mg/m3	3E-09	mg/kg-day	4E+01	1/(mg/kg-day)	1E-07	3E-08	mg/kg-day	3E-05	mg/kg-day	1E-03
				COPPER	5E-08	mg/m3	3E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	3E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	8E-09	mg/kg-day		1/(mg/kg-day)		9E-08	mg/kg-day	1E-05	mg/kg-day	7E-03
				MERCURY	8E-10	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day	9E-05	mg/kg-day	6E-06
				VANADIUM	6E-09	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	2E-11	mg/kg-day	2E+00	1/(mg/kg-day)	4E-11	3E-10	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	(a)	mg/kg-day		1/(mg/kg-day)		(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	4E-10	mg/kg-day		1/(mg/kg-day)		4E-09	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	3E-08	mg/kg-day	3E-02	1/(mg/kg-day)	8E-10	3E-07	mg/kg-day	9E-03	mg/kg-day	4E-05
			Exp. Route Total								1E-07					9E-03
		Exp. Point Total									1E-07					9E-03
	Exp. Medium Total										1E-07					9E-03
Medium Total											1E-07					9E-03
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	6E-13	mg/kg-day	2E+05	1/(mg/kg-day)	9E-08	7E-12	mg/kg-day	1E-09	mg/kg-day	7E-03
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	1E-06	mg/kg-day	3E-04	mg/kg-day	4E-03
				CADMIUM	2E+01	mg/kg	1E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-05	mg/kg-day	5E-03
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	8E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	9E-07	mg/kg-day	2E-05	mg/kg-day	5E-02
				ACENAPHTHYLENE	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	6E-05
				BENZ(A)ANTHRACENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-06	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	7E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	1E-07	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	4E-08	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-06	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	7E-07	(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	5E-04
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								4E-05					6E-02

TABLE 7.12a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	2E-11	mg/kg-day	2E+05	1/(mg/kg-day)	3E-06	2E-10	mg/kg-day	1E-09	mg/kg-day	2E-01
				ALUMINUM	5E+03	mg/kg	3E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	1E+00	mg/kg-day	3E-02
				ARSENIC	6E+00	mg/kg	3E-06	mg/kg-day	2E+00	1/(mg/kg-day)	5E-06	4E-05	mg/kg-day	3E-04	mg/kg-day	1E-01
				CADMIUM	2E+01	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-03	mg/kg-day	1E-01
				CHROMIUM	1E+02	mg/kg	7E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	3E-03	mg/kg-day	3E-01
				COPPER	1E+02	mg/kg	6E-05	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	4E-02	mg/kg-day	2E-02
				IRON	1E+04	mg/kg	7E-03	mg/kg-day		1/(mg/kg-day)		9E-02	mg/kg-day	7E-01	mg/kg-day	1E-01
				MANGANESE	3E+02	mg/kg	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E-01	mg/kg-day	2E-02
				MERCURY	2E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-04	mg/kg-day	4E-02
				VANADIUM	1E+01	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	9E-03	mg/kg-day	1E-02
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	5E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	6E-06	mg/kg-day	2E-05	mg/kg-day	3E-01
				ACENAPHTHYLENE	2E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	4E-04
				BENZ(A)ANTHRACENE	9E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-04	(a)	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-05	(a)	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	5E-04
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	9E-07	(a)	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	3E-07	(a)	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	2E-05	(a)	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	9E-07	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	(a)	mg/kg-day	(a)	1/(mg/kg-day)	5E-06	(a)	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	8E-06	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	3E-02	mg/kg-day	3E-03
				BENZENE	1E-03	mg/kg	5E-10	mg/kg-day	6E-02	1/(mg/kg-day)	3E-11	6E-09	mg/kg-day	4E-03	mg/kg-day	2E-06
					Exp. Route Total								3E-04			
				Exp. Point Total								3E-04				
		Exp. Medium Total									3E-04					1E+00
Medium Total											3E-04				1E+00	
Total of Receptor Risks Across All Media											3E-04	Total of Receptor Hazards Across All Media				1E+00

Notes:
(a) See Table 7.12a CT Supplement A for the intake and toxicity values for COPCs with an MMOA

TABLE 7.12a CT Supplement A
CALCULATION OF CHEMICAL CANCER RISKS FOR COPC WITH MUTAGENIC MODE OF ACTION - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Child Resident
Receptor Age:	0 to < 6 years old

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations						
					Value	Units	Intake(1)			CSF/Unit Risk (2)			Cancer Risk
							Value		Units	Value		Units	
							0-2 yrs	2-6 yrs		0-2 yrs (ADAF=10)	2-6 yrs (ADAF=3)		
Soil	Surface Soil	Exposure Unit 9	Ingestion	Benz(a)anthracene	9.3E+00	mg/kg	2.5E-06	3.2E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
				Benzo(a)pyrene	6.6E+00	mg/kg	1.8E-06	2.3E-06	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-04
				Benzo(b)fluoranthene	9.6E+00	mg/kg	2.6E-06	3.3E-06	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	3.E-05
				Benzo(k)fluoranthene	3.3E+00	mg/kg	8.8E-07	1.1E-06	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	9.E-07
				Chrysene	9.5E+00	mg/kg	2.6E-06	3.2E-06	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	3.E-07
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	1.6E-07	2.0E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-05
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	4.9E-07	6.1E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	5.E-06
			Dermal	Benz(a)anthracene	9.3E+00	mg/kg	3.5E-07	4.8E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	4.E-06
				Benzo(a)pyrene	6.6E+00	mg/kg	2.5E-07	3.4E-07	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	3.E-05
				Benzo(b)fluoranthene	9.6E+00	mg/kg	3.6E-07	4.9E-07	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	4.E-06
				Benzo(k)fluoranthene	3.3E+00	mg/kg	1.2E-07	1.7E-07	mg/kg/day	7.3E-01	2.2E-01	1/(mg/kg-day)	1.E-07
				Chrysene	9.5E+00	mg/kg	3.6E-07	4.9E-07	mg/kg/day	7.3E-02	2.2E-02	1/(mg/kg-day)	4.E-08
				Dibenz(a,h)anthracene	5.9E-01	mg/kg	2.2E-08	3.0E-08	mg/kg/day	7.3E+01	2.2E+01	1/(mg/kg-day)	2.E-06
				Indeno(1,2,3-cd)pyrene	1.8E+00	mg/kg	6.7E-08	9.3E-08	mg/kg/day	7.3E+00	2.2E+00	1/(mg/kg-day)	7.E-07
	Fugitive Dust	Exposure Unit 9	Inhalation	Benz(a)anthracene	2.7E-08	mg/m ³	4.1E-10	7.2E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(a)pyrene	1.9E-08	mg/m ³	2.9E-10	5.1E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(b)fluoranthene	2.8E-08	mg/m ³	4.2E-10	7.3E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Benzo(k)fluoranthene	9.4E-09	mg/m ³	1.4E-10	2.5E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Chrysene	2.7E-08	mg/m ³	4.2E-10	7.3E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Dibenz(a,h)anthracene	1.7E-09	mg/m ³	2.6E-11	4.5E-11	mg/kg/day	NA	NA	1/(mg/kg-day)	NA
				Indeno(1,2,3-cd)pyrene	5.2E-09	mg/m ³	7.9E-11	1.4E-10	mg/kg/day	NA	NA	1/(mg/kg-day)	NA

(1) - Intake equations derived from Table 4 series: Supplement A - Values Used for Daily Intake Calculations (mutagenic mode of action)

(2) - Cancer slope factor/unit risk (CSF/Unit Risk) derived from Table 6 series and adjusted using Age Dependent Adjustment Factors (ADAF) in accordance with the 2006 USEPA Memorandum.

Source: EPA Memorandum dated 14 June 2006: Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include

TABLE 7.13 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations						
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient		
							Value	Units	Value	Units		Value	Units	Value	Units			
Surface Soil	Outdoor Air	Exposure Unit 6	Inhalation	2,3,7,8-TCDD Equivalent	1E-07	mg/m3	3E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day		mg/kg-day			
				ALUMINUM	2E-06	mg/m3	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	1E-03	mg/kg-day	2E-04		
				ARSENIC	2E-09	mg/m3	5E-11	mg/kg-day	2E+01	1/(mg/kg-day)	7E-10	4E-10	mg/kg-day	1E-05	mg/kg-day	3E-05		
				BARIUM	9E-08	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		2E-08	mg/kg-day	1E-04	mg/kg-day	1E-04		
				CADMIUM	9E-09	mg/m3	2E-10	mg/kg-day	6E+00	1/(mg/kg-day)	1E-09	2E-09	mg/kg-day		mg/kg-day			
				CHROMIUM	3E-08	mg/m3	7E-10	mg/kg-day	4E+01	1/(mg/kg-day)	3E-08	5E-09	mg/kg-day	3E-05	mg/kg-day	2E-04		
				COPPER	6E-08	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day		mg/kg-day			
				IRON	3E-06	mg/m3	7E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day		mg/kg-day			
				LEAD	2E-07	mg/m3	4E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day		mg/kg-day			
				MANGANESE	8E-08	mg/m3	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	1E-05	mg/kg-day	9E-04		
				MERCURY	3E-09	mg/m3	7E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day	9E-05	mg/kg-day	6E-06		
				SILVER	4E-09	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day			
				THALLIUM	2E-10	mg/m3	5E-12	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day			
				VANADIUM	5E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day			
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	9E-12	mg/kg-day	2E+00	1/(mg/kg-day)	2E-11	7E-11	mg/kg-day		mg/kg-day			
				LESS CHLORINATED PCBs	2E-10	mg/m3	4E-12	mg/kg-day	2E+00	1/(mg/kg-day)	8E-12	3E-11	mg/kg-day		mg/kg-day			
				DIELDRIN	3E-11	mg/m3	6E-13	mg/kg-day	2E+01	1/(mg/kg-day)	1E-11	5E-12	mg/kg-day		mg/kg-day			
				2-METHYLNAPHTHALENE	3E-09	mg/m3	7E-11	mg/kg-day		1/(mg/kg-day)		6E-10	mg/kg-day		mg/kg-day			
				ACENAPHTHYLENE	1E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				BENZ(A)ANTHRACENE	2E-09	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				BENZO(A)PYRENE	2E-09	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day		mg/kg-day			
				BENZO(B)FLUORANTHENE	2E-09	mg/m3	4E-11	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		3E-10	mg/kg-day		mg/kg-day			
				CHRYSENE	2E-09	mg/m3	5E-11	mg/kg-day		1/(mg/kg-day)		4E-10	mg/kg-day		mg/kg-day			
				DIBENZ(A,H)ANTHRACENE	4E-10	mg/m3	9E-12	mg/kg-day		1/(mg/kg-day)		7E-11	mg/kg-day		mg/kg-day			
				DIBENZOFURAN	1E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				HEXACHLOROBEZENE	2E-10	mg/m3	5E-12	mg/kg-day	2E+00	1/(mg/kg-day)	9E-12	4E-11	mg/kg-day		mg/kg-day			
				INDENO(1,2,3-CD)PYRENE	1E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day			
				NAPHTHALENE	6E-09	mg/m3	1E-10	mg/kg-day	1E-01	1/(mg/kg-day)	2E-11	1E-09	mg/kg-day	9E-04	mg/kg-day	1E-06		
				PHENANTHRENE	5E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		8E-10	mg/kg-day		mg/kg-day			
				1,2,3-TRICHLOROBENZENE	1E-04	mg/m3	3E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day		mg/kg-day			
				1,2,4-TRICHLOROBENZENE	1E-04	mg/m3	3E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day		mg/kg-day			
				1,2-DICHLOROBENZENE	9E-04	mg/m3	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	4E-03		
				1,4-DICHLOROBENZENE	4E-03	mg/m3	8E-05	mg/kg-day	4E-02	1/(mg/kg-day)	3E-06	6E-04	mg/kg-day	2E-01	mg/kg-day	3E-03		
				BENZENE	3E-04	mg/m3	7E-06	mg/kg-day	3E-02	1/(mg/kg-day)	2E-07	5E-05	mg/kg-day	9E-03	mg/kg-day	6E-03		
				P-ISOPROPYLTOLUENE		mg/m3		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				DODECANE	2E-07	mg/m3	5E-09	mg/kg-day		1/(mg/kg-day)		4E-08	mg/kg-day		mg/kg-day			
								Exp. Route Total						3E-06				1E-02
								Exp. Point Total						3E-06				1E-02
								Exp. Medium Total						3E-06				1E-02
				Medium Total										3E-06				1E-02
Soil	Surface Soil	Exposure Unit 6	Dermal	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	2E-12	mg/kg-day	2E+05	1/(mg/kg-day)	2E-07	1E-11	mg/kg-day	1E-09	mg/kg-day	1E-02		
				ALUMINUM	7E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day			
				ARSENIC	8E+00	mg/kg	3E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	2E-07	mg/kg-day	3E-04	mg/kg-day	7E-04		
				BARIUM	4E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day			
				CADMIUM	4E+01	mg/kg	4E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	3E-05	mg/kg-day	1E-03		
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				COPPER	2E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day			
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day			
				LEAD	7E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day			
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day			
				MERCURY	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day			
				SILVER	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				THALLIUM	8E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day			
				VANADIUM	2E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day			
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	4E-08	2E-07	mg/kg-day	2E-05	mg/kg-day	8E-03		
				LESS CHLORINATED PCBs	7E-01	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-08	8E-08	mg/kg-day	7E-05	mg/kg-day	1E-03		

TABLE 7.13 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Dermal	DIELDRIN	1E-01	mg/kg		mg/kg-day	2E+01	1/(mg/kg-day)			mg/kg-day	5E-05	mg/kg-day	
				2-METHYLNAPHTHALENE	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	4E-03	mg/kg-day	3E-04
				ACENAPHTHYLENE	4E+00	mg/kg	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	3E-02	mg/kg-day	1E-05
				BENZ(A)ANTHRACENE	8E+00	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	7E-08	8E-07	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	1E-07	mg/kg-day	7E+00	1/(mg/kg-day)	9E-07	9E-07	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	9E-08	mg/kg-day	7E-01	1/(mg/kg-day)	6E-08	7E-07	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	3E-02	mg/kg-day	2E-05
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	7E-08	mg/kg-day	7E-02	1/(mg/kg-day)	5E-09	6E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	1E-07	mg/kg-day	7E-03	1/(mg/kg-day)	8E-10	8E-07	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	2E-08	mg/kg-day	7E+00	1/(mg/kg-day)	2E-07	2E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day	1E-03	mg/kg-day	3E-04
				HEXACHLOROBENZENE	1E+00	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-08	7E-08	mg/kg-day	8E-04	mg/kg-day	9E-05
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	6E-08	mg/kg-day	7E-01	1/(mg/kg-day)	4E-08	5E-07	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	2E-02	mg/kg-day	1E-04
				PHENANTHRENE	2E+01	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	6E-05
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg		mg/kg-day	4E-03	1/(mg/kg-day)			mg/kg-day	1E-02	mg/kg-day	
				1,2-DICHLOROBENZENE	8E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-02	mg/kg-day	
				1,4-DICHLOROBENZENE	3E+01	mg/kg		mg/kg-day	5E-03	1/(mg/kg-day)			mg/kg-day	7E-02	mg/kg-day	
				BENZENE	5E-01	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				P-ISOPROPYLTOLUENE	4E-01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
			Exp. Route Total								2E-06					2E-02
			Ingestion	2,3,7,8-TCDD Equivalent	5E-04	mg/kg	5E-11	mg/kg-day	2E+05	1/(mg/kg-day)	7E-06	4E-10	mg/kg-day	1E-09	mg/kg-day	4E-01
				ALUMINUM	7E+03	mg/kg	6E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	1E+00	mg/kg-day	5E-03
				ARSENIC	8E+00	mg/kg	7E-07	mg/kg-day	2E+00	1/(mg/kg-day)	1E-06	6E-06	mg/kg-day	3E-04	mg/kg-day	2E-02
				BARIUM	4E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	2E-01	mg/kg-day	1E-03
				CADMIUM	4E+01	mg/kg	3E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	1E-03	mg/kg-day	2E-02
				CHROMIUM	1E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	3E-03	mg/kg-day	3E-02
				COPPER	2E+02	mg/kg	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	4E-02	mg/kg-day	4E-03
				IRON	1E+04	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day	7E-01	mg/kg-day	1E-02
				LEAD	7E+02	mg/kg	6E-05	mg/kg-day		1/(mg/kg-day)		5E-04	mg/kg-day		mg/kg-day	
				MANGANESE	3E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	1E-03
				MERCURY	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day	3E-04	mg/kg-day	3E-02
				SILVER	2E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	5E-03	mg/kg-day	2E-03
				THALLIUM	8E-01	mg/kg	7E-08	mg/kg-day		1/(mg/kg-day)		5E-07	mg/kg-day	8E-05	mg/kg-day	7E-03
				VANADIUM	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	2E-03
				HIGHLY CHLORINATED PCBs	2E+00	mg/kg	1E-07	mg/kg-day	2E+00	1/(mg/kg-day)	3E-07	1E-06	mg/kg-day	2E-05	mg/kg-day	5E-02
				LESS CHLORINATED PCBs	7E-01	mg/kg	6E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	5E-07	mg/kg-day	7E-05	mg/kg-day	7E-03
				DIELDRIN	1E-01	mg/kg	1E-08	mg/kg-day	2E+01	1/(mg/kg-day)	2E-07	8E-08	mg/kg-day	5E-05	mg/kg-day	2E-03
				2-METHYLNAPHTHALENE	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	4E-03	mg/kg-day	2E-03
				ACENAPHTHYLENE	4E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	3E-02	mg/kg-day	9E-05
				BENZ(A)ANTHRACENE	8E+00	mg/kg	7E-07	mg/kg-day	7E-01	1/(mg/kg-day)	5E-07	5E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	9E+00	mg/kg	8E-07	mg/kg-day	7E+00	1/(mg/kg-day)	6E-06	6E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	7E+00	mg/kg	6E-07	mg/kg-day	7E-01	1/(mg/kg-day)	4E-07	5E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	mg/kg	5E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-02	mg/kg-day	1E-04
				BENZO(K)FLUORANTHENE	6E+00	mg/kg	5E-07	mg/kg-day	7E-02	1/(mg/kg-day)	4E-08	4E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	8E+00	mg/kg	7E-07	mg/kg-day	7E-03	1/(mg/kg-day)	5E-09	5E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	2E+00	mg/kg	1E-07	mg/kg-day	7E+00	1/(mg/kg-day)	1E-06	1E-06	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	4E+00	mg/kg	4E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	1E-03	mg/kg-day	3E-03
				HEXACHLOROBENZENE	1E+00	mg/kg	8E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	7E-07	mg/kg-day	8E-04	mg/kg-day	8E-04
				INDENO(1,2,3-CD)PYRENE	5E+00	mg/kg	4E-07	mg/kg-day	7E-01	1/(mg/kg-day)	3E-07	3E-06	mg/kg-day		mg/kg-day	
				NAPHTHALENE	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-02	mg/kg-day	8E-04
				PHENANTHRENE	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	4E-04
				1,2,3-TRICHLOROBENZENE	4E+00	mg/kg	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	4E+00	mg/kg	4E-07	mg/kg-day	4E-03	1/(mg/kg-day)	1E-09	3E-06	mg/kg-day	1E-02	mg/kg-day	3E-04
				1,2-DICHLOROBENZENE	8E+00	mg/kg	7E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	9E-02	mg/kg-day	6E-05
				1,4-DICHLOROBENZENE	3E+01	mg/kg	3E-06	mg/kg-day	5E-03	1/(mg/kg-day)	1E-08	2E-05	mg/kg-day	7E-02	mg/kg-day	3E-04

TABLE 7.13 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 6	Ingestion	BENZENE	5E-01	mg/kg	5E-08	mg/kg-day	6E-02	1/(mg/kg-day)	3E-09	4E-07	mg/kg-day	4E-03	mg/kg-day	9E-05
				P-ISOPROPYLTOLUENE	4E-01	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day	
				DODECANE	8E+02	mg/kg	7E-05	mg/kg-day		1/(mg/kg-day)		6E-04	mg/kg-day		mg/kg-day	
				Exp. Route Total						2E-05				6E-01		
		Exp. Point Total						2E-05				6E-01				
	Exp. Medium Total						2E-05				6E-01					
Medium Total												2E-05				6E-01
Ground Water	Potable Water	Exposure Unit 8	Dermal	ALUMINUM	2E+04	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E+00	mg/kg-day	2E-03
				ANTIMONY	2E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	6E-05	mg/kg-day	2E-03
				ARSENIC	9E+00	ug/l	7E-08	mg/kg-day	2E+00	1/(mg/kg-day)	1E-07	6E-07	mg/kg-day	3E-04	mg/kg-day	2E-03
				BARIUM	1E+03	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	1E-02	mg/kg-day	7E-03
				BERYLLIUM	8E-01	ug/l	6E-09	mg/kg-day		1/(mg/kg-day)		5E-08	mg/kg-day	1E-05	mg/kg-day	4E-03
				CADMIUM	2E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	3E-05	mg/kg-day	5E-03
				CHROMIUM	7E+01	ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		9E-06	mg/kg-day	8E-05	mg/kg-day	1E-01
				COBALT	1E+01	ug/l	4E-08	mg/kg-day		1/(mg/kg-day)		3E-07	mg/kg-day		mg/kg-day	
				COPPER	9E+01	ug/l	7E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day	4E-02	mg/kg-day	1E-04
				CYANIDE	3E+01	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	2E-02	mg/kg-day	9E-05
				IRON	4E+04	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	7E-01	mg/kg-day	3E-03
				LEAD	6E+01	ug/l	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day		mg/kg-day	
				MANGANESE	2E+03	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	6E-03	mg/kg-day	2E-02
				MERCURY	2E+00	ug/l	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	2E-05	mg/kg-day	6E-03
				NICKEL	5E+01	ug/l	8E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	8E-04	mg/kg-day	8E-04
				SELENIUM	4E+00	ug/l	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	5E-03	mg/kg-day	4E-05
				SILVER	2E+00	ug/l	1E-08	mg/kg-day		1/(mg/kg-day)		8E-08	mg/kg-day	2E-04	mg/kg-day	4E-04
				THALLIUM	7E+00	ug/l	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day	8E-05	mg/kg-day	5E-03
				VANADIUM	4E+01	ug/l	3E-07	mg/kg-day		1/(mg/kg-day)		3E-06	mg/kg-day	2E-04	mg/kg-day	1E-02
				ZINC	1E+02	ug/l	5E-07	mg/kg-day		1/(mg/kg-day)		4E-06	mg/kg-day	3E-01	mg/kg-day	1E-05
				HIGHLY CHLORINATED PCBs	7E-02	ug/l		mg/kg-day	2E+00	1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				4,4'-DDD	9E-02	ug/l	1E-06	mg/kg-day	2E-01	1/(mg/kg-day)	3E-07	1E-05	mg/kg-day		mg/kg-day	
				4,4'-DDT	1E+00	ug/l	3E-05	mg/kg-day	3E-01	1/(mg/kg-day)	1E-05	2E-04	mg/kg-day	5E-04	mg/kg-day	5E-01
				ALDRIN	3E-02	ug/l	7E-09	mg/kg-day	2E+01	1/(mg/kg-day)	1E-07	6E-08	mg/kg-day	3E-05	mg/kg-day	2E-03
				ALPHA-BHC	2E-01	ug/l		mg/kg-day	6E+00	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				ENDOSULFAN II	6E-02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				ENDOSULFAN SULFATE	2E-02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				HEPTACHLOR EPOXIDE	1E-02	ug/l		mg/kg-day	9E+00	1/(mg/kg-day)			mg/kg-day	1E-05	mg/kg-day	
				1,1'-BIPHENYL	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	5E-02	mg/kg-day	
				2,4-DICHLOROPHENOL	1E+01	ug/l	8E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	3E-03	mg/kg-day	2E-02
				2,4-DIMETHYLPHENOL	4E+03	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	2E-02	mg/kg-day	5E-01
				2-METHYLNAPHTHALENE	6E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
				2-METHYLPHENOL	1E+03	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	5E-02	mg/kg-day	3E-02
				2-NITROPHENOL	6E+00	ug/l	8E-07	mg/kg-day		1/(mg/kg-day)		6E-06	mg/kg-day		mg/kg-day	
				3&4-METHYLPHENOL	4E+03	ug/l	9E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	5E-02	mg/kg-day	1E-01
				4-CHLORO-3-METHYLPHENOL	1E+00	ug/l	1E-06	mg/kg-day		1/(mg/kg-day)		8E-06	mg/kg-day		mg/kg-day	
				4-METHYLPHENOL	8E+03	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	5E-02	mg/kg-day	3E-01
				4-NITROPHENOL	1E+01	ug/l	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day		mg/kg-day	
				ACENAPHTHENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-02	mg/kg-day	
				ACENAPHTHYLENE	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				ANTHRACENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-01	mg/kg-day	
				ATRAZINE	5E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				BENZ(A)ANTHRACENE	5E+01	ug/l	2E-03	mg/kg-day	7E-01	1/(mg/kg-day)	1E-03	1E-02	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+01	ug/l	1E-03	mg/kg-day	7E+00	1/(mg/kg-day)	7E-03	8E-03	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E+01	ug/l	1E-03	mg/kg-day	7E-01	1/(mg/kg-day)	8E-04	8E-03	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				BENZO(K)FLUORANTHENE	2E+01	ug/l		mg/kg-day	7E-02	1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	4E-05	mg/kg-day	1E-02	1/(mg/kg-day)	5E-07	3E-04	mg/kg-day	2E-02	mg/kg-day	1E-02
				CARBAZOLE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				CHRYSENE	4E+01	ug/l	1E-03	mg/kg-day	7E-03	1/(mg/kg-day)	8E-06	8E-03	mg/kg-day		mg/kg-day	

TABLE 7.13 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Potable Water	Exposure Unit 8	Dermal	DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	2E-04	mg/kg-day	7E+00	1/(mg/kg-day)	2E-03	2E-03	mg/kg-day	1E-03	mg/kg-day	4E-01
				DIBENZOFURAN	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				FLUORANTHENE	2E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	4E-02	mg/kg-day	
				FLUORENE	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				HEXACHLOROBUTADIENE	1E+00	ug/l	6E-06	mg/kg-day	8E-02	1/(mg/kg-day)	4E-07	4E-05	mg/kg-day	2E-02	mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	4E-04	mg/kg-day	7E-01	1/(mg/kg-day)	3E-04	3E-03	mg/kg-day	5E-04	mg/kg-day	
				NAPHTHALENE	4E+03	ug/l	6E-03	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	3E-02	mg/kg-day	
				NITROBENZENE	3E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	5E-04	mg/kg-day	
				PHENANTHRENE	4E+02	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	3E-02	mg/kg-day	
				PHENOL	2E+03	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-01	mg/kg-day	
				PYRENE	1E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	3E-02	mg/kg-day	
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	4E-05	mg/kg-day	4E-03	1/(mg/kg-day)	1E-07	3E-04	mg/kg-day	1E-02	mg/kg-day	
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,2-DICHLOROBENZENE	5E+02	ug/l	8E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day	9E-02	mg/kg-day	
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				1,3-DICHLOROBENZENE	5E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day		mg/kg-day	
				1,4-DICHLOROBENZENE	5E+02	ug/l	7E-04	mg/kg-day	5E-03	1/(mg/kg-day)	4E-06	6E-03	mg/kg-day	7E-02	mg/kg-day	
				2-HEXANONE	2E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
				ACETONE	8E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	9E-01	mg/kg-day	
				BENZENE	6E+03	ug/l	2E-03	mg/kg-day	6E-02	1/(mg/kg-day)	1E-04	2E-02	mg/kg-day	4E-03	mg/kg-day	
				BROMODICHLOROMETHANE	3E+00	ug/l	6E-07	mg/kg-day	6E-02	1/(mg/kg-day)	4E-08	4E-06	mg/kg-day	2E-02	mg/kg-day	
				CARBON DISULFIDE	1E+01	ug/l	5E-06	mg/kg-day		1/(mg/kg-day)		4E-05	mg/kg-day	1E-01	mg/kg-day	
				CHLOROBENZENE	2E+02	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-02	mg/kg-day	
				CHLOROETHANE	5E+00	ug/l	6E-07	mg/kg-day		1/(mg/kg-day)		5E-06	mg/kg-day		mg/kg-day	
				ETHYLBENZENE	1E+02	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	1E-01	mg/kg-day	
				ISOPROPYLBENZENE	4E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E-01	mg/kg-day	
				METHYLENE CHLORIDE	7E-01	ug/l	6E-08	mg/kg-day	8E-03	1/(mg/kg-day)	5E-10	5E-07	mg/kg-day	6E-02	mg/kg-day	
				P-ISOPROPYLTOLUENE	3E+00	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				SEC-BUTYLBENZENE	1E+01	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day		mg/kg-day	
				STYRENE	8E+02	ug/l	9E-04	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day	2E-01	mg/kg-day	
				TETRACHLOROETHENE	3E-01	ug/l	4E-07	mg/kg-day	5E-01	1/(mg/kg-day)	2E-07	3E-06	mg/kg-day	1E-02	mg/kg-day	
				TOLUENE	1E+03	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		8E-03	mg/kg-day	8E-02	mg/kg-day	
				VINYL CHLORIDE	1E+00	ug/l	1E-07	mg/kg-day	8E-01	1/(mg/kg-day)	1E-07	1E-06	mg/kg-day	3E-03	mg/kg-day	
				XYLENES, TOTAL	1E+03	ug/l		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-01	mg/kg-day	
			Exp. Route Total								1E-02					1E+01
			Ingestion	ALUMINUM	2E+04	ug/l	9E-02	mg/kg-day		1/(mg/kg-day)		7E-01	mg/kg-day	1E+00	mg/kg-day	7E-01
				ANTIMONY	2E+00	ug/l	8E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	4E-04	mg/kg-day	2E-01
				ARSENIC	9E+00	ug/l	3E-05	mg/kg-day	2E+00	1/(mg/kg-day)	5E-05	3E-04	mg/kg-day	3E-04	mg/kg-day	9E-01
				BARIUM	1E+03	ug/l	5E-03	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	2E-01	mg/kg-day	2E-01
				BERYLLIUM	8E-01	ug/l	3E-06	mg/kg-day		1/(mg/kg-day)		2E-05	mg/kg-day	2E-03	mg/kg-day	1E-02
				CADMIUM	2E+00	ug/l	7E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	1E-03	mg/kg-day	5E-02
				CHROMIUM	7E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	3E-03	mg/kg-day	6E-01
				COBALT	1E+01	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day	
				COPPER	9E+01	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	4E-02	mg/kg-day	6E-02
				CYANIDE	3E+01	ug/l	1E-04	mg/kg-day		1/(mg/kg-day)		8E-04	mg/kg-day	2E-02	mg/kg-day	4E-02
				IRON	4E+04	ug/l	1E-01	mg/kg-day		1/(mg/kg-day)		1E+00	mg/kg-day	7E-01	mg/kg-day	2E+00
				LEAD	6E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day		mg/kg-day	
				MANGANESE	2E+03	ug/l	6E-03	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	1E-01	mg/kg-day	4E-01
				MERCURY	2E+00	ug/l	8E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	3E-04	mg/kg-day	2E-01
				NICKEL	5E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	2E-02	mg/kg-day	7E-02
				SELENIUM	4E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	5E-03	mg/kg-day	2E-02
				SILVER	2E+00	ug/l	8E-06	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	5E-03	mg/kg-day	1E-02
				THALLIUM	7E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	8E-05	mg/kg-day	2E+00
				VANADIUM	4E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	9E-03	mg/kg-day	1E-01
				ZINC	1E+02	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-01	mg/kg-day	9E-03
				HIGHLY CHLORINATED PCBs	7E-02	ug/l	2E-07	mg/kg-day	2E+00	1/(mg/kg-day)	5E-07	2E-06	mg/kg-day	2E-05	mg/kg-day	1E-01
				4,4'-DDD	9E-02	ug/l	3E-07	mg/kg-day	2E-01	1/(mg/kg-day)	7E-08	2E-06	mg/kg-day		mg/kg-day	
				4,4'-DDT	1E+00	ug/l	4E-06	mg/kg-day	3E-01	1/(mg/kg-day)	1E-06	3E-05	mg/kg-day	5E-04	mg/kg-day	6E-02

TABLE 7.13 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ground Water	Potable Water	Exposure Unit 8	Ingestion	ALDRIN	3E-02	ug/l	1E-07	mg/kg-day	2E+01	1/(mg/kg-day)	2E-06	9E-07	mg/kg-day	3E-05	mg/kg-day	3E-02
				ALPHA-BHC	2E-01	ug/l	7E-07	mg/kg-day	6E+00	1/(mg/kg-day)	4E-06	5E-06	mg/kg-day		mg/kg-day	
				ENDOSULFAN II	6E-02	ug/l	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	6E-03	mg/kg-day	3E-04
				ENDOSULFAN SULFATE	2E-02	ug/l	7E-08	mg/kg-day		1/(mg/kg-day)		6E-07	mg/kg-day	6E-03	mg/kg-day	1E-04
				HEPTACHLOR EPOXIDE	1E-02	ug/l	4E-08	mg/kg-day	9E+00	1/(mg/kg-day)	3E-07	3E-07	mg/kg-day	1E-05	mg/kg-day	2E-02
				1,1'-BIPHENYL	1E+01	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	5E-02	mg/kg-day	7E-03
				2,4-DICHLOROPHENOL	1E+01	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	3E-03	mg/kg-day	9E-02
				2,4-DIMETHYLPHENOL	4E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	2E-02	mg/kg-day	6E+00
				2-METHYLNAPHTHALENE	6E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	4E-03	mg/kg-day	4E+00
				2-METHYLPHENOL	1E+03	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	5E-02	mg/kg-day	5E-01
				2-NITROPHENOL	6E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day	
				3&4-METHYLPHENOL	4E+03	ug/l	2E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	5E-02	mg/kg-day	2E+00
				4-CHLORO-3-METHYLPHENOL	1E+00	ug/l	4E-06	mg/kg-day		1/(mg/kg-day)		3E-05	mg/kg-day		mg/kg-day	
				4-METHYLPHENOL	8E+03	ug/l	3E-02	mg/kg-day		1/(mg/kg-day)		2E-01	mg/kg-day	5E-02	mg/kg-day	5E+00
				4-NITROPHENOL	1E+01	ug/l	3E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day	
				ACENAPHTHENE	1E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	6E-02	mg/kg-day	5E-02
				ACENAPHTHYLENE	2E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	3E-02	mg/kg-day	2E-01
				ANTHRACENE	1E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-01	mg/kg-day	1E-02
				ATRAZINE	5E+01	ug/l	2E-04	mg/kg-day		1/(mg/kg-day)		1E-03	mg/kg-day	4E-02	mg/kg-day	4E-02
				BENZ(A)ANTHRACENE	5E+01	ug/l	2E-04	mg/kg-day	7E-01	1/(mg/kg-day)	1E-04	1E-03	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	2E+01	ug/l	7E-05	mg/kg-day	7E+00	1/(mg/kg-day)	5E-04	5E-04	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	2E+01	ug/l	8E-05	mg/kg-day	7E-01	1/(mg/kg-day)	5E-05	6E-04	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	5E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E-02	mg/kg-day	5E-03
				BENZO(K)FLUORANTHENE	2E+01	ug/l	6E-05	mg/kg-day	7E-02	1/(mg/kg-day)	5E-06	5E-04	mg/kg-day		mg/kg-day	
				BIS(2-ETHYLHEXYL)PHTHALATE	1E+01	ug/l	4E-05	mg/kg-day	1E-02	1/(mg/kg-day)	5E-07	3E-04	mg/kg-day	2E-02	mg/kg-day	1E-02
				CARBAZOLE	1E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day		mg/kg-day	
				CHRYSENE	4E+01	ug/l	1E-04	mg/kg-day	7E-03	1/(mg/kg-day)	9E-07	1E-03	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E+00	ug/l	1E-05	mg/kg-day	7E+00	1/(mg/kg-day)	7E-05	8E-05	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+02	ug/l	7E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	1E-03	mg/kg-day	5E+00
				FLUORANTHENE	2E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	4E-02	mg/kg-day	1E-01
				FLUORENE	2E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	4E-02	mg/kg-day	1E-01
				HEXACHLOROBUTADIENE	1E+00	ug/l	4E-06	mg/kg-day	8E-02	1/(mg/kg-day)	3E-07	3E-05	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E+00	ug/l	3E-05	mg/kg-day	7E-01	1/(mg/kg-day)	2E-05	2E-04	mg/kg-day		mg/kg-day	
				NAPHTHALENE	4E+03	ug/l	1E-02	mg/kg-day		1/(mg/kg-day)		1E-01	mg/kg-day	2E-02	mg/kg-day	5E+00
				NITROBENZENE	3E+00	ug/l	9E-06	mg/kg-day		1/(mg/kg-day)		7E-05	mg/kg-day	5E-04	mg/kg-day	1E-01
				PHENANTHRENE	4E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	3E-02	mg/kg-day	4E-01
				PHENOL	2E+03	ug/l	7E-03	mg/kg-day		1/(mg/kg-day)		5E-02	mg/kg-day	3E-01	mg/kg-day	2E-01
				PYRENE	1E+02	ug/l	4E-04	mg/kg-day		1/(mg/kg-day)		3E-03	mg/kg-day	3E-02	mg/kg-day	9E-02
				1,2,3-TRICHLOROBENZENE	1E+01	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day	
				1,2,4-TRICHLOROBENZENE	1E+01	ug/l	5E-05	mg/kg-day	4E-03	1/(mg/kg-day)	2E-07	4E-04	mg/kg-day	1E-02	mg/kg-day	4E-02
				1,2,4-TRIMETHYLBENZENE	3E+02	ug/l	1E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day		mg/kg-day	
				1,2-DICHLOROBENZENE	5E+02	ug/l	2E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	9E-02	mg/kg-day	2E-01
				1,3,5-TRIMETHYLBENZENE	2E+02	ug/l	8E-04	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day		mg/kg-day	
				1,3-DICHLOROBENZENE	5E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day	
				1,4-DICHLOROBENZENE	5E+02	ug/l	2E-03	mg/kg-day	5E-03	1/(mg/kg-day)	9E-06	1E-02	mg/kg-day	7E-02	mg/kg-day	2E-01
				2-HEXANONE	2E+00	ug/l	7E-06	mg/kg-day		1/(mg/kg-day)		5E-05	mg/kg-day	2E-01	mg/kg-day	3E-04
				ACETONE	8E+01	ug/l	3E-04	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	9E-01	mg/kg-day	2E-03
				BENZENE	6E+03	ug/l	2E-02	mg/kg-day	6E-02	1/(mg/kg-day)	1E-03	2E-01	mg/kg-day	4E-03	mg/kg-day	4E+01
				BROMODICHLOROMETHANE	3E+00	ug/l	1E-05	mg/kg-day	6E-02	1/(mg/kg-day)	7E-07	8E-05	mg/kg-day	2E-02	mg/kg-day	4E-03
				CARBON DISULFIDE	1E+01	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day	1E-01	mg/kg-day	3E-03
				CHLOROBENZENE	2E+02	ug/l	6E-04	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	2E-02	mg/kg-day	2E-01
				CHLOROETHANE	5E+00	ug/l	2E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day	
				ETHYLBENZENE	1E+02	ug/l	5E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	1E-01	mg/kg-day	4E-02
				ISOPROPYLBENZENE	4E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-01	mg/kg-day	1E-03
				METHYLENE CHLORIDE	7E-01	ug/l	3E-06	mg/kg-day	8E-03	1/(mg/kg-day)	2E-08	2E-05	mg/kg-day	6E-02	mg/kg-day	3E-04
				P-ISOPROPYLTOLUENE	3E+00	ug/l	1E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day		mg/kg-day	
				SEC-BUTYLBENZENE	1E+01	ug/l	4E-05	mg/kg-day		1/(mg/kg-day)		3E-04	mg/kg-day		mg/kg-day	
				STYRENE	8E+02	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	2E-01	mg/kg-day	1E-01

TABLE 7.13 CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations									
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RID/RIC		Hazard Quotient					
							Value	Units	Value	Units		Value	Units	Value	Units						
Ground Water	Potable Water	Exposure Unit 8	Ingestion	TETRACHLOROETHENE	3E-01	ug/l	1E-06	mg/kg-day	5E-01	1/(mg/kg-day)	6E-07	8E-06	mg/kg-day	1E-02	mg/kg-day	8E-04					
				TOLUENE	1E+03	ug/l	4E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	8E-02	mg/kg-day	4E-01					
				VINYL CHLORIDE	1E+00	ug/l	4E-06	mg/kg-day	8E-01	1/(mg/kg-day)	3E-06	3E-05	mg/kg-day	3E-03	mg/kg-day	1E-02					
				XYLENES, TOTAL	1E+03	ug/l	3E-03	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	2E-01	mg/kg-day	1E-01					
		Exp. Route Total								2E-03				8E+01							
		Exp. Point Total								1E-02				9E+01							
		Exp. Medium Total								1E-02				9E+01							
	Shower Vapor	Exposure Unit 8	Inhalation	1,2,3-TRICHLOROBENZENE	6E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day		mg/kg-day						
				1,2,4-TRICHLOROBENZENE	7E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day		mg/kg-day						
				1,2,4-TRIMETHYLBENZENE	2E+00	mg/m3	4E-03	mg/kg-day		1/(mg/kg-day)		1E-02	mg/kg-day	2E-03	mg/kg-day	5E+00					
				1,2-DICHLOROBENZENE	3E+00	mg/m3	7E-03	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	4E-02	mg/kg-day	4E-01					
				1,3,5-TRIMETHYLBENZENE	1E+00	mg/m3	3E-03	mg/kg-day		1/(mg/kg-day)		7E-03	mg/kg-day		mg/kg-day						
				1,3-DICHLOROBENZENE	3E-02	mg/m3	7E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day		mg/kg-day						
				1,4-DICHLOROBENZENE	2E+00	mg/m3	6E-03	mg/kg-day	4E-02	1/(mg/kg-day)	3E-04	1E-02	mg/kg-day	2E-01	mg/kg-day	7E-02					
				2-HEXANONE	1E-02	mg/m3	3E-05	mg/kg-day		1/(mg/kg-day)		6E-05	mg/kg-day	6E-02	mg/kg-day	1E-03					
				ACETONE	4E-01	mg/m3	1E-03	mg/kg-day		1/(mg/kg-day)		2E-03	mg/kg-day	9E+00	mg/kg-day	3E-04					
				BENZENE	3E+01	mg/m3	8E-02	mg/kg-day	3E-02	1/(mg/kg-day)	2E-03	2E-01	mg/kg-day	9E-03	mg/kg-day	2E+01					
				BROMODICHLOROMETHANE	2E-02	mg/m3	4E-05	mg/kg-day	1E-01	1/(mg/kg-day)	5E-06	1E-04	mg/kg-day		mg/kg-day						
				CARBON DISULFIDE	6E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day	2E-01	mg/kg-day	2E-03					
				CHLOROBENZENE	9E-01	mg/m3	2E-03	mg/kg-day		1/(mg/kg-day)		6E-03	mg/kg-day		mg/kg-day						
				CHLOROETHANE	2E-02	mg/m3	6E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	3E+00	mg/kg-day	5E-05					
				CHLOROFORM	6E-02	mg/m3	2E-04	mg/kg-day	8E-02	1/(mg/kg-day)	1E-05	4E-04	mg/kg-day	3E-02	mg/kg-day	1E-02					
ETHYLBENZENE	7E-01	mg/m3	2E-03	mg/kg-day		1/(mg/kg-day)		5E-03	mg/kg-day	3E-01	mg/kg-day	2E-02									
ISOPROPYLBENZENE	2E-02	mg/m3	5E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day	1E-01	mg/kg-day	1E-03									
METHYLENE CHLORIDE	4E-03	mg/m3	1E-05	mg/kg-day	2E-03	1/(mg/kg-day)	2E-08	2E-05	mg/kg-day	3E-01	mg/kg-day	8E-05									
P-ISOPROPYLTOLUENE	2E-02	mg/m3	5E-05	mg/kg-day		1/(mg/kg-day)		1E-04	mg/kg-day		mg/kg-day										
SEC-BUTYLBENZENE	6E-02	mg/m3	2E-04	mg/kg-day		1/(mg/kg-day)		4E-04	mg/kg-day		mg/kg-day										
STYRENE	4E+00	mg/m3	1E-02	mg/kg-day		1/(mg/kg-day)		3E-02	mg/kg-day	3E+00	mg/kg-day	9E-03									
TETRACHLOROETHENE	1E-03	mg/m3	4E-06	mg/kg-day	2E-05	1/(mg/kg-day)	8E-11	9E-06	mg/kg-day	8E-02	mg/kg-day	1E-04									
TOLUENE	6E+00	mg/m3	2E-02	mg/kg-day		1/(mg/kg-day)		4E-02	mg/kg-day	1E+00	mg/kg-day	3E-02									
VINYL CHLORIDE	5E-03	mg/m3	1E-05	mg/kg-day	2E-02	1/(mg/kg-day)	2E-07	3E-05	mg/kg-day	3E-02	mg/kg-day	1E-03									
XYLENES, TOTAL	4E+00	mg/m3	1E-02	mg/kg-day		1/(mg/kg-day)		2E-02	mg/kg-day	3E-02	mg/kg-day	8E-01									
Exp. Route Total										2E-03				3E+01							
Exp. Point Total										2E-03				3E+01							
Exp. Medium Total										2E-03				3E+01							
Medium Total										2E-02				1E+02							
Total of Receptor Risks Across All Media										2E-02	Total of Receptor Hazards Across All Media										1E+02

TABLE 7.13a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Surface Soil	Outdoor Air	Exposure Unit 9	Inhalation	2,3,7,8-TCDD Equivalent	1E-08	mg/m3	3E-10	mg/kg-day		1/(mg/kg-day)		2E-09	mg/kg-day		mg/kg-day	
				ALUMINUM	2E-06	mg/m3	5E-08	mg/kg-day		1/(mg/kg-day)		4E-07	mg/kg-day		mg/kg-day	
				ARSENIC	3E-09	mg/m3	6E-11	mg/kg-day	2E+01	1/(mg/kg-day)	9E-10	5E-10	mg/kg-day	1E-05	mg/kg-day	3E-04
				CADMIUM	8E-09	mg/m3	2E-10	mg/kg-day	6E+00	1/(mg/kg-day)	1E-09	1E-09	mg/kg-day		mg/kg-day	
				CHROMIUM	5E-08	mg/m3	1E-09	mg/kg-day	4E+01	1/(mg/kg-day)	5E-08	9E-09	mg/kg-day	3E-05	mg/kg-day	3E-04
				COPPER	5E-08	mg/m3	1E-09	mg/kg-day		1/(mg/kg-day)		9E-09	mg/kg-day		mg/kg-day	
				IRON	6E-06	mg/m3	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day		mg/kg-day	
				MANGANESE	1E-07	mg/m3	3E-09	mg/kg-day		1/(mg/kg-day)		3E-08	mg/kg-day	1E-05	mg/kg-day	2E-03
				MERCURY	8E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day	9E-05	mg/kg-day	2E-06
				VANADIUM	6E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				HIGHLY CHLORINATED PCBs	4E-10	mg/m3	9E-12	mg/kg-day	2E+00	1/(mg/kg-day)	2E-11	7E-11	mg/kg-day		mg/kg-day	
				ACENAPHTHYLENE	9E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				BENZ(A)ANTHRACENE	4E-09	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	3E-09	mg/m3	6E-11	mg/kg-day		1/(mg/kg-day)		5E-10	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	4E-09	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	1E-09	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				BENZO(K)FLUORANTHENE	1E-09	mg/m3	3E-11	mg/kg-day		1/(mg/kg-day)		2E-10	mg/kg-day		mg/kg-day	
				CHRYSENE	4E-09	mg/m3	9E-11	mg/kg-day		1/(mg/kg-day)		7E-10	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	3E-10	mg/m3	6E-12	mg/kg-day		1/(mg/kg-day)		4E-11	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	7E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				INDENO(1,2,3-CD)PYRENE	8E-10	mg/m3	2E-11	mg/kg-day		1/(mg/kg-day)		1E-10	mg/kg-day		mg/kg-day	
				PHENANTHRENE	6E-09	mg/m3	1E-10	mg/kg-day		1/(mg/kg-day)		1E-09	mg/kg-day		mg/kg-day	
				BENZENE	5E-07	mg/m3	1E-08	mg/kg-day	3E-02	1/(mg/kg-day)	3E-10	9E-08	mg/kg-day	9E-03	mg/kg-day	1E-05
			Exp. Route Total								5E-08					2E-03
		Exp. Point Total									5E-08					2E-03
	Exp. Medium Total										5E-08					2E-03
Medium Total											5E-08					2E-03
Soil	Surface Soil	Exposure Unit 9	Dermal	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	9E-14	mg/kg-day	2E+05	1/(mg/kg-day)	1E-08	7E-13	mg/kg-day	1E-09	mg/kg-day	7E-04
				ALUMINUM	5E+03	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	1E+00	mg/kg-day	
				ARSENIC	6E+00	mg/kg	2E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	1E-07	mg/kg-day	3E-04	mg/kg-day	5E-04
				CADMIUM	2E+01	mg/kg	2E-09	mg/kg-day		1/(mg/kg-day)		1E-08	mg/kg-day	3E-05	mg/kg-day	5E-04
				CHROMIUM	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	8E-05	mg/kg-day	
				COPPER	1E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	4E-02	mg/kg-day	
				IRON	1E+04	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	7E-01	mg/kg-day	
				MANGANESE	3E+02	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	6E-03	mg/kg-day	
				MERCURY	2E+00	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-05	mg/kg-day	
				VANADIUM	1E+01	mg/kg		mg/kg-day		1/(mg/kg-day)			mg/kg-day	2E-04	mg/kg-day	
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	1E-08	mg/kg-day	2E+00	1/(mg/kg-day)	3E-08	1E-07	mg/kg-day	2E-05	mg/kg-day	5E-03
				ACENAPHTHYLENE	2E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	7E-06
				BENZ(A)ANTHRACENE	9E+00	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	9E-08	9E-07	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	9E-08	mg/kg-day	7E+00	1/(mg/kg-day)	6E-07	7E-07	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	1E-07	mg/kg-day	7E-01	1/(mg/kg-day)	9E-08	1E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	3E-08	mg/kg-day		1/(mg/kg-day)		2E-07	mg/kg-day	3E-02	mg/kg-day	8E-06
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	4E-08	mg/kg-day	7E-02	1/(mg/kg-day)	3E-09	3E-07	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	1E-07	mg/kg-day	7E-03	1/(mg/kg-day)	9E-10	1E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	8E-09	mg/kg-day	7E+00	1/(mg/kg-day)	6E-08	6E-08	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	2E-08	mg/kg-day		1/(mg/kg-day)		1E-07	mg/kg-day	1E-03	mg/kg-day	1E-04
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	2E-08	mg/kg-day	7E-01	1/(mg/kg-day)	2E-08	2E-07	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZENE	1E-03	mg/kg		mg/kg-day	6E-02	1/(mg/kg-day)			mg/kg-day	4E-03	mg/kg-day	
			Exp. Route Total								1E-06					7E-03

TABLE 7.13a CT
CALCULATION OF CHEMICAL CANCER RISKS AND NON-CANCER HAZARDS - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Chemical of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Exposure Unit 9	Ingestion	2,3,7,8-TCDD Equivalent	3E-05	mg/kg	3E-12	mg/kg-day	2E+05	1/(mg/kg-day)	4E-07	2E-11	mg/kg-day	1E-09	mg/kg-day	2E-02
				ALUMINUM	5E+03	mg/kg	5E-04	mg/kg-day		1/(mg/kg-day)		4E-03	mg/kg-day	1E+00	mg/kg-day	4E-03
				ARSENIC	6E+00	mg/kg	6E-07	mg/kg-day	2E+00	1/(mg/kg-day)	8E-07	4E-06	mg/kg-day	3E-04	mg/kg-day	1E-02
				CADMIUM	2E+01	mg/kg	2E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	1E-03	mg/kg-day	1E-02
				CHROMIUM	1E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		9E-05	mg/kg-day	3E-03	mg/kg-day	3E-02
				COPPER	1E+02	mg/kg	1E-05	mg/kg-day		1/(mg/kg-day)		8E-05	mg/kg-day	4E-02	mg/kg-day	2E-03
				IRON	1E+04	mg/kg	1E-03	mg/kg-day		1/(mg/kg-day)		9E-03	mg/kg-day	7E-01	mg/kg-day	1E-02
				MANGANESE	3E+02	mg/kg	3E-05	mg/kg-day		1/(mg/kg-day)		2E-04	mg/kg-day	1E-01	mg/kg-day	2E-03
				MERCURY	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-04	mg/kg-day	4E-03
				VANADIUM	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	9E-03	mg/kg-day	1E-03
				HIGHLY CHLORINATED PCBs	9E-01	mg/kg	8E-08	mg/kg-day	2E+00	1/(mg/kg-day)	2E-07	6E-07	mg/kg-day	2E-05	mg/kg-day	3E-02
				ACENAPHTHYLENE	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZO(A)ANTHRACENE	9E+00	mg/kg	8E-07	mg/kg-day	7E-01	1/(mg/kg-day)	6E-07	6E-06	mg/kg-day		mg/kg-day	
				BENZO(A)PYRENE	7E+00	mg/kg	6E-07	mg/kg-day	7E+00	1/(mg/kg-day)	4E-06	5E-06	mg/kg-day		mg/kg-day	
				BENZO(B)FLUORANTHENE	1E+01	mg/kg	8E-07	mg/kg-day	7E-01	1/(mg/kg-day)	6E-07	7E-06	mg/kg-day		mg/kg-day	
				BENZO(G,H,I)PERYLENE	2E+00	mg/kg	2E-07	mg/kg-day		1/(mg/kg-day)		2E-06	mg/kg-day	3E-02	mg/kg-day	5E-05
				BENZO(K)FLUORANTHENE	3E+00	mg/kg	3E-07	mg/kg-day	7E-02	1/(mg/kg-day)	2E-08	2E-06	mg/kg-day		mg/kg-day	
				CHRYSENE	1E+01	mg/kg	8E-07	mg/kg-day	7E-03	1/(mg/kg-day)	6E-09	7E-06	mg/kg-day		mg/kg-day	
				DIBENZ(A,H)ANTHRACENE	6E-01	mg/kg	5E-08	mg/kg-day	7E+00	1/(mg/kg-day)	4E-07	4E-07	mg/kg-day		mg/kg-day	
				DIBENZOFURAN	2E+00	mg/kg	1E-07	mg/kg-day		1/(mg/kg-day)		1E-06	mg/kg-day	1E-03	mg/kg-day	1E-03
				INDENO(1,2,3-CD)PYRENE	2E+00	mg/kg	2E-07	mg/kg-day	7E-01	1/(mg/kg-day)	1E-07	1E-06	mg/kg-day		mg/kg-day	
				PHENANTHRENE	1E+01	mg/kg	1E-06	mg/kg-day		1/(mg/kg-day)		1E-05	mg/kg-day	3E-02	mg/kg-day	3E-04
				BENZENE	1E-03	mg/kg	9E-11	mg/kg-day	6E-02	1/(mg/kg-day)	5E-12	7E-10	mg/kg-day	4E-03	mg/kg-day	2E-07
								Exp. Route Total						7E-06		
						Exp. Point Total						8E-06				1E-01
					Exp. Medium Total						8E-06				1E-01	
Medium Total										8E-06				1E-01		
Total of Receptor Risks Across All Media											8E-06	Total of Receptor Hazards Across All Media				1E-01

RAGS Table 9 RME Series

TABLE 9.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	8E-05	--	--	8E-05	Developmental effects	6E+00	--	--	6E+00
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	7E-01	--	--	7E-01
			ARSENIC	3E-06	--	--	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	8E-02	--	--	8E-02
			CHROMIUM	--	--	--	--	--	6E-02	--	--	6E-02
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	8E-02	--	--	8E-02
			MANGANESE	--	--	--	--	CNS (N)	7E-03	--	--	7E-03
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	3E+00	--	--	3E+00
			SELENIUM	--	--	--	--	Clinical selenosis	9E-02	--	--	9E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-02	--	--	2E-02
			ZINC	--	--	--	--	Decreased ESOD (B)	4E-02	--	--	4E-02
			HIGHLY CHLORINATED PCBs	3E-05	--	--	3E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	9E+00	--	--	9E+00
			LESS CHLORINATED PCBs	2E-05	--	--	2E-05	Reduced birth weights (W)	2E+00	--	--	2E+00
			4,4-DDD	8E-08	--	--	8E-08	--	--	--	--	--
			4,4'-DDT	8E-08	--	--	8E-08	Liver lesions (H)	6E-03	--	--	6E-03
			ALDRIN	1E-06	--	--	1E-06	Liver toxicity (H)	2E-02	--	--	2E-02
			DELTA-BHC	--	--	--	--	--	--	--	--	--
			DIELDRIN	2E-06	--	--	2E-06	Hepatic (H)	2E-02	--	--	2E-02
			HEPTACHLOR EPOXIDE	1E-06	--	--	1E-06	Increased liver-to-body weight ratio in males and females (H)	9E-02	--	--	9E-02
			BIS(2-ETHYLHEXYL)PHTHALATE	8E-07	--	--	8E-07	Increased relative liver weight (H)	3E-02	--	--	3E-02
		HEXACHLOROBENZENE	5E-07	--	--	5E-07	Hepatic (H)	5E-03	--	--	5E-03	
		Chemical Total	1E-04	--	--	1E-04	--	2E+01	--	--	2E+01	
Exposure Point Total			1E-04				2E+01					
Exposure Medium Total			1E-04				2E+01					
Medium Total			1E-04				2E+01					
Sediment	Surface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-07	--	8E-07	1E-06	Developmental effects	1E-02	--	7E-02	8E-02
			ARSENIC	2E-07	--	8E-07	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-03	--	2E-02	3E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-02	--	2E-03	1E-02
			CHROMIUM	--	--	--	--	None Reported (O)	1E-01	--	--	1E-01
			IRON	--	--	--	--	Gastrointestinal effects	3E-03	--	--	3E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	1E-02	--	--	1E-02
			MERCURY	--	--	--	--	Autoimmune effects	5E-02	--	--	5E-02
			THALLIUM	--	--	--	--	Hematological effects	2E-03	--	--	2E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-02	--	--	1E-02

TABLE 9.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	HIGHLY CHLORINATED PCBs	3E-08	--	6E-07	6E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	8E-03	--	2E-01	2E-01
			DIELDRIN	5E-09	--	--	5E-09	Hepatic (H)	7E-05	--	--	7E-05
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	3E-05	--	--	3E-05
			HEPTACHLOR EPOXIDE	1E-09	--	--	1E-09	Increased liver-to-body weight ratio in males and females (H)	1E-04	--	--	1E-04
			1-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E-03	--	3E-02	3E-02
			ACENAPHTHYLENE*	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-05	--	5E-04	6E-04
			BENZ(A)ANTHRACENE	9E-06	--	2E-04	2E-04	--	--	--	--	--
			BENZO(A)PYRENE	2E-05	--	4E-04	4E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-06	--	6E-05	6E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE*	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-04	--	6E-03	6E-03
			BENZO(K)FLUORANTHENE	--	--	2E-06	2E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	9E-09	--	--	9E-09	Increased relative liver weight (H)	4E-04	--	6E-03	7E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	3E-08	--	5E-07	6E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-06	--	7E-05	7E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E-03	--	7E-02	8E-02
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	7E-04	--	1E-02	2E-02
			HEXACHLOROBENZENE	4E-09	--	6E-08	6E-08	Hepatic (H)	3E-05	--	5E-04	6E-04
			INDENO(1,2,3-CD)PYRENE	1E-06	--	2E-05	2E-05	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	6E-04	--	1E-02	1E-02
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	8E-04	--	2E-02	2E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-03	--	2E-02	3E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	3E-11	--	--	3E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1E-05	--	--	1E-05
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	--	--
			1,3,5-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	2E-09	--	--	2E-09	Liver	7E-05	--	--	7E-05
			BENZENE	4E-09	--	--	4E-09	Reduced lymphocyte count	2E-04	--	--	2E-04
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-04	--	--	2E-04
			METHYLENE CHLORIDE	5E-11	--	--	5E-11	Liver toxicity (H)	1E-06	--	--	1E-06
			N-HEXADACANE	--	--	--	--	--	--	--	--	--

TABLE 9.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	3E-05	--	--	3E-05
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	4E-05	--	--	4E-05
		Chemical Total	4E-05	--	7E-04	7E-04		3E-01	--	5E-01	7E-01	
			Exposure Point Total				7E-04					7E-01
	Exposure Medium Total					7E-04					7E-01	
Medium Total							7E-04					7E-01
Soil	Surface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	1E-06	--	7E-06	8E-06	Developmental effects	1E-01	--	5E-01	6E-01
			ALUMINUM	--	--	--	--	Neurotoxicity	1E-03	--	--	1E-03
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	3E-04	--	--	3E-04
			ARSENIC	2E-07	--	1E-06	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	6E-03	--	3E-02	4E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	3E-04	--	--	3E-04
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-03	--	3E-02	4E-02
			CHROMIUM	--	--	--	--	--	8E-03	--	--	8E-03
			COPPER	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03
			IRON	--	--	--	--	Gastrointestinal effects	4E-03	--	--	4E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	5E-04	--	--	5E-04
			MERCURY	--	--	--	--	Autoimmune effects	6E-03	--	--	6E-03
			SILVER	--	--	--	--	Argyria (In)	4E-04	--	--	4E-04
			THALLIUM	--	--	--	--	Hematological effects	2E-03	--	--	2E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	5E-04	--	--	5E-04
								Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-02	--	3E-01	3E-01
			HIGHLY CHLORINATED PCBs	5E-08	--	1E-06	1E-06					
			LESS CHLORINATED PCBs	3E-08	--	7E-07	7E-07	Reduced birth weights (W)	3E-03	--	6E-02	6E-02
			DIELDRIN	3E-09	--	--	3E-09	Hepatic (H)	5E-05	--	--	5E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	4E-04	--	9E-03	1E-02
			ACENAPHTHYLENE	--	--	--	--	--	4E-05	--	8E-04	8E-04
			BENZ(A)ANTHRACENE	5E-07	--	9E-06	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	5E-06	--	9E-05	1E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	4E-07	--	8E-06	8E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	7E-05	--	1E-03	1E-03
			BENZO(K)FLUORANTHENE	4E-08	--	7E-07	8E-07	--	--	--	--	--
			CHRYSENE	5E-09	--	9E-08	1E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-05	2E-05	--	--	--	--	--

TABLE 9.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 1	DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	7E-04	--	1E-02	1E-02
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	2E-04	--	4E-03	4E-03
			HEXACHLOROBENZENE	2E-08	--	3E-07	3E-07	Hepatic (H)	2E-04	--	3E-03	3E-03
			INDENO(1,2,3-CD)PYRENE	3E-07	--	6E-06	6E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-04	--	3E-03	4E-03
			PHENANTHRENE	--	--	--	--	--	2E-04	--	3E-03	3E-03
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	2E-10	--	--	2E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	6E-05	--	--	6E-05
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	1E-05	--	--	1E-05
			1,4-DICHLOROBENZENE	2E-09	--	--	2E-09	Liver	6E-05	--	--	6E-05
			BENZENE	3E-10	--	--	3E-10	Reduced lymphocyte count	2E-05	--	--	2E-05
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	9E-06	--	1E-04	2E-04		2E-01	--	1E+00	1E+00
		Exposure Point Total					2E-04					1E+00
	Exposure Medium Total						2E-04					1E+00
Medium Total							2E-04					1E+00
Surface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	4E-05	--	4E-05
			ANTIMONY	--	--	--	--	--	--	--	--	--
			ARSENIC	--	9E-11	--	9E-11	Development, cardiovascular, nervous system	--	5E-06	--	5E-06
			BARIUM	--	--	--	--	Renal toxicity	--	1E-05	--	1E-05
			CADMIUM	--	9E-11	--	9E-11	--	--	--	--	--
			CHROMIUM	--	3E-09	--	3E-09	--	--	3E-05	--	3E-05
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E-04	--	2E-04
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	7E-07	--	7E-07
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-12	--	2E-12	--	--	--	--	--
			LESS CHLORINATED PCBs	--	1E-12	--	1E-12	--	--	--	--	--
			DIELDRIN	--	1E-13	--	1E-13	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	4E-12	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	4E-11	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	4E-12	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 1	BENZO(K)FLUORANTHENE	--	3E-13	--	--	--	--	--	--	--
			CHRYSENE	--	4E-14	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	9E-12	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORANTHENE	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	6E-13	--	6E-13	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	1E-12	--	1E-12	Nasal/respiratory (P)	--	1E-07	--	1E-07
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	1E-04	--	1E-04	--
			1,4-DICHLOROBENZENE	--	8E-08	--	8E-08	Liver	--	1E-04	--	1E-04
			BENZENE	--	5E-09	--	5E-09	Decreased lymphocyte count	--	2E-04	--	2E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
	DODECANE	--	--	--	--	--	--	--	--	--		
		Chemical Total	--	9E-08	--	9E-08		--	7E-04	--	7E-04	
		Exposure Point Total					9E-08					7E-04
		Exposure Medium Total					9E-08					7E-04
Medium Total							9E-08					7E-04
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	1E-03	1E-03
			ARSENIC	--	--	2E-08	2E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	5E-04	5E-04
			CHROMIUM	--	--	--	--	--	--	--	7E-03	7E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	4E-04	4E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	3E-03	3E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	2E-04	2E-04
			THALLIUM	--	--	--	--	Hematological effects	--	--	2E-03	2E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	3E-04	3E-04
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	3E-05	3E-05
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	2E-03	2E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	5E-04	5E-04
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	2E-05	2E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	2E-04	2E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	3E-05	3E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
					BIS(2-ETHYLHEXYL)PHTHALATE	--	--	4E-08	4E-08	Increased relative liver weight (H)	--	--

TABLE 9.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	2E-07	2E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	1E-05	1E-05	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	1E-01	1E-01
			PHENANTHRENE	--	--	--	--	--	--	--	5E-03	5E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	9E-09	9E-09	Liver	--	--	3E-04	3E-04
			BENZENE	--	--	1E-07	1E-07	Reduced lymphocyte count	--	--	8E-03	8E-03
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	4E-03	4E-03
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total	--	--	3E-04	3E-04		--	--	2E-01	2E-01
		Exposure Point Total					3E-04					2E-01
	Exposure Medium Total						3E-04					2E-01
Medium Total							3E-04					2E-01
Receptor Total							1E-03					2E+01

Total Risk Across All Media = 1E-03

Total Hazard Across All Media = 2E+01

Total Liver HI Across All Media = 2E-01
Total Kidney HI Across All Media = 1E-01
Total Nervous System Effects HI Across All Media = 3E+00
Total Lymphocyte Effects HI Across All Media = 8E-03
Total Nasal/Respiratory Effects HI Across All Media = 4E-02
Total Ocular Effects HI Across All Media = 9E+00
Total Other Effects HI Across All Media = 1E+01

TABLE 9.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	5E-04	--	--	5E-04	Developmental effects	7E+00	--	--	7E+00		
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	9E-01	--	--	9E-01		
			ARSENIC	2E-05	--	--	2E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	--	1E-01		
			CHROMIUM	--	--	--	--	--	7E-02	--	--	7E-02		
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	1E-01	--	--	1E-01		
			MANGANESE	--	--	--	--	CNS (N)	8E-03	--	--	8E-03		
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	4E+00	--	--	4E+00		
			SELENIUM	--	--	--	--	Clinical selenosis	1E-01	--	--	1E-01		
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-02	--	--	3E-02		
			ZINC	--	--	--	--	Decreased ESOD (B)	5E-02	--	--	5E-02		
			HIGHLY CHLORINATED PCBs	2E-04	--	--	2E-04	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E+01	--	--	1E+01		
			LESS CHLORINATED PCBs	1E-04	--	--	1E-04	Reduced birth weights (W)	2E+00	--	--	2E+00		
			4,4-DDD	5E-07	--	--	5E-07	--	--	--	--	--		
			4,4'-DDT	5E-07	--	--	5E-07	Liver lesions (H)	7E-03	--	--	7E-03		
			ALDRIN	7E-06	--	--	7E-06	Liver toxicity (H)	3E-02	--	--	3E-02		
			DELTA-BHC	--	--	--	--	--	--	--	--	--		
			DIELDRIN	9E-06	--	--	9E-06	--	3E-02	--	--	3E-02		
			HEPTACHLOR EPOXIDE	6E-06	--	--	6E-06	Increased liver-to-body weight ratio in males and females (H)	1E-01	--	--	1E-01		
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-06	--	--	5E-06	Increased relative liver weight (H)	4E-02	--	--	4E-02		
			HEXACHLOROBENZENE	3E-06	--	--	3E-06	Hepatic (H)	6E-03	--	--	6E-03		
			Chemical Total	8E-04	--	--	8E-04		3E+01	--	--	3E+01		
			Exposure Point Total				8E-04				3E+01			
			Exposure Medium Total				8E-04				3E+01			
			Medium Total				8E-04				3E+01			
Sediment	Surface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	3E-07	--	4E-07	7E-07	Developmental effects	5E-03	--	6E-03	1E-02		
			ARSENIC	3E-07	--	4E-07	7E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-03	--	2E-03	4E-03		
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-03	--	2E-04	5E-03		
			CHROMIUM	--	--	--	--	None Reported (O)	5E-02	--	--	5E-02		
			IRON	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--	4E-03		
			MERCURY	--	--	--	--	Autoimmune effects	2E-02	--	--	2E-02		
			THALLIUM	--	--	--	--	Hematological effects	6E-04	--	--	6E-04		

TABLE 9.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	VANADIUM	--	--	--	--	Decreased hair cystine	5E-03	--	--	5E-03
			HIGHLY CHLORINATED PCBs	5E-08	--	3E-07	3E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-03	--	1E-02	2E-02
			DIELDRIN	9E-09	--	--	9E-09	Hepatic (H)	3E-05	--	--	3E-05
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	1E-05	--	--	1E-05
			HEPTACHLOR EPOXIDE	2E-09	--	--	2E-09	Increased liver-to-body weight ratio in males and females (H)	5E-05	--	--	5E-05
			1-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	6E-04	--	3E-03	3E-03
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-05	--	5E-05	6E-05
			BENZ(A)ANTHRACENE	7E-06	--	3E-05	4E-05	--	--	--	--	--
			BENZO(A)PYRENE	2E-05	--	7E-05	9E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-06	--	1E-05	1E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-04	--	5E-04	6E-04
			BENZO(K)FLUORANTHENE	9E-08	--	4E-07	5E-07	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	2E-08	--	6E-08	8E-08	Increased relative liver weight (H)	2E-04	--	5E-04	7E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	2E-08	--	1E-07	1E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-06	--	1E-05	2E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	6E-03	8E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-04	--	1E-03	2E-03
			HEXACHLOROBENZENE	7E-09	--	2E-08	3E-08	Hepatic (H)	1E-05	--	4E-05	6E-05
			INDENO(1,2,3-CD)PYRENE	8E-07	--	4E-06	5E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-04	--	1E-03	1E-03
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-04	--	1E-03	2E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	5E-04	--	2E-03	3E-03
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	7E-11	--	--	7E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	4E-06	--	--	4E-06
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	--	--
			1,3,5-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	4E-09	--	--	4E-09	Liver	3E-05	--	--	3E-05
			BENZENE	8E-09	--	--	8E-09	Reduced lymphocyte count	8E-05	--	--	8E-05
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	9E-05	--	--	9E-05
			METHYLENE CHLORIDE	1E-10	--	--	1E-10	Liver toxicity (H)	6E-07	--	--	6E-07
			N-HEXADACANE	--	--	--	--	--	--	--	--	--
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--

TABLE 9.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	TOLUENE	--	--	--	--	Increased kidney weight (R)	1E-05	--	--	1E-05
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-05	--	--	1E-05
			Chemical Total	3E-05	--	1E-04	2E-04		1E-01	--	4E-02	1E-01
		Exposure Point Total				2E-04				1E-01		
	Exposure Medium Total						2E-04				1E-01	
Medium Total							2E-04				1E-01	
Soil	Surface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	3E-06	--	3E-06	6E-06	Developmental effects	4E-02	--	4E-02	9E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	6E-04	--	--	6E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	1E-04	--	--	1E-04
			ARSENIC	5E-07	--	5E-07	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-03	--	3E-03	5E-03
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-04	--	--	1E-04
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-03	--	3E-03	5E-03
			CHROMIUM	--	--	--	--	--	3E-03	--	--	3E-03
			COPPER	--	--	--	--	Gastrointestinal effects	4E-04	--	--	4E-04
			IRON	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	2E-04	--	--	2E-04
			MERCURY	--	--	--	--	Autoimmune effects	2E-03	--	--	2E-03
			SILVER	--	--	--	--	Argyria (In)	2E-04	--	--	2E-04
			THALLIUM	--	--	--	--	Hematological effects	7E-04	--	--	7E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-04	--	--	2E-04
			HIGHLY CHLORINATED PCBs	1E-07	--	5E-07	6E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-03	--	3E-02	3E-02
			LESS CHLORINATED PCBs	6E-08	--	3E-07	4E-07	Reduced birth weights (W)	1E-03	--	5E-03	6E-03
			DIELDRIN	6E-09	--	--	6E-09	Hepatic (H)	2E-05	--	--	2E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E-04	--	8E-04	1E-03
			ACENAPHTHYLENE	--	--	--	--	--	1E-05	--	7E-05	8E-05
			BENZ(A)ANTHRACENE	4E-07	--	2E-06	2E-06	--	--	--	--	--
			BENZO(A)PYRENE	4E-06	--	2E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-07	--	1E-06	2E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	3E-05	--	1E-04	1E-04
			BENZO(K)FLUORANTHENE	3E-08	--	1E-07	2E-07	--	--	--	--	--
			CHRYSENE	4E-09	--	2E-08	2E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	8E-07	--	4E-06	5E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-04	--	9E-04	1E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	7E-05	--	3E-04	4E-04
			HEXACHLOROBENZENE	4E-08	--	1E-07	2E-07	Hepatic (H)	7E-05	--	2E-04	3E-04

TABLE 9.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Soil	Surface Soil	Exposure Unit 1	INDENO(1,2,3-CD)PYRENE	2E-07	--	1E-06	1E-06	--	--	--	--		
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	6E-05	--	3E-04	4E-04	
			PHENANTHRENE	--	--	--	--	--	6E-05	--	3E-04	3E-04	
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--	
			1,2,4-TRICHLOROBENZENE	3E-10	--	--	3E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	2E-05	--	--	2E-05	
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	5E-06	--	--	5E-06	
			1,4-DICHLOROBENZENE	4E-09	--	--	4E-09	Liver	2E-05	--	--	2E-05	
			BENZENE	7E-10	--	--	7E-10	Reduced lymphocyte count	7E-06	--	--	7E-06	
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--	
			DODECANE	--	--	--	--	--	--	--	--	--	
			Chemical Total	9E-06	--	3E-05	4E-05		6E-02	--	9E-02	1E-01	
			Exposure Point Total							4E-05			
	Exposure Medium Total							4E-05					1E-01
Medium Total							4E-05					1E-01	
Surface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--	
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	4E-05	--	4E-05	
			ANTIMONY	--	--	--	--	--	--	--	--	--	
			ARSENIC	--	5E-10	--	5E-10	Development, cardiovascular, nervous system	--	5E-06	--	5E-06	
			BARIUM	--	--	--	--	Renal toxicity	--	1E-05	--	1E-05	
			CADMIUM	--	5E-10	--	5E-10	--	--	--	--	--	
			CHROMIUM	--	2E-08	--	2E-08	--	--	3E-05	--	3E-05	
			COPPER	--	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--	--	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E-04	--	2E-04	
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	7E-07	--	7E-07	
			SILVER	--	--	--	--	--	--	--	--	--	
			THALLIUM	--	--	--	--	--	--	--	--	--	
			VANADIUM	--	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	9E-12	--	9E-12	--	--	--	--	--	
			LESS CHLORINATED PCBs	--	6E-12	--	6E-12	--	--	--	--	--	
			DIELDRIN	--	6E-13	--	6E-13	--	--	--	--	--	
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--	
			FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			HEXACHLOROBENZENE	--	3E-12	--	3E-12	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--	

TABLE 9.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 1	NAPHTHALENE	--	6E-12	--	6E-12	Nasal/respiratory (P)	--	1E-07	--	1E-07
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	2E-04	--	2E-04	--
			1,4-DICHLOROBENZENE	--	4E-07	--	4E-07	Liver	--	1E-04	--	1E-04
			BENZENE	--	3E-08	--	3E-08	Decreased lymphocyte count	--	3E-04	--	3E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	--	5E-07	--	5E-07		--	8E-04	--	8E-04
Exposure Point Total			5E-07				8E-04					
Exposure Medium Total			5E-07				8E-04					
Medium Total			5E-07				8E-04					
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	--	--	--	1E-03	1E-03
			ARSENIC	--	--	8E-08	8E-08	Development, cardiovascular, nervous system	--	--	4E-04	4E-04
			CHROMIUM	--	--	--	--	--	--	6E-03	6E-03	--
			IRON	--	--	--	--	--	--	3E-04	3E-04	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	--	3E-03	3E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	2E-04	2E-04
			THALLIUM	--	--	--	--	--	--	2E-03	2E-03	--
			VANADIUM	--	--	--	--	--	--	3E-04	3E-04	--
			ZINC	--	--	--	--	--	--	3E-05	3E-05	--
			2,4-DIMETHYLPHENOL	--	--	--	--	--	--	1E-03	1E-03	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	--	--	4E-04	4E-04	--
			ACENAPHTHENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	4E-05	4E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	4E-04	4E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	5E-05	5E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	2E-07	2E-07	--	--	1E-03	1E-03	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	3E-07	3E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORENE	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	3E-05	3E-05	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Nasal/respiratory (P)	--	--	1E-01	1E-01
			PHENANTHRENE	--	--	--	--	--	--	5E-03	5E-03	--
PYRENE	--	--	--	--	--	--	--	--	--			
1,2,4-TRIMETHYLBENZENE	--	--	--	--	Hematological and Pulmonary	--	--	--	--			

TABLE 9.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	4E-08	4E-08	Liver	--	--	2E-04	2E-04
			BENZENE	--	--	6E-07	6E-07	Decreased lymphocyte count	--	--	7E-03	7E-03
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Neurological effects	--	--	4E-03	4E-03
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	--	--	--
			Chemical Total	--	--	5E-04	5E-04		--	--	1E-01	1E-01
		Exposure Point Total					5E-04					1E-01
	Exposure Medium Total						5E-04					1E-01
Medium Total							5E-04					1E-01
Receptor Total							2E-03				Receptor HI Total	3E+01

Total Risk Across All Media = 2E-03

Total Hazard Across All Media = 3E+01

Total Liver HI Across All Media =	2E-01
Total Kidney HI Across All Media =	1E-02
Total Nervous System Effects HI Across All Media =	4E+00
Total Lymphocyte Effects HI Across All Media =	7E-03
Total Nasal/Respiratory Effects HI Across All Media =	1E-01
Total Ocular Effects HI Across All Media =	1E+01
Total Other Effects HI Across All Media =	1E+01

TABLE 9.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	8E-07	--	2E-07	1E-06	Developmental effects	2E-01	--	4E-03	2E-01
			ARSENIC	4E-07	--	1E-07	5E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	6E-04	3E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	2E-03	--	--	2E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	4E-03	--	1E-04	4E-03
			CHROMIUM	--	--	--	--	None Reported (O)	5E-02	--	--	5E-02
			IRON	--	--	--	--	Gastrointestinal effects	5E-02	--	--	5E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	8E-03	--	--	8E-03
			MERCURY	--	--	--	--	Autoimmune effects	6E-02	--	--	6E-02
			THALLIUM	--	--	--	--	Hematological effects	2E-02	--	--	2E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	4E-03	--	--	4E-03
			HIGHLY CHLORINATED PCBs	1E-07	--	2E-07	3E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	1E-02	1E-01
			DELTA-BHC	--	--	--	--	--	--	--	--	--
			DIELDRIN	2E-08	--	--	2E-08	Hepatic (H)	9E-04	--	--	9E-04
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	--	--	--	--
			HEPTACHLOR EPOXIDE	--	--	--	--	Increased liver-to-body weight ratio in males and females (H)	--	--	--	--
			1-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E+00	--	2E-01	2E+00
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	2E-02	--	2E-03	3E-02
			ACENAPHTHYLENE*	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	6E-02	--	6E-03	7E-02
			ANTHRACENE	--	--	--	--	No observed effects (O)	4E-03	--	4E-04	4E-03
			BENZ(A)ANTHRACENE	1E-05	--	1E-05	3E-05	--	--	--	--	--
			BENZO(A)PYRENE	3E-05	--	3E-05	6E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	4E-06	--	5E-06	9E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-03	--	3E-04	3E-03
			BENZO(K)FLUORANTHENE	2E-07	--	2E-07	4E-07	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	--	--	Increased relative liver weight (H)	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	9E-08	--	1E-07	2E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	5E-06	--	6E-06	1E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E+00	--	2E-01	3E+00
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	5E-02	--	5E-03	5E-02
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	7E-02	--	5E-03	7E-02
			HEXACHLOROBENZENE	1E-08	--	1E-08	2E-08	Hepatic (H)	3E-04	--	2E-05	4E-04
			INDENO(1,2,3-CD)PYRENE	1E-06	--	2E-06	3E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+00	--	9E-02	1E+00
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-01	--	2E-02	2E-01

TABLE 9.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	5E-02	--	4E-03	5E-02		
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLOROBENZENE	1E-10	--	--	1E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1E-04	--	--	1E-04		
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	--	--		
			1,3,5-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,4-DICHLOROBENZENE	8E-09	--	--	8E-09	Liver	7E-04	--	--	7E-04		
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	4E-02	--	--	4E-02		
			BENZENE	3E-07	--	--	3E-07	Reduced lymphocyte count	5E-02	--	--	5E-02		
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-03	--	--	2E-03		
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	3E-03	--	--	3E-03		
			METHYLENE CHLORIDE	6E-10	--	--	6E-10	Liver toxicity (H)	5E-05	--	--	5E-05		
			N-HEXADACANE	--	--	--	--	--	--	--	--	--		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	2E-03	--	--	2E-03		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	1E-02	--	--	1E-02		
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-02	--	--	1E-02		
	Chemical Total			5E-05	--	6E-05	1E-04	6E+00					7E+00	
Exposure Point Total							1E-04						7E+00	
Exposure Medium Total							1E-04						7E+00	
Medium Total							1E-04						7E+00	
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	7E-06	--	6E-07	8E-06	Developmental effects	1E-01	--	1E-02	1E-01		
			ALUMINUM	--	--	--	--	Neurotoxicity	2E-03	--	--	2E-03		
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	4E-04	--	--	4E-04		
			ARSENIC	2E-06	--	1E-07	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	9E-04	1E-02		
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	7E-04	--	--	7E-04		
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-03	--	6E-04	6E-03		
			CHROMIUM	--	--	--	--	--	9E-03	--	--	9E-03		
			COPPER	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03		
			IRON	--	--	--	--	Gastrointestinal effects	5E-03	--	--	5E-03		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	CNS (N)	6E-04	--	--	6E-04		
			MERCURY	--	--	--	--	Autoimmune effects	1E-02	--	--	1E-02		
			SILVER	--	--	--	--	Argyria (In)	8E-04	--	--	8E-04		
			THALLIUM	--	--	--	--	Hematological effects	4E-03	--	--	4E-03		
			VANADIUM	--	--	--	--	Decreased hair cystine	6E-04	--	--	6E-04		
			HIGHLY CHLORINATED PCBs			5E-07	--	2E-07	7E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	4E-02	--	2E-02	5E-02
			LESS CHLORINATED PCBs			6E-07	--	2E-07	8E-07	Reduced birth weights (W)	1E-02	--	5E-03	2E-02
			DIELDRIN			1E-08	--	--	1E-08	Hepatic (H)	5E-05	--	--	5E-05
			2,4-DIMETHYLPHENOL			--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	4E-05	--	1E-05	6E-05
			2-METHYLNAPHTHALENE			--	--	--	--	Pulmonary alveolar proteinosis	2E-02	--	6E-03	2E-02
			3&4-METHYLPHENOL			--	--	--	--	Decreased body weight and neurotoxicity	5E-05	--	1E-05	6E-05

TABLE 9.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	3E-04	--	1E-04	5E-04		
			ACENAPHTHYLENE	--	--	--	--	--	3E-04	--	1E-04	5E-04		
			ANTHRACENE	--	--	--	--	No observed effects (O)	1E-04	--	5E-05	2E-04		
			BENZ(A)ANTHRACENE	6E-06	--	2E-06	9E-06	--	--	--	--	--		
			BENZO(A)PYRENE	4E-05	--	2E-05	6E-05	--	--	--	--	--		
			BENZO(B)FLUORANTHENE	6E-06	--	2E-06	8E-06	--	--	--	--	--		
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-04	--	7E-05	2E-04		
			BENZO(K)FLUORANTHENE	2E-07	--	9E-08	3E-07	--	--	--	--	--		
			CARBAZOLE	--	--	--	--	--	--	--	--	--		
			CHRYSENE	5E-08	--	2E-08	7E-08	--	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	5E-06	--	2E-06	7E-06	--	--	--	--	--		
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-02	--	8E-03	3E-02		
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	2E-03	--	7E-04	2E-03		
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	9E-04	--	3E-04	1E-03		
			HEXACHLOROBENZENE	1E-07	--	3E-08	1E-07	Hepatic (H)	2E-04	--	7E-05	3E-04		
			INDENO(1,2,3-CD)PYRENE	1E-06	--	6E-07	2E-06	--	--	--	--	--		
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E-02	--	5E-03	2E-02		
			PHENANTHRENE	--	--	--	--	--	4E-03	--	1E-03	5E-03		
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-03	--	7E-04	3E-03		
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLOROBENZENE	5E-09	--	--	5E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	4E-04	--	--	4E-04		
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	3E-04	--	--	3E-04		
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,4-DICHLOROBENZENE	6E-08	--	--	6E-08	Liver	4E-04	--	--	4E-04		
			BENZENE	5E-08	--	--	5E-08	Reduced lymphocyte count	6E-04	--	--	6E-04		
			BROMOMETHANE	--	--	--	--	Epithelial hyperplasia of the forestomach	1E-04	--	--	1E-04		
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	1E-04	--	--	1E-04		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	7E-05	--	--	7E-05		
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	6E-05	--	--	6E-05		
			DODECANE	--	--	--	--	--	--	--	--	--		
						Chemical Total	7E-05	--	3E-05	1E-04		3E-01	--	6E-02
					Exposure Point Total				1E-04					4E-01
				Exposure Medium Total				1E-04					4E-01	
Medium Total							1E-04					4E-01		
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--		
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	5E-02	--	5E-02		
			ANTIMONY	--	--	--	--	--	--	--	--	--		
			ARSENIC	--	6E-07	--	6E-07	Development, cardiovascular, nervous system	--	8E-03	--	8E-03		
			CADMIUM	--	5E-07	--	5E-07	--	--	--	--	--		

TABLE 9.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	CHROMIUM	--	2E-05	--	2E-05	--	--	4E-02	--	4E-02
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O) PNS (N); CNS (N)	--	2E-01	--	2E-01
			MERCURY	--	--	--	--		--	1E-03	--	1E-03
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-08	--	2E-08	--	--	--	--	--
			LESS CHLORINATED PCBs	--	2E-08	--	2E-08	--	--	--	--	--
			DIELDRIN	--	6E-10	--	6E-10	--	--	--	--	--
			2,4-DIMETHYLPHENOL	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORANTHENE	--	--	--	--	--	--	--	--	--
			FLUORENE	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	4E-09	--	4E-09	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	5E-07	--	5E-07	Nasal/respiratory (P)	--	1E-02	--	1E-02
			PHENANTHRENE	--	--	--	--		--	--	--	--
			PYRENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	2E-06	--	2E-06	Liver Decreased lymphocyte count	--	6E-04	--	6E-04
			BENZENE	--	5E-07	--	5E-07		--	6E-03	--	6E-03
			BROMOMETHANE	--	--	--	--	Nasal lesions and membrane degeneration	--	4E-03	--	4E-03
			TOLUENE	--	--	--	--	Neurological effects	--	6E-05	--	6E-05
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	7E-01	--	7E-01
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	--	2E-05	--	2E-05	--	--	1E+00	--	1E+00
		Exposure Point Total					2E-05					1E+00
	Exposure Medium Total						2E-05					1E+00
Medium Total							2E-05					1E+00

TABLE 9.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	ALUMINUM	--	--	--	--	Neurotoxicity	--	--	7E-05	7E-05
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	8E-04	8E-04
			ARSENIC	--	--	7E-08	7E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	4E-04	4E-04
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	5E-03	5E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	2E-03	2E-03
			CHROMIUM	--	--	--	--	--	--	--	1E-02	1E-02
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	--	--	3E-05	3E-05
			IRON	--	--	--	--	Gastrointestinal effects	--	--	3E-04	3E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	5E-03	5E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	1E-03	1E-03
			SILVER	--	--	--	--	Argyria (In)	--	--	2E-04	2E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	9E-04	9E-04
			HIGHLY CHLORINATED PCBs	--	--	--	--	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	--	--	--	--
			4,4'-DDT	--	--	3E-08	3E-08	Liver lesions (H)	--	--	5E-04	5E-04
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	--	--	--	--
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	--	--	2E-06	2E-06
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	1E-05	1E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	--	--	6E-06	6E-06
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	2E-05	2E-05
			4-METHYLPHENOL	--	--	--	--	--	--	--	9E-06	9E-06
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ANTHRACENE	--	--	--	--	No observed effects (O)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	1E-07	1E-07	--	--	--	--	--
			BENZO(A)PYRENE	--	--	2E-06	2E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	1E-07	1E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	2E-10	2E-10	Increased relative liver weight (H)	--	--	2E-06	2E-06
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	1E-09	1E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	9E-07	9E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	--	--	5E-05	5E-05
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			HEXACHLOROBUTADIENE	--	--	7E-11	7E-11	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	2E-07	2E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	3E-04	3E-04

TABLE 9.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	PHENANTHRENE	--	--	--	--	--	--	--	1E-04	1E-04
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	--	--	1E-06	1E-06
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	1E-10	1E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	--	--	9E-06	9E-06
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	1E-05	1E-05
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	1E-09	1E-09	Liver	--	--	1E-05	1E-05
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	--	--	--	--
			BENZENE	--	--	7E-09	7E-09	Reduced lymphocyte count	--	--	9E-05	9E-05
			BROMODICHLOROMETHANE	--	--	2E-12	2E-12	Renal cytomegaly (R)	--	--	3E-09	3E-09
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	--	--	6E-06	6E-06
			CHLOROFORM	--	--	--	--	Moderate/marked fatty cyst formation in the liver and elevated SGPT	--	--	3E-08	3E-08
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	--	--	1E-06	1E-06
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	--	--	--	--
			METHYLENE CHLORIDE	--	--	5E-12	5E-12	Liver toxicity (H)	--	--	3E-08	3E-08
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	--	--	1E-06	1E-06
			TETRACHLOROETHENE	--	--	7E-11	7E-11	Hepatotoxicity in mice (H), weight gain in rats	--	--	4E-08	4E-08
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	1E-05	1E-05
			VINYL CHLORIDE	--	--	5E-11	5E-11	Liver cell polymorphism (H)	--	--	6E-08	6E-08
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total	--	--	4E-06	4E-06		--	--	3E-02	3E-02
		Exposure Point Total					4E-06					3E-02
	Exposure Medium Total						4E-06					3E-02
Medium Total							4E-06					3E-02
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	6E-04	6E-04
			ARSENIC	--	--	4E-08	4E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	2E-04	2E-04
			CHROMIUM	--	--	--	--	--	--	--	3E-03	3E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	2E-04	2E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	2E-03	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	9E-05	9E-05
			THALLIUM	--	--	--	--	Hematological effects	--	--	1E-03	1E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	1E-04	1E-04
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	1E-05	1E-05
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	7E-04	7E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	2E-04	2E-04
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--

TABLE 9.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	1E-05	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-04	1E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-05	2E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	5E-08	5E-08	Increased relative liver weight (H)	--	--	5E-04	5E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	1E-07	1E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	9E-06	9E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	5E-02	5E-02
			PHENANTHRENE	--	--	--	--	--	--	--	2E-03	2E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	1E-08	1E-08	Liver	--	--	1E-04	1E-04
			BENZENE	--	--	3E-07	3E-07	Reduced lymphocyte count	--	--	3E-03	3E-03
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	2E-03	2E-03
		XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--	
Chemical Total			--	--	2E-04	2E-04	7E-02			7E-02		
Exposure Point Total			2E-04				7E-02					
Exposure Medium Total			2E-04				7E-02					
Medium Total			2E-04				7E-02					
Receptor Total			4E-04				Receptor HI Total 8E+00					

4E-04

Receptor HI Total

8E+00

Total Liver HI Across All Media =	1E-01
Total Kidney HI Across All Media =	4E-01
Total Nervous System Effects HI Across All Media =	4E-01
Total Lymphocyte Effects HI Across All Media =	6E-02
Total Nasal/Respiratory Effects HI Across All Media =	2E+00
Total Ocular Effects HI Across All Media =	2E-01
Total Other Effects HI Across All Media =	5E+00

TABLE 9.3a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	4E-07	--	4E-08	5E-07	Developmental effects	8E-03	--	7E-04	9E-03
			ALUMINUM	--	--	--	--	Neurotoxicity	1E-03	--	--	1E-03
			ARSENIC	8E-07	--	7E-08	9E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-03	--	5E-04	6E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-03	--	6E-04	5E-03
			CHROMIUM	--	--	--	--	--	1E-02	--	--	1E-02
			COPPER	--	--	--	--	Gastrointestinal effects	8E-04	--	--	8E-04
			IRON	--	--	--	--	Gastrointestinal effects	5E-03	--	--	5E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	6E-04	--	--	6E-04
			MERCURY	--	--	--	--	Autoimmune effects	2E-03	--	--	2E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	4E-04	--	--	4E-04
			HIGHLY CHLORINATED PCBs	2E-07	--	7E-08	2E-07	--	1E-02	--	5E-03	2E-02
			LESS CHLORINATED PCBs	5E-09	--	2E-09	8E-09	--	1E-04	--	4E-05	2E-04
			ACENAPHTHYLENE	--	--	--	--	--	2E-05	--	8E-06	3E-05
			BENZ(A)ANTHRACENE	6E-07	--	2E-07	8E-07	--	--	--	--	--
			BENZO(A)PYRENE	5E-06	--	2E-06	6E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-07	--	2E-07	9E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-05	--	8E-06	3E-05
			BENZO(K)FLUORANTHENE	2E-08	--	8E-09	3E-08	--	--	--	--	--
			CHRYSENE	6E-09	--	2E-09	9E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-07	--	2E-07	6E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E-04	--	1E-04	5E-04
			INDENO(1,2,3-CD)PYRENE	1E-07	--	5E-08	2E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-05	--	9E-06	3E-05
			PHENANTHRENE	--	--	--	--	--	1E-04	--	5E-05	2E-04
			BENZENE	9E-12	--	--	9E-12	Reduced lymphocyte count	1E-07	--	--	1E-07
			Chemical Total	8E-06	--	3E-06	1E-05		5E-02	--	7E-03	6E-02
		Exposure Point Total										6E-02
	Exposure Medium Total											6E-02
Medium Total												6E-02
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-05	--	2E-05
			ARSENIC	--	1E-10	--	1E-10	Development, cardiovascular, nervous system	--	2E-06	--	2E-06
			CADMIUM	--	2E-10	--	2E-10	--	--	--	--	--
			CHROMIUM	--	9E-09	--	9E-09	--	--	2E-05	--	2E-05
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	9E-05	--	9E-05
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	8E-08	--	8E-08
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	3E-12	--	3E-12	--	--	--	--	--

TABLE 9.3a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	LESS CHLORINATED PCBs	--	8E-14	--	8E-14	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	3E-13	--	3E-13	Nasal/respiratory (P)	--	8E-09	--	8E-09
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	9E-11	--	9E-11	Decreased lymphocyte count	--	1E-06	--	1E-06
	Chemical Total	--	9E-09	--	9E-09		--	1E-04	--	1E-04		
	Exposure Point Total				9E-09					1E-04		
	Exposure Medium Total				9E-09					1E-04		
Medium Total				9E-09					1E-04			
Shallow Ground Water	Shallow Ground Water	Exposure Unit 9	ALUMINUM	--	--	--	--	Neurotoxicity	--	--	9E-04	9E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	2E-03	2E-03
			ARSENIC	--	--	2E-07	2E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	1E-03	1E-03
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	1E-03	1E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	9E-03	9E-03
			CHROMIUM	--	--	--	--	--	--	--	9E-02	9E-02
			COPPER	--	--	--	--	Gastrointestinal effects	--	--	2E-04	2E-04
			IRON	--	--	--	--	Gastrointestinal effects	--	--	1E-03	1E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	4E-03	4E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	2E-03	2E-03
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	--	--	3E-04	3E-04
			SELENIUM	--	--	--	--	Clinical selenosis	--	--	4E-05	4E-05
			THALLIUM	--	--	--	--	Hematological effects	--	--	6E-03	6E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	6E-03	6E-03
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	2E-05	2E-05
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	2E-05	2E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	3E-04	3E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	4E-05	4E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.3a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Shallow Ground Water	Shallow Ground Water	Exposure Unit 9	BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	5E-08	5E-08	Increased relative liver weight (H)	--	--	5E-04	5E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	2E-07	2E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	5E-03	5E-03
			PHENANTHRENE	--	--	--	--	--	--	--	7E-04	7E-04
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	6E-10	6E-10	Liver	--	--	4E-06	4E-06
			BENZENE	--	--	5E-09	5E-09	Reduced lymphocyte count	--	--	7E-05	7E-05
			Chemical Total	--	--	4E-04	4E-04		--	--	1E-01	1E-01
		Exposure Point Total										1E-01
	Exposure Medium Total										1E-01	
Medium Total										1E-01		
Receptor Total										2E-01		

Total Risk Across All Media = 4E-04

Total Hazard Across All Media = 2E-01

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	4E-07	--	1E-07	5E-07	Developmental effects	2E-01	--	5E-02	2E-01
			ARSENIC	2E-07	--	5E-08	2E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	8E-03	4E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	2E-03	--	--	2E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	4E-03	--	1E-03	5E-03
			CHROMIUM	--	--	--	--	None Reported (O)	5E-02	--	--	5E-02
			IRON	--	--	--	--	Gastrointestinal effects	5E-02	--	--	5E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	8E-03	--	--	8E-03
			MERCURY	--	--	--	--	Autoimmune effects	6E-02	--	--	6E-02
			THALLIUM	--	--	--	--	Hematological effects	2E-02	--	--	2E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	4E-03	--	--	4E-03
			HIGHLY CHLORINATED PCBs	6E-08	--	8E-08	1E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	1E-01	3E-01
			DELTA-BHC	--	--	--	--	--	--	--	--	--
			DIELDRIN	1E-08	--	--	1E-08	Hepatic (H)	9E-04	--	--	9E-04
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	--	--	--	--
			HEPTACHLOR EPOXIDE	--	--	--	--	Increased liver-to-body weight ratio in males and females (H)	--	--	--	--
			1-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E+00	--	2E+00	4E+00
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	2E-02	--	3E-02	5E-02
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	6E-02	--	7E-02	1E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	4E-03	--	4E-03	8E-03
			BENZ(A)ANTHRACENE	6E-06	--	7E-06	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	1E-05	--	2E-05	3E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-06	--	2E-06	5E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-03	--	3E-03	6E-03
			BENZO(K)FLUORANTHENE	8E-08	--	9E-08	2E-07	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	--	--	Increased relative liver weight (H)	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	5E-08	--	5E-08	1E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-06	--	3E-06	5E-06	--	--	--	--	--

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E+00	--	2E+00	5E+00
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	5E-02	--	6E-02	1E-01
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	7E-02	--	6E-02	1E-01
			HEXACHLOROBENZENE	6E-09	--	6E-09	1E-08	Hepatic (H)	3E-04	--	3E-04	7E-04
			INDENO(1,2,3-CD)PYRENE	7E-07	--	9E-07	2E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+00	--	1E+00	2E+00
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-01	--	2E-01	4E-01
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	5E-02	--	6E-02	1E-01
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	6E-11	--	--	6E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1E-04	--	--	1E-04
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	--	--
			1,3,5-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	4E-09	--	--	4E-09	Liver	7E-04	--	--	7E-04
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	4E-02	--	--	4E-02
			BENZENE	2E-07	--	--	2E-07	Reduced lymphocyte count	5E-02	--	--	5E-02
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-03	--	--	2E-03
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	3E-03	--	--	3E-03
			METHYLENE CHLORIDE	3E-10	--	--	3E-10	Liver toxicity (H)	5E-05	--	--	5E-05
			N-HEXADACANE	--	--	--	--	--	--	--	--	--
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	2E-03	--	--	2E-03
			TOLUENE	--	--	--	--	Increased kidney weight (R)	1E-02	--	--	1E-02
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-02	--	--	1E-02
			Chemical Total	3E-05	--	3E-05	6E-05		6E+00	--	6E+00	1E+01
		Exposure Point Total					6E-05					1E+01
	Exposure Medium Total						6E-05					1E+01
Medium Total							6E-05					1E+01
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	4E-06	--	3E-07	4E-06	Developmental effects	2E+00	--	2E-01	2E+00
			ALUMINUM	--	--	--	--	Neurotoxicology	2E-02	--	--	2E-02
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	5E-03	--	--	5E-03
			ARSENIC	8E-07	--	7E-08	8E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	1E-02	1E-01

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	8E-03	--	--	8E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	7E-02	--	8E-03	7E-02
			CHROMIUM	--	--	--	--	--	1E-01	--	--	1E-01
			COPPER	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02
			IRON	--	--	--	--	Gastrointestinal effects	6E-02	--	--	6E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	7E-03	--	--	7E-03
			MERCURY	--	--	--	--	Autoimmune effects	1E-01	--	--	1E-01
			SILVER	--	--	--	--	Argyria (In)	1E-02	--	--	1E-02
			THALLIUM	--	--	--	--	Hematological effects	5E-02	--	--	5E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	7E-03	--	--	7E-03
			HIGHLY CHLORINATED PCBs	3E-07	--	1E-07	4E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	5E-01	--	2E-01	6E-01
			LESS CHLORINATED PCBs	3E-07	--	1E-07	4E-07	Reduced birth weights (W)	1E-01	--	6E-02	2E-01
			DIELDRIN	7E-09	--	--	7E-09	Hepatic (H)	6E-04	--	--	6E-04
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	5E-04	--	2E-04	7E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E-01	--	8E-02	3E-01
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	6E-04	--	2E-04	8E-04
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	4E-03	--	2E-03	6E-03
			ACENAPHTHYLENE	--	--	--	--	--	4E-03	--	2E-03	6E-03
			ANTHRACENE	--	--	--	--	No observed effects (O)	2E-03	--	6E-04	2E-03
			BENZ(A)ANTHRACENE	3E-06	--	1E-06	4E-06	--	--	--	--	--
			BENZO(A)PYRENE	2E-05	--	9E-06	3E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-06	--	1E-06	4E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-03	--	8E-04	3E-03
			BENZO(K)FLUORANTHENE	1E-07	--	5E-08	2E-07	--	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	3E-08	--	1E-08	4E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-06	--	9E-07	3E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-01	--	1E-01	4E-01
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	2E-02	--	8E-03	3E-02
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	1E-02	--	4E-03	2E-02
			HEXACHLOROBENZENE	5E-08	--	2E-08	7E-08	Hepatic (H)	3E-03	--	9E-04	4E-03
			INDENO(1,2,3-CD)PYRENE	7E-07	--	3E-07	1E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-01	--	6E-02	2E-01

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	PHENANTHRENE	--	--	--	--	--	5E-02	--	2E-02	7E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-02	--	9E-03	3E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	3E-09	--	--	3E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	5E-03	--	--	5E-03
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	3E-03	--	--	3E-03
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	3E-08	--	--	3E-08	Liver	5E-03	--	--	5E-03
			BENZENE	2E-08	--	--	2E-08	Reduced lymphocyte count	7E-03	--	--	7E-03
			BROMOMETHANE	--	--	--	--	Epithelial hyperplasia of the forestomach	2E-03	--	--	2E-03
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	1E-03	--	--	1E-03
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	9E-04	--	--	9E-04
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	7E-04	--	--	7E-04
			DODECANE	--	--	--	--	--	--	--	--	--
	Chemical Total			4E-05	--	1E-05	5E-05	4E+00			--	7E-01
Exposure Point Total			5E-05				4E+00					
Exposure Medium Total			5E-05				4E+00					
Medium Total			5E-05				4E+00					
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E+00	--	1E+00
			ANTIMONY	--	--	--	--	--	--	--	--	--
			ARSENIC	--	7E-07	--	7E-07	Development, cardiovascular, nervous system	--	2E-01	--	2E-01
			CADMIUM	--	5E-07	--	5E-07	--	--	--	--	--
			CHROMIUM	--	2E-05	--	2E-05	--	1E+00	--	1E+00	
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	6E+00	--	6E+00
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	4E-02	--	4E-02
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-08	--	2E-08	--	--	--	--	--
			LESS CHLORINATED PCBs	--	2E-08	--	2E-08	--	--	--	--	--
			DIELDRIN	--	6E-10	--	6E-10	--	--	--	--	--
2,4-DIMETHYLPHENOL	--	--	--	--	--	--	--	--	--			
2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--			

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	3&4-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORANTHENE	--	--	--	--	--	--	--	--	--
			FLUORENE	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	5E-09	--	5E-09	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	5E-07	--	5E-07	Nasal/respiratory (P)	--	3E-01	--	3E-01
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			PYRENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	2E-06	--	2E-06	Liver	--	2E-02	--	2E-02
			BENZENE	--	5E-07	--	5E-07	Decreased lymphocyte count	--	2E-01	--	2E-01
			BROMOMETHANE	--	--	--	--	Nasal lesions and membrane degeneration	--	1E-01	--	1E-01
			TOLUENE	--	--	--	--	Neurological effects	--	2E-03	--	2E-03
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	7E-01	--	7E-01
			DODECANE	--	--	--	--	--	--	--	--	--
		Chemical Total			2E-05		2E-05			1E+01		1E+01
		Exposure Point Total					2E-05					1E+01
	Exposure Medium Total						2E-05					1E+01
Medium Total							2E-05					1E+01
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	ALUMINUM	--	--	--	--	Neurotoxicology	--	--	9E-04	9E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	1E-02	1E-02
			ARSENIC	--	--	4E-08	4E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	6E-03	6E-03
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	7E-02	7E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	2E-02	2E-02
			CHROMIUM	--	--	--	--	--	--	--	1E-01	1E-01
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	--	--	3E-04	3E-04

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	IRON	--	--	--	--	Gastrointestinal effects	--	--	4E-03	4E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	6E-02	6E-02
			MERCURY	--	--	--	--	Autoimmune effects	--	--	2E-02	2E-02
			SILVER	--	--	--	--	Argyria (In)	--	--	2E-03	2E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	1E-02	1E-02
			HIGHLY CHLORINATED PCBs	--	--	--	--	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	--	--	--	--
			4,4'-DDT	--	--	1E-08	1E-08	Liver lesions (H)	--	--	6E-03	6E-03
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	--	--	--	--
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	--	--	3E-05	3E-05
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	1E-04	1E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	--	--	8E-05	8E-05
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	2E-04	2E-04
			4-METHYLPHENOL	--	--	--	--	--	--	--	1E-04	1E-04
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ANTHRACENE	--	--	--	--	No observed effects (O)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	7E-08	7E-08	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-06	1E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	7E-08	7E-08	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	8E-11	8E-11	Increased relative liver weight (H)	--	--	2E-05	2E-05
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	6E-10	6E-10	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	5E-07	5E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	--	--	7E-04	7E-04
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			HEXACHLOROBUTADIENE	--	--	4E-11	4E-11	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	1E-07	1E-07	--	--	--	--	--

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	3E-03	3E-03		
			PHENANTHRENE	--	--	--	--	--	--	--	1E-03	1E-03		
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	--	--	1E-05	1E-05		
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--		
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLOROBENZENE	--	--	6E-11	6E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	--	--	1E-04	1E-04		
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	1E-04	1E-04		
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,4-DICHLOROBENZENE	--	--	7E-10	7E-10	Liver	--	--	1E-04	1E-04		
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	--	--	--	--		
			BENZENE	--	--	3E-09	3E-09	Reduced lymphocyte count	--	--	1E-03	1E-03		
			BROMODICHLOROMETHANE	--	--	8E-13	8E-13	Renal cytomegaly (R)	--	--	4E-08	4E-08		
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	--	--	8E-05	8E-05		
			CHLOROFORM	--	--	--	--	Moderate/marked fatty cyst formation in the liver and elevated SGPT	--	--	4E-07	4E-07		
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	--	--	1E-05	1E-05		
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	--	--	--	--		
			METHYLENE CHLORIDE	--	--	3E-12	3E-12	Liver toxicity (H)	--	--	4E-07	4E-07		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--		
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	--	--	1E-05	1E-05		
			TETRACHLOROETHENE	--	--	3E-11	3E-11	Hepatotoxicity in mice (H), weight gain in rats	--	--	4E-07	4E-07		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	2E-04	2E-04		
			VINYL CHLORIDE	--	--	2E-11	2E-11	Liver cell polymorphism (H)	--	--	7E-07	7E-07		
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--		
					Chemical Total	--	--	2E-06	2E-06		--	--	3E-01	3E-01
					Exposure Point Total				2E-06					3E-01
		Exposure Medium Total					2E-06					3E-01		
Medium Total						2E-06					3E-01			

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	8E-03	8E-03
			ARSENIC	--	--	2E-08	2E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	3E-03	3E-03
			CHROMIUM	--	--	--	--	--	--	--	4E-02	4E-02
			IRON	--	--	--	--	Gastrointestinal effects	--	--	2E-03	2E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	2E-02	2E-02
			MERCURY	--	--	--	--	Autoimmune effects	--	--	1E-03	1E-03
			THALLIUM	--	--	--	--	Hematological effects	--	--	1E-02	1E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	2E-03	2E-03
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	2E-04	2E-04
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	8E-03	8E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	2E-03	2E-03
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	7E-06	7E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	6E-05	6E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	9E-06	9E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	2E-08	2E-08	Increased relative liver weight (H)	--	--	6E-03	6E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	5E-08	5E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	4E-06	4E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	7E-01	7E-01
			PHENANTHRENE	--	--	--	--	--	--	--	2E-02	2E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	7E-09	7E-09	Liver	--	--	1E-03	1E-03
			BENZENE	--	--	1E-07	1E-07	Reduced lymphocyte count	--	--	4E-02	4E-02
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--

TABLE 9.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	2E-02	2E-02
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total			8E-05	8E-05				9E-01	9E-01
		Exposure Point Total					8E-05					9E-01
	Exposure Medium Total						8E-05					9E-01
Medium Total							8E-05					9E-01
Receptor Total							2E-04					Receptor HI Total 3E+01

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 3E+01

Total Liver HI Across All Media =	2E-01
Total Kidney HI Across All Media =	9E-01
Total Nervous System Effects HI Across All Media =	8E+00
Total Lymphocyte Effects HI Across All Media =	3E-01
Total Nasal/Respiratory Effects HI Across All Media =	5E+00
Total Ocular Effects HI Across All Media =	9E-01
Total Other Effects HI Across All Media =	1E+01

TABLE 9.4a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	2E-07	--	2E-08	2E-07	Developmental effects	1E-01	--	9E-03	1E-01
			ALUMINUM	--	--	--	--	Neurotoxicology	2E-02	--	--	2E-02
			ARSENIC	4E-07	--	4E-08	5E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	6E-02	--	6E-03	7E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	6E-02	--	7E-03	6E-02
			CHROMIUM	--	--	--	--	--	2E-01	--	--	2E-01
			COPPER	--	--	--	--	Gastrointestinal effects	9E-03	--	--	9E-03
			IRON	--	--	--	--	Gastrointestinal effects	6E-02	--	--	6E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	8E-03	--	--	8E-03
			MERCURY	--	--	--	--	Autoimmune effects	2E-02	--	--	2E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	5E-03	--	--	5E-03
			HIGHLY CHLORINATED PCBs	9E-08	--	4E-08	1E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-01	--	6E-02	2E-01
			LESS CHLORINATED PCBs	3E-09	--	1E-09	4E-09	Reduced birth weights (W)	1E-03	--	6E-04	2E-03
			ACENAPHTHYLENE	--	--	--	--	--	3E-04	--	1E-04	4E-04
			BENZ(A)ANTHRACENE	3E-07	--	1E-07	4E-07	--	--	--	--	--
			BENZO(A)PYRENE	2E-06	--	9E-07	3E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-07	--	1E-07	4E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	3E-04	--	1E-04	4E-04
			BENZO(K)FLUORANTHENE	1E-08	--	4E-09	1E-08	--	--	--	--	--
			CHRYSENE	3E-09	--	1E-09	4E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-07	--	8E-08	3E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E-03	--	1E-03	6E-03
			INDENO(1,2,3-CD)PYRENE	6E-08	--	2E-08	9E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	3E-04	--	1E-04	4E-04
			PHENANTHRENE	--	--	--	--	--	2E-03	--	6E-04	2E-03
			BENZENE	5E-12	--	--	5E-12	Reduced lymphocyte count	1E-06	--	--	1E-06
			Chemical Total	4E-06	--	1E-06	5E-06		7E-01	--	9E-02	7E-01
		Exposure Point Total					5E-06					7E-01
	Exposure Medium Total						5E-06					7E-01
Medium Total							5E-06					7E-01
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	4E-04	--	4E-04
			ARSENIC	--	1E-10	--	1E-10	Development, cardiovascular, nervous system	--	5E-05	--	5E-05
			CADMIUM	--	2E-10	--	2E-10	--	--	--	--	--
			CHROMIUM	--	1E-08	--	1E-08	--	--	6E-04	--	6E-04

TABLE 9.4a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	
			LEAD	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E-03	--	2E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-06	--	2E-06
			VANADIUM	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	3E-12	--	3E-12	--	--	--	--	
			LESS CHLORINATED PCBs	--	9E-14	--	9E-14	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	
			NAPHTHALENE	--	3E-13	--	3E-13	Nasal/respiratory (P)	--	2E-07	--	2E-07
			PHENANTHRENE	--	--	--	--	--	--	--	--	
		BENZENE	--	9E-11	--	9E-11	Decreased lymphocyte count	--	3E-05	--	3E-05	
Chemical Total			--	1E-08	--	1E-08	--	4E-03	--	4E-03		
Exposure Point Total			1E-08				4E-03					
Exposure Medium Total			1E-08				4E-03					
Medium Total			1E-08				4E-03					
Shallow Ground Water	Shallow Ground Water	Exposure Unit 9	ALUMINUM	--	--	--	--	Neurotoxicology	--	--	1E-02	1E-02
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	2E-02	2E-02
			ARSENIC	--	--	1E-07	1E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	2E-02	2E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	2E-02	2E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	1E-01	1E-01
			CHROMIUM	--	--	--	--	--	--	--	1E+00	1E+00
			COPPER	--	--	--	--	Gastrointestinal effects	--	--	2E-03	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	2E-02	2E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	5E-02	5E-02
			MERCURY	--	--	--	--	Autoimmune effects	--	--	3E-02	3E-02
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	--	--	4E-03	4E-03
			SELENIUM	--	--	--	--	Clinical selenosis	--	--	5E-04	5E-04
			THALLIUM	--	--	--	--	Hematological effects	--	--	7E-02	7E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	8E-02	8E-02
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	2E-04	2E-04
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--

TABLE 9.4a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Shallow Ground Water	Shallow Ground Water	Exposure Unit 9	ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	9E-06	9E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	2E-04	2E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-05	2E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	2E-08	2E-08	Increased relative liver weight (H)	--	--	6E-03	6E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	9E-08	9E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	7E-02	7E-02
			PHENANTHRENE	--	--	--	--	--	--	--	9E-03	9E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	3E-10	3E-10	Liver	--	--	5E-05	5E-05
			BENZENE	--	--	3E-09	3E-09	Reduced lymphocyte count	--	--	9E-04	9E-04
		Chemical Total	--	--	2E-04	2E-04		--	--	2E+00	2E+00	
Exposure Point Total			2E-04				2E+00					
Exposure Medium Total			2E-04				2E+00					
Medium Total			2E-04				2E+00					
Receptor Total			2E-04				Receptor HI Total 2E+00					

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 2E+00

Total Liver HI Across All Media = 6E-03
Total Kidney HI Across All Media = 2E-01
Total Nervous System Effects HI Across All Media = 2E-01
Total Lymphocyte Effects HI Across All Media = 9E-04
Total Nasal/Respiratory Effects HI Across All Media = 2E-07
Total Other Effects HI Across All Media = 2E+00

TABLE 9.5 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Surveillance Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Soil	Surface Soil	Exposure Unit 2	2,3,7,8-TCDD Equivalent	3E-06	--	2E-07	4E-06	Developmental effects	6E-02	--	3E-03	7E-02	
			ALUMINUM	--	--	--	--	Neurotoxicity	1E-03	--	--	1E-03	
			ARSENIC	7E-07	--	4E-08	7E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	4E-03	--	2E-04	5E-03	
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	3E-04	--	--	3E-04	
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	6E-03	--	4E-04	7E-03	
			CHROMIUM	--	--	--	--	--	6E-03	--	--	6E-03	
			COPPER	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03	
			IRON	--	--	--	--	Gastrointestinal effects	3E-03	--	--	3E-03	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	CNS (N)	3E-04	--	--	3E-04	
			MERCURY	--	--	--	--	Autoimmune effects	7E-03	--	--	7E-03	
			SILVER	--	--	--	--	Argyria (In)	6E-04	--	--	6E-04	
			THALLIUM	--	--	--	--	Hematological effects	1E-03	--	--	1E-03	
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-04	--	--	3E-04	
			HIGHLY CHLORINATED PCBs	2E-07	--	4E-08	2E-07	--	1E-02	--	3E-03	1E-02	
			LESS CHLORINATED PCBs	7E-08	--	2E-08	9E-08	--	1E-03	--	4E-04	2E-03	
			DIELDRIN	2E-07	--	--	2E-07	Hepatic (H)	6E-04	--	--	6E-04	
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	6E-04	--	1E-04	7E-04	
			ACENAPHTHYLENE	--	--	--	--	--	2E-05	--	4E-06	2E-05	
			BENZ(A)ANTHRACENE	6E-08	--	1E-08	8E-08	--	--	--	--	--	
			BENZO(A)PYRENE	7E-07	--	2E-07	8E-07	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	9E-08	--	2E-08	1E-07	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	6E-06	--	1E-06	7E-06	
			BENZO(K)FLUORANTHENE	4E-09	--	1E-09	5E-09	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	1E-07	--	3E-08	2E-07	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E-04	--	7E-05	5E-04	
			HEXACHLOROBENZENE	8E-08	--	1E-08	1E-07	Hepatic (H)	2E-04	--	3E-05	2E-04	
			INDENO(1,2,3-CD)PYRENE	4E-08	--	9E-09	5E-08	--	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-04	--	4E-05	2E-04	
			PHENANTHRENE	--	--	--	--	--	6E-05	--	1E-05	7E-05	
			1,2,3TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--	
			1,2,4-TRICHLOROBENZENE	9E-10	--	--	9E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	7E-05	--	--	7E-05	
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	3E-05	--	--	3E-05	
			1,4-DICHLOROBENZENE	1E-08	--	--	1E-08	Liver	7E-05	--	--	7E-05	
			BENZENE	2E-09	--	--	2E-09	Reduced lymphocyte count	2E-05	--	--	2E-05	
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--	
						Chemical Total	6E-06	--	5E-07	6E-06			
					Exposure Point Total								1E-01
					Exposure Medium Total								1E-01
	Medium Total							6E-06					1E-01

TABLE 9.5 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Surveillance Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 2	2,3,7,8-TCDD Equivalent	--	--	--	--	Development, cardiovascular, nervous system	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-05	--	2E-05
			ARSENIC	--	2E-10	--	2E-10	Development, cardiovascular, nervous system	--	2E-06	--	2E-06
			BARIUM	--	--	--	--	Renal toxicity	--	1E-05	--	1E-05
			CADMIUM	--	3E-10	--	3E-10	--	--	--	--	--
			CHROMIUM	--	6E-09	--	6E-09	--	--	1E-05	--	1E-05
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	8E-05	--	8E-05
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	5E-07	--	5E-07
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	4E-12	--	4E-12	--	--	--	--	--
			LESS CHLORINATED PCBs	--	2E-12	--	2E-12	--	--	--	--	--
			DIELDRIN	--	4E-12	--	4E-12	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	2E-12	--	2E-12	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	4E-12	--	4E-12	Nasal/respiratory (P)	--	1E-07	--	1E-07
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	6E-04	--	6E-04
			1,4-DICHLOROBENZENE	--	7E-07	--	7E-07	Liver	--	2E-04	--	2E-04
			BENZENE	--	4E-08	--	4E-08	Decreased lymphocyte count	--	5E-04	--	5E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			Chemical Total	--	7E-07	--	7E-07		--	1E-03	--	1E-03
		Exposure Point Total					7E-07					1E-03
	Exposure Medium Total						7E-07					1E-03
Medium Total							7E-07					1E-03
Receptor Total							7E-06					1E-01

Total Risk Across All Media = 7E-06

Total Hazard Across All Media = 1E-01

TABLE 9.6 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Ditch Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 3	BENZENE	--	1E-08	--	1E-08	Decreased lymphocyte count	--	1E-04	--	1E-04
			Chemical Total	--	1E-08	--	1E-08	--	1E-04	--	1E-04	
		Exposure Point Total	1E-08				1E-04					
	Exposure Medium Total			1E-08				1E-04				
Medium Total				1E-08				1E-04				
Sediment	Surface Sediment	Exposure Unit 3	2,3,7,8-TCDD Equivalent	6E-08	--	2E-08	8E-08	--	1E-03	--	3E-04	2E-03
			ARSENIC	3E-07	--	7E-08	3E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-03	--	4E-04	2E-03
			CHROMIUM	--	--	--	--	--	7E-03	--	--	7E-03
			IRON	--	--	--	--	Gastrointestinal effects	3E-03	--	--	3E-03
			MANGANESE	--	--	--	--	CNS (N)	3E-04	--	--	3E-04
			MERCURY	--	--	--	--	Autoimmune effects	3E-04	--	--	3E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-04	--	--	2E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	5E-04	--	6E-04	1E-03
			ACENAPHTHYLENE	--	--	--	--	--	2E-05	--	2E-05	3E-05
			BENZ(A)ANTHRACENE	5E-08	--	5E-08	1E-07	--	--	--	--	--
			BENZO(A)PYRENE	5E-07	--	5E-07	1E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	5E-08	--	6E-08	1E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-06	--	6E-06	1E-05
			DIBENZ(A,H)ANTHRACENE	3E-08	--	4E-08	7E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E-04	--	4E-04	9E-04
			INDENO(1,2,3-CD)PYRENE	3E-08	--	4E-08	7E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	4E-04	--	5E-04	9E-04
			PHENANTHRENE	--	--	--	--	--	4E-05	--	5E-05	1E-04
			BENZENE	2E-09	--	--	2E-09	Reduced lymphocyte count	3E-05	--	--	3E-05
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			Chemical Total	9E-07	--	8E-07	2E-06		1E-02	--	2E-03	2E-02
		Exposure Point Total			2E-06				2E-02			
	Exposure Medium Total			2E-06				2E-02				
Medium Total				2E-06				2E-02				
Surface Water	Surface Water	Exposure Unit 3	CHROMIUM	--	--	--	--	--	--	--	3E-03	3E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	2E-05	2E-05
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	2E-07	2E-07
			MERCURY	--	--	--	--	Autoimmune effects	--	--	1E-04	1E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	2E-04	2E-04
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	2E-05	2E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	3E-04	3E-04
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.6 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Ditch Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 3	CARBAZOLE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	2E-02	2E-02
			PHENANTHRENE	--	--	--	--	--	--	--	9E-04	9E-04
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	--	2E-07	2E-07	Reduced lymphocyte count	--	--	3E-03	3E-03
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	6E-04	6E-04
		XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--	
		Chemical Total	--	--	2E-07	2E-07		--	--	3E-02	3E-02	
		Exposure Point Total				2E-07					3E-02	
		Exposure Medium Total				2E-07					3E-02	
Medium Total						2E-07					3E-02	
Receptor Total						2E-06		Receptor HI Total			4E-02	

Total Risk Across All Media = 2E-06

Total Hazard Across All Media = 4E-02

TABLE 9.7 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Railroad Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 4	ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	3E-04	--	3E-04
			ARSENIC	--	3E-09	--	3E-09	Development, cardiovascular, nervous system	--	4E-05	--	4E-05
			BARIUM	--	--	--	--	Renal toxicity	--	9E-05	--	9E-05
			CHROMIUM	--	1E-08	--	1E-08	--	--	3E-05	--	3E-05
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-03	--	1E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	5E-07	--	5E-07
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-12	--	2E-12	--	--	--	--	--
			LESS CHLORINATED PCBs	--	1E-13	--	1E-13	--	--	--	--	--
			DIELDRIN	--	1E-11	--	1E-11	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	2E-10	--	2E-10	Decreased lymphocyte count	--	2E-06	--	2E-06
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
	Chemical Total			--	2E-08	--	2E-08		--	2E-03	--	2E-03
Exposure Point Total							2E-08					2E-03
Exposure Medium Total							2E-08					2E-03
Medium Total							2E-08					2E-03
Soil	Surface Soil	Exposure Unit 4	ALUMINUM	--	--	--	--	Neurotoxicity	7E-03	--	--	7E-03
			ARSENIC	5E-06	--	1E-06	7E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	7E-03	4E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-03	--	--	1E-03
			CHROMIUM	--	--	--	--	--	5E-03	--	--	5E-03
			IRON	--	--	--	--	Gastrointestinal effects	2E-02	--	--	2E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	2E-03	--	--	2E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	3E-08	--	3E-08	6E-08	--	2E-03	--	2E-03	4E-03
			LESS CHLORINATED PCBs	2E-09	--	1E-09	3E-09	--	3E-05	--	3E-05	6E-05
			DIELDRIN	2E-07	--	--	2E-07	--	7E-04	--	--	7E-04
			ACENAPHTHYLENE	--	--	--	--	Hepatic (H)	4E-06	--	4E-06	8E-06
			BENZ(A)ANTHRACENE	8E-08	--	7E-08	1E-07	--	--	--	--	--
			BENZO(A)PYRENE	8E-07	--	7E-07	1E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-07	--	8E-08	2E-07	--	--	--	--	--

TABLE 9.7 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Railroad Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 4	BENZO(G,H,I)PERYLENE	--	--	--	--	--	6E-06	--	5E-06	1E-05
			BENZO(K)FLUORANTHENE	6E-09	--	5E-09	1E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-07	--	2E-07	5E-07	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	5E-08	--	4E-08	9E-08	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	1E-05	--	1E-05	2E-05
			BENZENE	1E-11	--	--	1E-11	Reduced lymphocyte count	2E-07	--	--	2E-07
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			Chemical Total	7E-06	--	2E-06	9E-06	--	7E-02	--	9E-03	8E-02
		Exposure Point Total					9E-06					8E-02
	Exposure Medium Total						9E-06					8E-02
Medium Total							9E-06					8E-02
Receptor Total							9E-06				Receptor HI Total	8E-02

Total Risk Across All Media = 9E-06

Total Hazard Across All Media = 8E-02

TABLE 9.7a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	6E-05	--	6E-05
			ARSENIC	--	5E-10	--	5E-10	Development, cardiovascular, nervous system	--	7E-06	--	7E-06
			CADMIUM	--	6E-10	--	6E-10	--	--	--	--	
			CHROMIUM	--	3E-08	--	3E-08	--	--	7E-05	--	7E-05
			COPPER	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	4E-04	--	4E-04
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	3E-07	--	3E-07
			VANADIUM	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	1E-11	--	1E-11	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	
			PHENANTHRENE	--	--	--	--	--	--	--	--	
			BENZENE	--	2E-10	--	2E-10	Decreased lymphocyte count	--	2E-06	--	2E-06
	Chemical Total			--	3E-08	--	3E-08	--			5E-04	
Exposure Point Total			3E-08				5E-04					
Exposure Medium Total			3E-08				5E-04					
Medium Total			3E-08				5E-04					
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	1E-06	--	2E-07	1E-06	--	2E-02	--	5E-03	3E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	4E-03	--	--	4E-03
			ARSENIC	2E-06	--	5E-07	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	3E-03	2E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-02	--	3E-03	2E-02
			CHROMIUM	--	--	--	--	--	3E-02	--	--	3E-02
			COPPER	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	5E-03	--	--	5E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-03	--	--	1E-03
			HIGHLY CHLORINATED PCBs	5E-07	--	5E-07	9E-07	--	3E-02	--	3E-02	7E-02
			ACENAPHTHYLENE	--	--	--	--	--	5E-05	--	4E-05	9E-05
			BENZ(A)ANTHRACENE	2E-06	--	2E-06	3E-06	--	--	--	--	--

TABLE 9.7a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	BENZO(A)PYRENE	1E-05	--	1E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-06	--	2E-06	3E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	6E-05	--	5E-05	1E-04
			BENZO(K)FLUORANTHENE	6E-08	--	5E-08	1E-07	--	--	--	--	--
			CHRYSENE	2E-08	--	2E-08	3E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-03	--	8E-04	2E-03
			INDENO(1,2,3-CD)PYRENE	3E-07	--	3E-07	6E-07	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	4E-04	--	3E-04	7E-04
			BENZENE	1E-11	--	--	1E-11	Reduced lymphocyte count	2E-07	--	--	2E-07
			Chemical Total	2E-05	--	2E-05	4E-05		1E-01	--	4E-02	2E-01
		Exposure Point Total					4E-05					2E-01
	Exposure Medium Total						4E-05					2E-01
Medium Total							4E-05					2E-01
Receptor Total							4E-05				Receptor HI Total	2E-01

Total Risk Across All Media = 4E-05

Total Hazard Across All Media = 2E-01

TABLE 9.8 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 5	ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E-03	--	1E-03	
			ANTIMONY	--	--	--	--	--	--	--	--	--	
			ARSENIC	--	2E-08	--	2E-08	Development, cardiovascular, nervous system	--	2E-04	--	2E-04	
			CHROMIUM	--	1E-07	--	1E-07	--	--	3E-04	--	3E-04	
			IRON	--	--	--	--	--	--	--	--	--	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	5E-03	--	5E-03	
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	6E-06	--	6E-06	
			THALLIUM	--	--	--	--	--	--	--	--	--	
			VANADIUM	--	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	9E-10	--	9E-10	--	--	--	--	--	
			ENDOSULFAN SULFATE	--	--	--	--	--	--	--	--	--	
			ENDRIN ALDEHYDE	--	--	--	--	--	--	--	--	--	
			3&4-METHYLPHENOL	--	--	--	--	--	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--	
			FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--	
			NAPHTHALENE	--	7E-11	--	7E-11	Nasal/respiratory (P)	--	2E-06	--	2E-06	
			PHENANTHRENE	--	--	--	--	--	--	--	--	--	
			BENZENE	--	4E-09	--	4E-09	Decreased lymphocyte count	--	5E-05	--	5E-05	
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--	
					Chemical Total	--	1E-07	--	1E-07		--	6E-03	--
			Exposure Point Total										6E-03
			Exposure Medium Total										6E-03
												6E-03	
Medium Total												6E-03	
Soil	Surface Soil	Exposure Unit 5	ALUMINUM	--	--	--	--	Neurotoxicology	7E-03	--	--	7E-03	
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	4E-03	--	--	4E-03	
			ARSENIC	8E-06	--	2E-06	1E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-02	--	1E-02	6E-02	
			CHROMIUM	--	--	--	--	--	1E-02	--	--	1E-02	
			IRON	--	--	--	--	Gastrointestinal effects	3E-02	--	--	3E-02	
			LEAD	--	--	--	--	--	--	--	--	0E+00	
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03	
			MERCURY	--	--	--	--	Autoimmune effects	8E-03	--	--	8E-03	
			THALLIUM	--	--	--	--	Hematological effects	1E-02	--	--	1E-02	
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-03	--	--	3E-03	
								Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-01	--	4E-01	7E-01	
			HIGHLY CHLORINATED PCBs	4E-06	--	6E-06	1E-05						

TABLE 9.8 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Soil	Surface Soil	Exposure Unit 5	ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	2E-05	--	--	2E-05	
			ENDRIN ALDEHYDE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	2E-04	--	--	2E-04	
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	9E-07	--	9E-07	2E-06	
			ACENAPHTHYLENE	--	--	--	--	--	5E-04	--	6E-04	1E-03	
			BENZ(A)ANTHRACENE	9E-06	--	1E-05	2E-05	--	--	--	--	--	
			BENZO(A)PYRENE	9E-05	--	1E-04	2E-04	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	8E-06	--	1E-05	2E-05	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-03	--	1E-03	2E-03	
			BENZO(K)FLUORANTHENE	1E-06	--	1E-06	2E-06	--	--	--	--	--	
			CHRYSENE	9E-08	--	1E-07	2E-07	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	3E-05	--	3E-05	6E-05	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	6E-03	--	6E-03	1E-02	
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	2E-03	--	2E-03	4E-03	
			INDENO(1,2,3-CD)PYRENE	7E-06	--	9E-06	2E-05	--	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	4E-04	--	5E-04	8E-04	
			PHENANTHRENE	--	--	--	--	--	1E-03	--	2E-03	3E-03	
			BENZENE	2E-10	--	--	2E-10	Reduced lymphocyte count	2E-06	--	--	2E-06	
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--	
					Chemical Total	2E-04	--	2E-04	3E-04		4E-01	--	4E-01
				Exposure Point Total				3E-04					9E-01
		Exposure Medium Total				3E-04					9E-01		
Medium Total						3E-04					9E-01		
Receptor Total						3E-04				Receptor HI Total	9E-01		

Total Risk Across All Media = 3E-04

Total Hazard Across All Media = 9E-01

TABLE 9.9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 7	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--	
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	3E-04	--	3E-04	
			ANTIMONY	--	--	--	--	--	--	--	--	--	
			ARSENIC	--	4E-09	--	4E-09	Development, cardiovascular, nervous system	--	5E-05	--	5E-05	
			BARIUM	--	--	--	--	Renal toxicity	--	2E-04	--	2E-04	
			CADMIUM	--	5E-09	--	5E-09	--	--	--	--	--	
			CHROMIUM	--	1E-07	--	1E-07	--	--	2E-04	--	2E-04	
			COPPER	--	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--	--	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-03	--	1E-03	
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	8E-06	--	8E-06	
			SILVER	--	--	--	--	--	--	--	--	--	
			THALLIUM	--	--	--	--	--	--	--	--	--	
			VANADIUM	--	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	7E-11	--	7E-11	--	--	--	--	--	
			LESS CHLORINATED PCBs	--	4E-11	--	4E-11	--	--	--	--	--	
			DIELDRIN	--	5E-11	--	5E-11	--	--	--	--	--	
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--	
			FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			HEXACHLOROBENZENE	--	3E-11	--	3E-11	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--	
			NAPHTHALENE	--	6E-11	--	6E-11	Nasal/respiratory (P)	--	2E-06	--	2E-06	
			PHENANTHRENE	--	--	--	--	--	--	--	--	--	
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--	
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--	
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	2E-03	--	2E-03	
			1,4-DICHLOROBENZENE	--	5E-06	--	5E-06	Liver	--	2E-03	--	2E-03	
			BENZENE	--	3E-07	--	3E-07	Decreased lymphocyte count	--	3E-03	--	3E-03	
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--	
			DODECANE	--	--	--	--	--	--	--	--	--	
			Chemical Total			--	5E-06	--	5E-06		--	9E-03	--
		Exposure Point Total							5E-06				
	Exposure Medium Total							5E-06					9E-03
Medium Total							5E-06					9E-03	

TABLE 9.9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 7	2,3,7,8-TCDD Equivalent	3E-05	--	8E-06	4E-05	Developmental effects	5E-01	--	2E-01	7E-01
			ALUMINUM	--	--	--	--	Neurotoxicology	7E-03	--	--	7E-03
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	2E-03	--	--	2E-03
			ARSENIC	5E-06	--	1E-06	6E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	9E-03	4E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-03	--	--	1E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-02	--	1E-02	4E-02
			CHROMIUM	--	--	--	--	--	3E-02	--	--	3E-02
			COPPER	--	--	--	--	Gastrointestinal effects	6E-03	--	--	6E-03
			IRON	--	--	--	--	Gastrointestinal effects	2E-02	--	--	2E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	3E-02	--	--	3E-02
			SILVER	--	--	--	--	Argyria (In)	2E-03	--	--	2E-03
			THALLIUM	--	--	--	--	Hematological effects	9E-03	--	--	9E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	1E-06	--	1E-06	2E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	7E-02	--	1E-01	2E-01
			LESS CHLORINATED PCBs	5E-07	--	7E-07	1E-06	Reduced birth weights (W)	1E-02	--	1E-02	2E-02
			DIELDRIN	6E-07	--	--	6E-07	Hepatic (H)	2E-03	--	--	2E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E-03	--	3E-03	6E-03
			ACENAPHTHYLENE	--	--	--	--	--	2E-04	--	3E-04	5E-04
			BENZ(A)ANTHRACENE	5E-06	--	6E-06	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	4E-05	--	6E-05	1E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-06	--	4E-06	7E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	4E-04	--	5E-04	9E-04
			BENZO(K)FLUORANTHENE	4E-07	--	5E-07	8E-07	--	--	--	--	--
			CHRYSENE	4E-08	--	6E-08	1E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	8E-06	--	1E-05	2E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E-03	--	4E-03	8E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	9E-04	--	1E-03	2E-03
			HEXACHLOROBENZENE	5E-07	--	5E-07	9E-07	Hepatic (H)	1E-03	--	1E-03	2E-03
			INDENO(1,2,3-CD)PYRENE	3E-06	--	3E-06	6E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	9E-04	--	1E-03	2E-03
			PHENANTHRENE	--	--	--	--	--	9E-04	--	1E-03	2E-03
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	4E-09	--	--	4E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-04	--	--	3E-04
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	7E-05	--	--	7E-05

TABLE 9.9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 7	1,4-DICHLOROBENZENE	4E-08	--	--	4E-08	Liver	3E-04	--	--	3E-04
			BENZENE	8E-09	--	--	8E-09	Reduced lymphocyte count	1E-04	--	--	1E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
		Chemical Total		1E-04	--	9E-05	2E-04		8E-01	--	3E-01	1E+00
		Exposure Point Total					2E-04					1E+00
	Exposure Medium Total						2E-04					1E+00
Medium Total							2E-04					1E+00
Ground Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicology	5E-01	--	--	5E-01
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	1E-01	--	--	1E-01
			ARSENIC	1E-04	--	--	1E-04	Hyperpigmentation (In); Vascular (V); PNS (N)	6E-01	--	--	6E-01
			BARIIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-01	--	--	1E-01
			BERYLLIUM	--	--	--	--	Small intestinal lesions	8E-03	--	--	8E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	4E-02	--	--	4E-02
			CHROMIUM	--	--	--	--	--	5E-01	--	--	5E-01
			COBALT	--	--	--	--	--	--	--	--	--
			COPPER	--	--	--	--	Gastrointestinal effects	5E-02	--	--	5E-02
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	3E-02	--	--	3E-02
			IRON	--	--	--	--	Gastrointestinal effects	1E+00	--	--	1E+00
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	3E-01	--	--	3E-01
			MERCURY	--	--	--	--	Autoimmune effects	1E-01	--	--	1E-01
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	5E-02	--	--	5E-02
			SELENIUM	--	--	--	--	Clinical selenosis	1E-02	--	--	1E-02
			SILVER	--	--	--	--	Argyria (In)	8E-03	--	--	8E-03
			THALLIUM	--	--	--	--	Hematological effects	2E+00	--	--	2E+00
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-01	--	--	1E-01
			ZINC	--	--	--	--	Decreased ESOD (B)	6E-03	--	--	6E-03
			HIGHLY CHLORINATED PCBs	1E-06	--	--	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	7E-02	--	--	7E-02
			4,4'-DDD	1E-07	--	--	1E-07	--	--	--	--	--
			4,4'-DDT	3E-06	--	--	3E-06	Liver lesions (H)	4E-02	--	--	4E-02
			ALDRIN	4E-06	--	--	4E-06	Liver toxicity (H)	2E-02	--	--	2E-02

TABLE 9.9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	ALPHA-BHC	8E-06	--	--	8E-06	--	--	--	--	--
			ENDOSULFAN II	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	2E-04	--	--	2E-04
			ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	7E-05	--	--	7E-05
			HEPTACHLOR EPOXIDE	6E-07	--	--	6E-07	Increased liver-to-body weight ratio in males and females (H)	2E-02	--	--	2E-02
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	5E-03	--	--	5E-03
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	6E-02	--	--	6E-02
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	4E+00	--	--	4E+00
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E+00	--	--	3E+00
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	4E-01	--	--	4E-01
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	2E+00	--	--	2E+00
			4-CHLORO-3-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			4-METHYLPHENOL	--	--	--	--	--	3E+00	--	--	3E+00
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	3E-02	--	--	3E-02
			ACENAPHTHYLENE	--	--	--	--	--	1E-01	--	--	1E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	7E-03	--	--	7E-03
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	3E-02	--	--	3E-02
			BENZ(A)ANTHRACENE	3E-04	--	--	3E-04	--	--	--	--	--
			BENZO(A)PYRENE	1E-03	--	--	1E-03	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-04	--	--	1E-04	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	3E-03	--	--	3E-03
			BENZO(K)FLUORANTHENE	9E-06	--	--	9E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	1E-06	--	--	1E-06	Increased relative liver weight (H)	1E-02	--	--	1E-02
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	2E-06	--	--	2E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-04	--	--	1E-04	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E+00	--	--	4E+00
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	8E-02	--	--	8E-02
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	9E-02	--	--	9E-02
			HEXACHLOROBUTADIENE	5E-07	--	--	5E-07	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	4E-05	--	--	4E-05	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	4E+00	--	--	4E+00
			NITROBENZENE	--	--	--	--	Hematologic (B), adrenal, renal (R) and hepatic (H) lesions	1E-01	--	--	1E-01
			PHENANTHRENE	--	--	--	--	--	3E-01	--	--	3E-01

TABLE 9.9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Ground Water	Potable Water	Exposure Unit 8	PHENOL	--	--	--	--	Decreased maternal weight gain (W)	1E-01	--	--	1E-01		
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	7E-02	--	--	7E-02		
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLOROBENZENE	3E-07	--	--	3E-07	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-02	--	--	3E-02		
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	1E-01	--	--	1E-01		
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,4-DICHLOROBENZENE	2E-05	--	--	2E-05	Liver	1E-01	--	--	1E-01		
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	2E-04	--	--	2E-04		
			ACETONE	--	--	--	--	Nephropathy	2E-03	--	--	2E-03		
			BENZENE	2E-03	--	--	2E-03	Reduced lymphocyte count	3E+01	--	--	3E+01		
			BROMODICHLOROMETHANE	1E-06	--	--	1E-06	Renal cytomegaly (R)	3E-03	--	--	3E-03		
			CARBON DISULFIDE	--	--	--	--	Fetal toxicity/malformations	2E-03	--	--	2E-03		
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-01	--	--	2E-01		
			CHLOROETHANE	--	--	--	--	--	--	--	--	--		
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	3E-02	--	--	3E-02		
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	8E-04	--	--	8E-04		
			METHYLENE CHLORIDE	4E-08	--	--	4E-08	Liver toxicity (H)	2E-04	--	--	2E-04		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--		
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	8E-02	--	--	8E-02		
			TETRACHLOROETHENE	1E-06	--	--	1E-06	Hepatotoxicity in mice (H), weight gain in rats	6E-04	--	--	6E-04		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	3E-01	--	--	3E-01		
			VINYL CHLORIDE	6E-06	--	--	6E-06	Liver cell polymorphism (H)	7E-03	--	--	7E-03		
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	9E-02	--	--	9E-02		
			Chemical Total			4E-03	--	--	4E-03		6E+01	--	--	6E+01
			Exposure Point Total							4E-03				
Exposure Medium Total							4E-03					6E+01		
Medium Total							4E-03					6E+01		
Receptor Total							4E-03	Receptor HI Total				6E+01		

Total Risk Across All Media = 4E-03

Total Hazard Across All Media = 6E+01

Total Liver HI Across All Media = 7E-01
Total Kidney HI Across All Media = 8E-01
Total Nervous System Effects HI Across All Media = 7E+00
Total Lymphocyte Effects HI Across All Media = 3E+01
Total Nasal/Respiratory Effects HI Across All Media = 3E+00
Total Ocular Effects HI Across All Media = 2E-01
Total Other Effects HI Across All Media = 2E+01

TABLE 9.9a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-04	--	2E-04
			ARSENIC	--	2E-09	--	2E-09	Development, cardiovascular, nervous system	--	2E-05	--	2E-05
			CADMIUM	--	2E-09	--	2E-09	--	--	--	--	--
			CHROMIUM	--	1E-07	--	1E-07	--	--	2E-04	--	2E-04
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-03	--	1E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	1E-06	--	1E-06
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	4E-11	--	4E-11	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
	BENZENE	--	6E-10	--	6E-10	Decreased lymphocyte count	--	8E-06	--	8E-06		
Chemical Total			--	1E-07	--	1E-07	--			2E-03		
Exposure Point Total			1E-07				2E-03					
Exposure Medium Total			1E-07				2E-03					
Medium Total				1E-07				2E-03				
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	2E-06	--	5E-07	2E-06	Developmental effects	3E-02	--	9E-03	4E-02
			ALUMINUM	--	--	--	--	Neurotoxicology	5E-03	--	--	5E-03
			ARSENIC	3E-06	--	1E-06	4E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	6E-03	3E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-02	--	7E-03	2E-02
			CHROMIUM	--	--	--	--	--	4E-02	--	--	4E-02
			COPPER	--	--	--	--	Gastrointestinal effects	3E-03	--	--	3E-03
			IRON	--	--	--	--	Gastrointestinal effects	2E-02	--	--	2E-02
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	6E-03	--	--	6E-03

TABLE 9.9a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	7E-07	--	9E-07	2E-06	--	5E-02	--	6E-02	1E-01
			ACENAPHTHYLENE	--	--	--	--	--	7E-05	--	9E-05	2E-04
			BENZ(A)ANTHRACENE	2E-06	--	3E-06	5E-06	--	--	--	--	--
			BENZO(A)PYRENE	2E-05	--	2E-05	4E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-06	--	3E-06	6E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	8E-05	--	1E-04	2E-04
			BENZO(K)FLUORANTHENE	8E-08	--	1E-07	2E-07	--	--	--	--	--
			CHRYSENE	2E-08	--	3E-08	6E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-06	3E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	2E-03	3E-03
			INDENO(1,2,3-CD)PYRENE	5E-07	--	6E-07	1E-06	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	5E-04	--	6E-04	1E-03
	BENZENE	2E-11	--	--	2E-11	Reduced lymphocyte count	2E-07	--	--	2E-07		
	Chemical Total	3E-05	--	3E-05	6E-05		2E-01	--	9E-02	3E-01		
	Exposure Point Total				6E-05					3E-01		
	Exposure Medium Total				6E-05					3E-01		
Medium Total					6E-05					3E-01		
Receptor Total					6E-05				Receptor HI Total	3E-01		

Total Risk Across All Media = 6E-05

Total Hazard Across All Media = 3E-01

TABLE 9.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	1E-04	--	--	1E-04	Developmental effects	1E+01	--	--	1E+01		
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	1E+00	--	--	1E+00		
			ARSENIC	6E-06	--	--	6E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	--	1E-01		
			CHROMIUM	--	--	--	--	--	1E-01	--	--	1E-01		
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	2E-01	--	--	2E-01		
			MANGANESE	--	--	--	--	CNS (N)	1E-02	--	--	1E-02		
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	6E+00	--	--	6E+00		
			SELENIUM	--	--	--	--	Clinical selenosis	2E-01	--	--	2E-01		
			VANADIUM	--	--	--	--	Decreased hair cystine	4E-02	--	--	4E-02		
			ZINC	--	--	--	--	Decreased ESOD (B)	8E-02	--	--	8E-02		
			HIGHLY CHLORINATED PCBs	6E-05	--	--	6E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+01	--	--	2E+01		
			LESS CHLORINATED PCBs	5E-05	--	--	5E-05	Reduced birth weights (W)	4E+00	--	--	4E+00		
			4,4-DDD	1E-07	--	--	1E-07	--	--	--	--	--		
			4,4'-DDT	2E-07	--	--	2E-07	Liver lesions (H)	1E-02	--	--	1E-02		
			ALDRIN	2E-06	--	--	2E-06	Liver toxicity (H)	5E-02	--	--	5E-02		
			DELTA-BHC	--	--	--	--	--	--	--	--	--		
			DIELDRIN	3E-06	--	--	3E-06	Hepatic (H)	4E-02	--	--	4E-02		
			HEPTACHLOR EPOXIDE	2E-06	--	--	2E-06	Increased liver-to-body weight ratio in males and females (H)	2E-01	--	--	2E-01		
			BIS(2-ETHYLHEXYL)PHTHALATE	2E-06	--	--	2E-06	Increased relative liver weight (H)	6E-02	--	--	6E-02		
			HEXACHLOROBENZENE	1E-06	--	--	1E-06	Hepatic (H)	9E-03	--	--	9E-03		
			Chemical Total	3E-04			3E-04		4E+01			4E+01		
			Exposure Point Total											4E+01
			Exposure Medium Total											4E+01
			Medium Total											4E+01
	Sediment	Surface Sediment	Exposure Unit 6	2,3,7,8-TCDD Equivalent	2E-06	--	3E-06	5E-06	Developmental effects	2E-01	--	2E-01	4E-01	
				ARSENIC	2E-06	--	3E-06	5E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-02	--	7E-02	1E-01	
				CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-01	--	1E-02	3E-01	
CHROMIUM				--	--	--	--	None Reported (O)	1E+00	--	--	1E+00		
IRON				--	--	--	--	Gastrointestinal effects	3E-02	--	--	3E-02		
LEAD				--	--	--	--	--	--	--	--	--		
MANGANESE				--	--	--	--	CNS (N)	7E-02	--	--	7E-02		
MERCURY				--	--	--	--	Autoimmune effects	6E-01	--	--	6E-01		

TABLE 9.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 6	THALLIUM	--	--	--	--	Hematological effects	1E-02	--	--	1E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-01	--	--	1E-01
			HIGHLY CHLORINATED PCBs	2E-07	--	1E-06	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-02	--	3E-01	4E-01
			DIELDRIN	4E-08	--	--	4E-08	Hepatic (H)	6E-04	--	--	6E-04
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	3E-04	--	--	3E-04
			HEPTACHLOR EPOXIDE	1E-08	--	--	1E-08	Increased liver-to-body weight ratio in males and females (H)	1E-03	--	--	1E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E-02	--	8E-02	9E-02
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-04	--	2E-03	2E-03
			BENZ(A)ANTHRACENE	1E-04	--	5E-04	6E-04	--	--	--	--	--
			BENZO(A)PYRENE	7E-04	--	4E-03	4E-03	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-04	--	8E-04	1E-03	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-03	--	2E-02	2E-02
			BENZO(K)FLUORANTHENE	4E-06	--	2E-05	2E-05	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	1E-07	--	5E-07	6E-07	Increased relative liver weight (H)	5E-03	--	2E-02	2E-02
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	1E-06	--	7E-06	9E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	8E-05	--	4E-04	5E-04	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E-02	--	2E-01	2E-01
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	5E-03	--	3E-02	3E-02
			HEXACHLOROBENZENE	3E-08	--	1E-07	1E-07	Hepatic (H)	2E-04	--	1E-03	1E-03
			INDENO(1,2,3-CD)PYRENE	4E-05	--	2E-04	2E-04	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	5E-03	--	3E-02	3E-02
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-02	--	7E-02	8E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-02	--	1E-01	1E-01
			1,2,4-TRICHLOROBENZENE	4E-10	--	--	4E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1E-04	--	--	1E-04
			1,4-DICHLOROBENZENE	4E-08	--	--	4E-08	Liver	1E-03	--	--	1E-03
			BENZENE	6E-08	--	--	6E-08	Reduced lymphocyte count	3E-03	--	--	3E-03
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	4E-03	--	--	4E-03
			METHYLENE CHLORIDE	7E-10	--	--	7E-10	Liver toxicity (H)	2E-05	--	--	2E-05

TABLE 9.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 6	TOLUENE	--	--	--	--	Increased kidney weight (R)	4E-04	--	--	4E-04
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	5E-04	--	--	5E-04
			Chemical Total	1E-03	--	6E-03	7E-03		3E+00	--	1E+00	4E+00
		Exposure Point Total				7E-03					4E+00	
		Exposure Medium Total				7E-03					4E+00	
Medium Total							7E-03					4E+00
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	--	--	5E-05	--	5E-05
			ARSENIC	--	1E-10	--	1E-10	Neurobehavioral changes (N, O)	--	5E-06	--	5E-06
			BARIUM	--	--	--	--	PNS (N); CNS (N)	--	2E-05	--	2E-05
			CADMIUM	--	2E-10	--	2E-10	--	--	--	--	--
			CHROMIUM	--	4E-09	--	4E-09	--	--	4E-05	--	4E-05
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	--	--	2E-04	--	2E-04
			MERCURY	--	--	--	--	--	--	1E-06	--	1E-06
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-12	--	2E-12	--	--	--	--	--
			LESS CHLORINATED PCBs	--	1E-12	--	1E-12	--	--	--	--	--
			DIELDRIN	--	1E-12	--	1E-12	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	Nasal/respiratory (P)	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	1E-12	--	1E-12	Liver	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	Decreased lymphocyte count	--	--	--	--
			NAPHTHALENE	--	2E-12	--	2E-12	--	--	3E-07	--	3E-07
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	8E-04	--	8E-04
			1,4-DICHLOROBENZENE	--	4E-07	--	4E-07	Psychomotor and cognitive impairments	--	6E-04	--	6E-04

TABLE 9.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 6	BENZENE	--	3E-08	--	3E-08	--	--	1E-03	--	1E-03
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	--	5E-07	--	5E-07	--	--	3E-03	--	3E-03
		Exposure Point Total					5E-07					3E-03
	Exposure Medium Total						5E-07					3E-03
Medium Total							5E-07					3E-03
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	1E-05	--	1E-05	2E-05	Developmental effects	8E-01	--	1E+00	2E+00
			ALUMINUM	--	--	--	--	Neurotoxicity	1E-02	--	--	1E-02
			ARSENIC	2E-06	--	2E-06	4E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	4E-02	--	5E-02	1E-01
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	3E-03	--	--	3E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-02	--	9E-02	1E-01
			CHROMIUM	--	--	--	--	--	6E-02	--	--	6E-02
			COPPER	--	--	--	--	Gastrointestinal effects	9E-03	--	--	9E-03
			IRON	--	--	--	--	Gastrointestinal effects	3E-02	--	--	3E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	3E-03	--	--	3E-03
			MERCURY	--	--	--	--	Autoimmune effects	6E-02	--	--	6E-02
			SILVER	--	--	--	--	Argyria (In)	5E-03	--	--	5E-03
			THALLIUM	--	--	--	--	Hematological effects	2E-02	--	--	2E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	4E-03	--	--	4E-03
			HIGHLY CHLORINATED PCBs	4E-07	--	2E-06	3E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	7E-01	8E-01
			LESS CHLORINATED PCBs	2E-07	--	1E-06	1E-06	Reduced birth weights (W)	2E-02	--	9E-02	1E-01
			DIELDRIN	2E-07	--	--	2E-07	Hepatic (H)	3E-03	--	--	3E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	5E-03	--	3E-02	3E-02
			ACENAPHTHYLENE	--	--	--	--	--	2E-04	--	1E-03	1E-03
			BENZ(A)ANTHRACENE	5E-06	--	2E-06	7E-06	--	--	--	--	--
			BENZO(A)PYRENE	6E-05	--	2E-05	8E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	4E-06	--	2E-06	6E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	3E-04	--	1E-03	2E-03
			BENZO(K)FLUORANTHENE	4E-07	--	1E-07	5E-07	--	--	--	--	--
			CHRYSENE	5E-08	--	2E-08	7E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-05	--	4E-06	1E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	6E-03	--	3E-02	3E-02
			HEXACHLOROBENZENE	2E-07	--	8E-07	1E-06	Hepatic (H)	2E-03	--	8E-03	1E-02
			INDENO(1,2,3-CD)PYRENE	3E-06	--	1E-06	4E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-03	--	1E-02	1E-02

TABLE 9.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 6	PHENANTHRENE	--	--	--	--	--	1E-03	--	5E-03	6E-03
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	2E-09	--	--	2E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	6E-04	--	--	6E-04
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	1E-04	--	--	1E-04
			1,4-DICHLOROBENZENE	2E-08	--	--	2E-08	Liver	7E-04	--	--	7E-04
			BENZENE	4E-09	--	--	4E-09	Reduced lymphocyte count	2E-04	--	--	2E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	1E-04	--	5E-05	1E-04		1E+00	--	2E+00	3E+00
		Exposure Point Total				1E-04				3E+00		
	Exposure Medium Total				1E-04				3E+00			
Medium Total						1E-04				3E+00		
Surface Water	Surface Water	Exposure Unit 6	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	3E-03	3E-03
			ARSENIC	--	--	2E-08	2E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	5E-04	5E-04
			CHROMIUM	--	--	--	--	--	--	--	1E-02	1E-02
			IRON	--	--	--	--	Gastrointestinal effects	--	--	6E-04	6E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MERCURY	--	--	--	--	Autoimmune effects	--	--	5E-04	5E-04
			THALLIUM	--	--	--	--	Hematological effects	--	--	4E-03	4E-03
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	7E-03	7E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	3E-03	3E-03
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	1E-04	1E-04	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-03	1E-03	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-04	2E-04	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	9E-08	9E-08	Increased relative liver weight (H)	--	--	4E-03	4E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	9E-07	9E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	4E-01	4E-01
			PHENANTHRENE	--	--	--	--	--	--	--	2E-02	2E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	2E-08	2E-08	Liver	--	--	5E-04	5E-04
			BENZENE	--	--	5E-07	5E-07	Reduced lymphocyte count	--	--	3E-02	3E-02

TABLE 9.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 6	DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	1E-02	1E-02
			XYLENES, TOTAL	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	1E-03	1E-03	--	--	--	5E-01	5E-01
		Exposure Point Total					1E-03					5E-01
	Exposure Medium Total						1E-03					5E-01
Medium Total							1E-03					5E-01
Receptor Total							9E-03					Receptor HI Total 5E+01

Total Risk Across All Media = 9E-03

Total Hazard Across All Media = 5E+01

Total Liver HI Across All Media =	4E-01
Total Kidney HI Across All Media =	7E-01
Total Nervous System Effects HI Across All Media =	7E+00
Total Lymphocyte Effects HI Across All Media =	3E-02
Total Nasal/Respiratory Effects HI Across All Media =	1E-01
Total Ocular Effects HI Across All Media =	2E+01
Total Other Effects HI Across All Media =	2E+01

TABLE 9.10a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	6E-07	--	8E-07	1E-06	--	5E-02	--	6E-02	1E-01
			ALUMINIUM	--	--	--	--	Neurotoxicity	8E-03	--	--	8E-03
			ARSENIC	1E-06	--	2E-06	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	4E-02	7E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-02	--	4E-02	7E-02
			CHROMIUM	--	--	--	--	--	6E-02	--	--	6E-02
			COPPER	--	--	--	--	Gastrointestinal effects	5E-03	--	--	5E-03
			IRON	--	--	--	--	Gastrointestinal effects	3E-02	--	--	3E-02
			MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--	4E-03
			MERCURY	--	--	--	--	Autoimmune effects	1E-02	--	--	1E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	2E-07	--	1E-06	2E-06	--	7E-02	--	4E-01	5E-01
			ACENAPHTHYLENE	--	--	--	--	--	1E-04	--	6E-04	7E-04
			BENZ(A)ANTHRACENE	6E-06	--	3E-05	4E-05	--	--	--	--	--
			BENZO(A)PYRENE	4E-05	--	2E-04	3E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-06	--	3E-05	4E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-04	--	7E-04	8E-04
			BENZO(K)FLUORANTHENE	2E-07	--	1E-06	1E-06	--	--	--	--	--
			CHRYSENE	6E-08	--	3E-07	4E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-06	--	2E-05	2E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-03	--	1E-02	1E-02
			INDENO(1,2,3-CD)PYRENE	1E-06	--	6E-06	7E-06	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	8E-04	--	4E-03	5E-03
			BENZENE	7E-12	--	--	7E-12	Reduced lymphocyte count	4E-07	--	--	4E-07
			Chemical Total	6E-05		3E-04	4E-04		3E-01		6E-01	9E-01
		Exposure Point Total				4E-04					9E-01	
	Exposure Medium Total					4E-04					9E-01	
Medium Total						4E-04					9E-01	
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINIUM	--	--	--	--	Psychomotor and cognitive impairments	--	6E-05	--	6E-05
			ARSENIC	--	1E-10	--	1E-10	Development, cardiovascular, nervous svstem	--	7E-06	--	7E-06
			CADMIUM	--	2E-10	--	2E-10	--	--	--	--	--
			CHROMIUM	--	7E-09	--	7E-09	--	--	7E-05	--	7E-05
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	4E-04	--	4E-04
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	3E-07	--	3E-07
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	3E-12	--	3E-12	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.10a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	
			PHENANTHRENE	--	--	--	--	--	--	--	--	
			BENZENE	--	4E-11	--	4E-11	Decreased lymphocyte count	--	2E-06	--	2E-06
	Chemical Total	--	7E-09	--	7E-09		--	5E-04	--	5E-04		
	Exposure Point Total			7E-09						5E-04		
	Exposure Medium Total			7E-09						5E-04		
Medium Total			7E-09						5E-04			
Receptor Total						4E-04	Receptor HI Total			9E-01		

Total Risk Across All Media = 4E-04

Total Hazard Across All Media = 9E-01

TABLE 9.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	5E-04	--	--	5E-04	Developmental effects	7E+00	--	--	7E+00			
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	9E-01	--	--	9E-01			
			ARSENIC	2E-05	--	--	2E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	--	1E-01			
			CHROMIUM	--	--	--	--	--	7E-02	--	--	7E-02			
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	1E-01	--	--	1E-01			
			MANGANESE	--	--	--	--	CNS (N)	8E-03	--	--	8E-03			
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	4E+00	--	--	4E+00			
			SELENIUM	--	--	--	--	Clinical selenosis	1E-01	--	--	1E-01			
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-02	--	--	3E-02			
			ZINC	--	--	--	--	Decreased ESOD (B)	5E-02	--	--	5E-02			
			HIGHLY CHLORINATED PCBs	2E-04	--	--	2E-04	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E+01	--	--	1E+01			
			LESS CHLORINATED PCBs	1E-04	--	--	1E-04	Reduced birth weights (W)	2E+00	--	--	2E+00			
			4,4-DDD	5E-07	--	--	5E-07	--	--	--	--	--			
			4,4'-DDT	5E-07	--	--	5E-07	Liver lesions (H)	7E-03	--	--	7E-03			
			ALDRIN	7E-06	--	--	7E-06	Liver toxicity (H)	3E-02	--	--	3E-02			
			DELTA-BHC	--	--	--	--	--	--	--	--	--			
			DIELDRIN	9E-06	--	--	9E-06	Hepatic (H)	3E-02	--	--	3E-02			
			HEPTACHLOR EPOXIDE	6E-06	--	--	6E-06	Increased liver-to-body weight ratio in males and females (H)	1E-01	--	--	1E-01			
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-06	--	--	5E-06	Increased relative liver weight (H)	4E-02	--	--	4E-02			
			HEXACHLOROBENZENE	3E-06	--	--	3E-06	Hepatic (H)	6E-03	--	--	6E-03			
			Chemical Total	8E-04	--	--	8E-04		3E+01	--	--	3E+01			
			Exposure Point Total				8E-04				3E+01				
			Exposure Medium Total				8E-04				3E+01				
			Medium Total				8E-04				3E+01				
			Sediment	Surface Sediment	Exposure Unit 6	2,3,7,8-TCDD Equivalent	6E-07	--	6E-07	1E-06	Developmental effects	9E-03	--	9E-03	2E-02
						ARSENIC	5E-07	--	6E-07	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-03	--	3E-03	6E-03
	CADMIUM	--				--	--	--	Renal (R); Significant Proteinuria	2E-02	--	5E-04	2E-02		
CHROMIUM	--	--				--	--	None Reported (O)	7E-02	--	--	7E-02			
IRON	--	--				--	--	Gastrointestinal effects	1E-03	--	--	1E-03			
LEAD	--	--				--	--	--	--	--	--	--			

TABLE 9.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 6	MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--	4E-03
			MERCURY	--	--	--	--	Autoimmune effects	3E-02	--	--	3E-02
			THALLIUM	--	--	--	--	Hematological effects	8E-04	--	--	8E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	5E-03	--	--	5E-03
			HIGHLY CHLORINATED PCBs	5E-08	--	3E-07	3E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-03	--	2E-02	2E-02
			DIELDRIN	1E-08	--	--	1E-08	Hepatic (H)	3E-05	--	--	3E-05
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	1E-05	--	--	1E-05
			HEPTACHLOR EPOXIDE	3E-09	--	--	3E-09	Increased liver-to-body weight ratio in males and females (H)	6E-05	--	--	6E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	7E-04	--	3E-03	4E-03
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-05	--	7E-05	8E-05
			BENZ(A)ANTHRACENE	4E-06	--	2E-05	2E-05	--	--	--	--	--
			BENZO(A)PYRENE	3E-05	--	1E-04	1E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-06	--	3E-05	3E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-04	--	8E-04	1E-03
			BENZO(K)FLUORANTHENE	2E-07	--	7E-07	8E-07	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHthalate	3E-08	--	1E-07	1E-07	Increased relative liver weight (H)	3E-04	--	9E-04	1E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	6E-08	--	2E-07	3E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-06	--	1E-05	2E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	8E-03	1E-02
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-04	--	1E-03	1E-03
			HEXACHLOROBENZENE	7E-09	--	2E-08	3E-08	Hepatic (H)	1E-05	--	4E-05	6E-05
			INDENO(1,2,3-CD)PYRENE	1E-06	--	6E-06	8E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	3E-04	--	1E-03	2E-03
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	7E-04	--	3E-03	4E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-03	--	5E-03	7E-03

TABLE 9.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 6	1,2,4-TRICHLOROBENZENE	1E-10	--	--	1E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	7E-06	--	--	7E-06
			1,4-DICHLOROBENZENE	1E-08	--	--	1E-08	Liver	6E-05	--	--	6E-05
			BENZENE	2E-08	--	--	2E-08	Reduced lymphocyte count	2E-04	--	--	2E-04
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-04	--	--	2E-04
			METHYLENE CHLORIDE	2E-10	--	--	2E-10	Liver toxicity (H)	1E-06	--	--	1E-06
			TOLUENE	--	--	--	--	Increased kidney weight (R)	2E-05	--	--	2E-05
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	3E-05	--	--	3E-05
			Chemical Total	4E-05	--	2E-04	2E-04		2E-01	--	5E-02	2E-01
			Exposure Point Total			2E-04						2E-01
			Exposure Medium Total			2E-04						2E-01
Medium Total			2E-04						2E-01			
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	Development, cardiovascular, nervous system	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E-05	--	1E-05
			ARSENIC	--	1E-10	--	1E-10	Development, cardiovascular, nervous system	--	2E-06	--	2E-06
			BARIUM	--	--	--	--	Renal toxicity	--	7E-06	--	7E-06
			CADMIUM	--	3E-10	--	3E-10	--	--	--	--	--
			CHROMIUM	--	6E-09	--	6E-09	--	1E-05	--	1E-05	
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	6E-05	--	6E-05
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	4E-07	--	4E-07
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	3E-12	--	3E-12	--	--	--	--	--
			LESS CHLORINATED PCBs	--	2E-12	--	2E-12	--	--	--	--	--
			DIELDRIN	--	2E-12	--	2E-12	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--

TABLE 9.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Surface Soil	Outdoor Air	Exposure Unit 6	CHRYSENE	--	--	--	--	--	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--		
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--		
			HEXACHLOROBENZENE	--	2E-12	--	2E-12	--	--	--	--	--		
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--		
			NAPHTHALENE	--	3E-12	--	3E-12	Nasal/respiratory (P)	--	7E-08	--	7E-08		
			PHENANTHRENE	--	--	--	--	--	--	--	--	--		
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	2E-04	--	2E-04		
			1,4-DICHLOROBENZENE	--	6E-07	--	6E-07	Liver	--	2E-04	--	2E-04		
			BENZENE	--	4E-08	--	4E-08	Decreased lymphocyte count	--	4E-04	--	4E-04		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
	BENZ(A)ANTHRACENE	--	3E-12	--	3E-12	--	--	--	--	--				
		Chemical Total	--	7E-07	--	7E-07		--	8E-04	--	8E-04			
		Exposure Point Total					7E-07					8E-04		
		Exposure Medium Total					7E-07					8E-04		
Medium Total							7E-07					8E-04		
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	3E-06	--	3E-06	6E-06	Developmental effects	4E-02	--	4E-02	9E-02		
			ALUMINUM	--	--	--	--	Neurotoxicity	6E-04	--	--	6E-04		
			ARSENIC	4E-07	--	5E-07	9E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-03	--	2E-03	5E-03		
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	2E-04	--	--	2E-04		
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-03	--	4E-03	7E-03		
			CHROMIUM	--	--	--	--	--	3E-03	--	--	3E-03		
			COPPER	--	--	--	--	Gastrointestinal effects	5E-04	--	--	5E-04		
			IRON	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	CNS (N)	2E-04	--	--	2E-04		
			MERCURY	--	--	--	--	Autoimmune effects	3E-03	--	--	3E-03		
			SILVER	--	--	--	--	Argyria (In)	2E-04	--	--	2E-04		
			THALLIUM	--	--	--	--	Hematological effects	8E-04	--	--	8E-04		
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-04	--	--	2E-04		
					HIGHLY CHLORINATED PCBs	1E-07	--	5E-07	6E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-03	--	3E-02	4E-02

TABLE 9.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Soil	Surface Soil	Exposure Unit 6	LESS CHLORINATED PCBs	5E-08	--	2E-07	3E-07	Reduced birth weights (W)	8E-04	--	4E-03	5E-03			
			DIELDRIN	6E-08	--	--	6E-08	Hepatic (H)	2E-04	--	--	2E-04			
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E-04	--	1E-03	1E-03			
			ACENAPHTHYLENE	--	--	--	--	--	1E-05	--	5E-05	6E-05			
			BENZ(A)ANTHRACENE	2E-07	--	9E-07	1E-06	--	--	--	--	--			
			BENZO(A)PYRENE	2E-06	--	1E-05	1E-05	--	--	--	--	--			
			BENZO(B)FLUORANTHENE	2E-07	--	8E-07	9E-07	--	--	--	--	--			
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-05	--	6E-05	8E-05			
			BENZO(K)FLUORANTHENE	1E-08	--	6E-08	8E-08	--	--	--	--	--			
			CHRYSENE	2E-09	--	9E-09	1E-08	--	--	--	--	--			
			DIBENZ(A,H)ANTHRACENE	4E-07	--	2E-06	2E-06	--	--	--	--	--			
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-04	--	1E-03	1E-03			
			HEXACHLOROBENZENE	5E-08	--	2E-07	2E-07	Hepatic (H)	1E-04	--	3E-04	4E-04			
			INDENO(1,2,3-CD)PYRENE	1E-07	--	5E-07	6E-07	--	--	--	--	--			
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E-04	--	4E-04	5E-04			
			PHENANTHRENE	--	--	--	--	--	5E-05	--	2E-04	3E-04			
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--			
			1,2,4-TRICHLOROBENZENE	5E-10	--	--	5E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-05	--	--	3E-05			
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	8E-06	--	--	8E-06			
			1,4-DICHLOROBENZENE	6E-09	--	--	6E-09	Liver	4E-05	--	--	4E-05			
			BENZENE	1E-09	--	--	1E-09	Reduced lymphocyte count	1E-05	--	--	1E-05			
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--			
			DODECANE	--	--	--	--	--	--	--	--	--			
			Chemical Total			7E-06	--	2E-05	3E-05				7E-02	--	9E-02
		Exposure Point Total							3E-05					2E-01	
	Exposure Medium Total							3E-05					2E-01		
Medium Total							3E-05					2E-01			
Surface Water	Surface Water	Exposure Unit 6	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	1E-03	1E-03			
			ARSENIC	--	--	4E-08	4E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	2E-04	2E-04			
			CHROMIUM	--	--	--	--	--	--	--	5E-03	5E-03			
			IRON	--	--	--	--	Gastrointestinal effects	--	--	3E-04	3E-04			
			LEAD	--	--	--	--	--	--	--	--	--			
			MERCURY	--	--	--	--	Autoimmune effects	--	--	2E-04	2E-04			
			THALLIUM	--	--	--	--	Hematological effects	--	--	2E-03	2E-03			
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	3E-03	3E-03			

TABLE 9.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 6	2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	1E-03	1E-03
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	4E-05	4E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	4E-04	4E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	6E-05	6E-05	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	2E-07	2E-07	Increased relative liver weight (H)	--	--	2E-03	2E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	4E-07	4E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	2E-01	2E-01
			PHENANTHRENE	--	--	--	--	--	--	--	8E-03	8E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	4E-08	4E-08	Liver	--	--	2E-04	2E-04
			BENZENE	--	--	1E-06	1E-06	Reduced lymphocyte count	--	--	1E-02	1E-02
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	6E-03	6E-03
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total	--	--	5E-04	5E-04		--	--	2E-01	2E-01
		Exposure Point Total					5E-04					2E-01
	Exposure Medium Total						5E-04					2E-01
Medium Total							5E-04					2E-01
Receptor Total							2E-03					Receptor HI Total 3E+01

Total Risk Across All Media = 2E-03

Total Hazard Across All Media = 3E+01

Total Liver HI Across All Media =	2E-01
Total Kidney HI Across All Media =	4E-02
Total Nervous System Effects HI Across All Media =	4E+00
Total Lymphocyte Effects HI Across All Media =	1E-02
Total Nasal/Respiratory Effects HI Across All Media =	5E-03
Total Ocular Effects HI Across All Media =	1E+01
Total Other Effects HI Across All Media =	1E+01

TABLE 9.11a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-05	--	2E-05
			ARSENIC	--	2E-10	--	2E-10	Development, cardiovascular, nervous system	--	2E-06	--	2E-06
			CADMIUM	--	2E-10	--	2E-10	--	--	--	--	
			CHROMIUM	--	1E-08	--	1E-08	--	--	2E-05	--	2E-05
			COPPER	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-04	--	1E-04
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	1E-07	--	1E-07
			VANADIUM	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	4E-12	--	4E-12	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	
			PHENANTHRENE	--	--	--	--	--	--	--	--	
			BENZENE	--	6E-11	--	6E-11	Decreased lymphocyte count	--	6E-07	--	6E-07
			Chemical Total				1E-08		1E-08		1E-04	
	Exposure Point Total							1E-08				1E-04
	Exposure Medium Total							1E-08				1E-04
Medium Total								1E-08				1E-04
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	2E-07	--	2E-07	3E-07	--	3E-03	--	3E-03	5E-03
			ALUMINUM	--	--	--	--	Neurotoxicity	4E-04	--	--	4E-04
			ARSENIC	3E-07	--	3E-07	7E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-03	--	2E-03	3E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-03	--	2E-03	3E-03
			CHROMIUM	--	--	--	--	--	3E-03	--	--	3E-03
			COPPER	--	--	--	--	Gastrointestinal effects	2E-04	--	--	2E-04
			IRON	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			MANGANESE	--	--	--	--	CNS (N)	2E-04	--	--	2E-04
			MERCURY	--	--	--	--	Autoimmune effects	5E-04	--	--	5E-04

TABLE 9.11a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	VANADIUM	--	--	--	--	Decreased hair cystine	1E-04	--	--	1E-04
			HIGHLY CHLORINATED PCBs	7E-08	--	3E-07	4E-07	--	4E-03	--	2E-02	2E-02
			ACENAPHTHYLENE	--	--	--	--	--	6E-06	--	2E-05	3E-05
			BENZ(A)ANTHRACENE	2E-07	--	1E-06	1E-06	--	--	--	--	--
			BENZO(A)PYRENE	2E-06	--	8E-06	9E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-07	--	1E-06	1E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	6E-06	--	3E-05	3E-05
			BENZO(K)FLUORANTHENE	8E-09	--	4E-08	5E-08	--	--	--	--	--
			CHRYSENE	2E-09	--	1E-08	1E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-07	--	7E-07	8E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-04	--	5E-04	6E-04
			INDENO(1,2,3-CD)PYRENE	5E-08	--	2E-07	3E-07	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	4E-05	--	2E-04	2E-04
			BENZENE	2E-12	--	--	2E-12	Reduced lymphocyte count	2E-08	--	--	2E-08
			Chemical Total	3E-06		1E-05	1E-05		2E-02		3E-02	4E-02
		Exposure Point Total				1E-05				4E-02		
	Exposure Medium Total				1E-05				4E-02			
Medium Total					1E-05				4E-02			
Receptor Total						1E-05	Receptor HI Total			4E-02		

Total Risk Across All Media = 1E-05

Total Hazard Across All Media = 4E-02

TABLE 9.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	Development, cardiovascular, nervous system	--	--	--	--	
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	8E-04	--	8E-04	
			ARSENIC	--	2E-09	--	2E-09	Development, cardiovascular, nervous system	--	1E-04	--	1E-04	
			BARIUM	--	--	--	--	Renal toxicity	--	4E-04	--	4E-04	
			CADMIUM	--	3E-09	--	3E-09	--	--	--	--	--	
			CHROMIUM	--	7E-08	--	7E-08	--	--	7E-04	--	7E-04	
			COPPER	--	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--	--	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O) PNS (N); CNS (N)	--	3E-03	--	3E-03	
			MERCURY	--	--	--	--		2E-05	--	2E-05		
			SILVER	--	--	--	--	--	--	--	--	--	
			THALLIUM	--	--	--	--	--	--	--	--	--	
			VANADIUM	--	--	--	--	--	--	--	--	--	
			DODECANE	--	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	4E-11	--	4E-11	--	--	--	--	--	
			LESS CHLORINATED PCBs	--	2E-11	--	2E-11	--	--	--	--	--	
			DIELDRIN	--	3E-11	--	3E-11	--	--	--	--	--	
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--	
			HEXACHLORO BENZENE	--	2E-11	--	2E-11	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--	
			NAPHTHALENE	--	4E-11	--	4E-11	Nasal/respiratory (P)	--	4E-06	--	4E-06	
			PHENANTHRENE	--	--	--	--		--	--	--	--	--
		1,2,3-TRICHLORO BENZENE	--	--	--	--	--		--	--	--	--	
1,2,4-TRICHLORO BENZENE	--	--	--	--	--	--	--		--	--			
			1,2-DICHLORO BENZENE	--	--	--	--	1E-02	--	1E-02			
			1,4-DICHLORO BENZENE	--	8E-06	--	8E-06	Liver	--	1E-02			
			BENZENE	--	4E-07	--	4E-07	Decreased lymphocyte count	--	2E-02			
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--			
			Chemical Total	--	8E-06	--	8E-06		--	5E-02	--		
		Exposure Point Total									5E-02		
	Exposure Medium Total									5E-02			
Medium Total								8E-06					5E-02

TABLE 9.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	8E-05	--	1E-04	2E-04	Developmental effects	7E+00	--	8E+00	1E+01
			ALUMINUM	--	--	--	--	Neurotoxicity	9E-02	--	--	9E-02
			ARSENIC	1E-05	--	2E-05	3E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	4E-01	--	4E-01	8E-01
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	2E-02	--	--	2E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-01	--	8E-01	1E+00
			CHROMIUM	--	--	--	--	--	5E-01	--	--	5E-01
			COPPER	--	--	--	--	Gastrointestinal effects	8E-02	--	--	8E-02
			IRON	--	--	--	--	Gastrointestinal effects	2E-01	--	--	2E-01
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	3E-02	--	--	3E-02
			MERCURY	--	--	--	--	Autoimmune effects	5E-01	--	--	5E-01
			SILVER	--	--	--	--	Argyria (In)	4E-02	--	--	4E-02
			THALLIUM	--	--	--	--	Hematological effects	1E-01	--	--	1E-01
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-02	--	--	3E-02
			HIGHLY CHLORINATED PCBs	3E-06	--	2E-05	2E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E+00	--	6E+00	7E+00
			LESS CHLORINATED PCBs	2E-06	--	9E-06	1E-05	Reduced birth weights (W)	1E-01	--	8E-01	9E-01
			DIELDRIN	2E-06	--	--	2E-06	Hepatic (H)	3E-02	--	--	3E-02
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	4E-02	--	2E-01	3E-01
			ACENAPHTHYLENE	--	--	--	--	--	2E-03	--	9E-03	1E-02
			BENZ(A)ANTHRACENE	4E-05	--	1E-05	6E-05	--	--	--	--	--
			BENZO(A)PYRENE	5E-04	--	2E-04	7E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	4E-05	--	1E-05	5E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-03	--	1E-02	1E-02
			BENZO(K)FLUORANTHENE	3E-06	--	1E-06	4E-06	--	--	--	--	--
			CHRYSENE	4E-07	--	2E-07	6E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	9E-05	--	3E-05	1E-04	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E-02	--	2E-01	3E-01
			HEXACHLOROBENZENE	2E-06	--	7E-06	9E-06	Hepatic (H)	2E-02	--	6E-02	8E-02
			INDENO(1,2,3-CD)PYRENE	3E-05	--	9E-06	3E-05	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-02	--	8E-02	1E-01
			PHENANTHRENE	--	--	--	--	--	8E-03	--	4E-02	5E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	2E-08	--	--	2E-08	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	5E-03	--	--	5E-03
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	1E-03	--	--	1E-03
			1,4-DICHLOROBENZENE	2E-07	--	--	2E-07	Liver	6E-03	--	--	6E-03

TABLE 9.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Soil	Surface Soil	Exposure Unit 6	BENZENE	3E-08	--	--	3E-08	Reduced lymphocyte count	2E-03	--	--	2E-03	
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--		
			DODECANE	--	--	--	--	--	--	--	--		
			Chemical Total	8E-04	--	4E-04	1E-03		1E+01	--	2E+01	3E+01	
		Exposure Point Total				1E-03					3E+01		
	Exposure Medium Total				1E-03					3E+01			
Medium Total							1E-03					3E+01	
Ground Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicity	2E+00	--	1E-02	2E+00	
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	4E-01	--	2E-02	4E-01	
			ARSENIC	8E-05	--	5E-07	8E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	2E+00	--	1E-02	2E+00	
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	5E-01	--	4E-02	5E-01	
			BERYLLIUM	--	--	--	--	Small intestinal lesions	3E-02	--	2E-02	5E-02	
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-01	--	3E-02	2E-01	
			CHROMIUM	--	--	--	--	--	1E+00	--	8E-01	2E+00	
			COBALT	--	--	--	--	--	--	--	--	--	
			COPPER	--	--	--	--	Gastrointestinal effects	1E-01	--	1E-03	1E-01	
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	1E-01	--	6E-04	1E-01	
			IRON	--	--	--	--	Gastrointestinal effects	4E+00	--	2E-02	4E+00	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	CNS (N)	8E-01	--	1E-01	1E+00	
			MERCURY	--	--	--	--	Autoimmune effects	5E-01	--	1E-02	5E-01	
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	2E-01	--	5E-03	2E-01	
			SELENIUM	--	--	--	--	Clinical selenosis	5E-02	--	3E-04	5E-02	
			SILVER	--	--	--	--	Argyria (In)	3E-02	--	3E-03	3E-02	
			THALLIUM	--	--	--	--	Hematological effects	6E+00	--	4E-02	6E+00	
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-01	--	8E-02	4E-01	
			ZINC	--	--	--	--	Decreased ESOD (B)	2E-02	--	8E-05	2E-02	
				HIGHLY CHLORINATED PCBs	8E-07	--	--	8E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-01	--	--	2E-01
				4,4'-DDD	1E-07	--	8E-07	9E-07	--	--	--	--	--
				4,4'-DDT	2E-06	--	2E-05	2E-05	Liver lesions (H)	1E-01	--	2E+00	2E+00
				ALDRIN	3E-06	--	3E-07	3E-06	Liver toxicity (H)	7E-02	--	6E-03	8E-02
				ALPHA-BHC	7E-06	--	--	7E-06	--	--	--	--	--

TABLE 9.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	ENDOSULFAN II	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	7E-04	--	--	7E-04
			ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	2E-04	--	--	2E-04
			HEPTACHLOR EPOXIDE	5E-07	--	--	5E-07	Increased liver-to-body weight ratio in males and females (H)	5E-02	--	--	5E-02
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	2E-02	--	--	2E-02
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	2E-01	--	7E-02	3E-01
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	1E+01	--	2E+00	1E+01
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E+01	--	--	1E+01
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	1E+00	--	1E-01	1E+00
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	6E+00	--	5E-01	6E+00
			4-CHLORO-3-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			4-METHYLPHENOL	--	--	--	--	--	1E+01	--	1E+00	1E+01
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	1E-01	--	--	1E-01
			ACENAPHTHYLENE	--	--	--	--	--	4E-01	--	--	4E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	2E-02	--	--	2E-02
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	1E-01	--	--	1E-01
			BENZ(A)ANTHRACENE	2E-04	--	2E-02	2E-02	--	--	--	--	--
			BENZO(A)PYRENE	8E-04	--	5E-01	5E-01	--	--	--	--	--
			BENZO(B)FLUORANTHENE	8E-05	--	6E-02	6E-02	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-02	--	--	1E-02
			BENZO(K)FLUORANTHENE	7E-06	--	--	7E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	8E-07	--	1E-06	2E-06	Increased relative liver weight (H)	3E-02	--	5E-02	8E-02
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	1E-06	--	5E-04	5E-04	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-04	--	1E-01	1E-01	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E+01	--	--	1E+01
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-01	--	1E+00	2E+00
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	3E-01	--	--	3E-01
			HEXACHLOROBUTADIENE	4E-07	--	1E-06	1E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	3E-05	--	2E-02	2E-02	--	--	--	--	--

TABLE 9.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+01	--	8E+00	2E+01
			NITROBENZENE	--	--	--	--	Hematologic (B), adrenal, renal (R) and hepatic (H) lesions	3E-01	--	--	3E-01
			PHENANTHRENE	--	--	--	--	--	9E-01	--	2E+00	3E+00
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	4E-01	--	2E-02	4E-01
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-01	--	--	2E-01
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	3E-07	--	3E-07	6E-07	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	9E-02	--	1E-01	2E-01
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	4E-01	--	2E-01	6E-01
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	1E-05	--	9E-06	2E-05	Liver	4E-01	--	3E-01	7E-01
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	6E-04	--	--	6E-04
			ACETONE	--	--	--	--	Nephropathy	6E-03	--	--	6E-03
			BENZENE	2E-03	--	3E-04	2E-03	Reduced lymphocyte count	9E+01	--	1E+01	1E+02
			BROMODICHLOROMETHANE	1E-06	--	8E-08	1E-06	Renal cytomegaly (R)	1E-02	--	8E-04	1E-02
			CARBON DISULFIDE	--	--	--	--	Fetal toxicity/malformations	8E-03	--	1E-03	9E-03
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	6E-01	--	2E-01	8E-01
			CHLOROETHANE	--	--	--	--	--	--	--	--	--
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	9E-02	--	5E-02	1E-01
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	3E-03	--	--	3E-03
			METHYLENE CHLORIDE	3E-08	--	1E-09	3E-08	Liver toxicity (H)	8E-04	--	3E-05	8E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	3E-01	--	1E-01	4E-01
			TETRACHLOROETHENE	9E-07	--	5E-07	1E-06	Hepatotoxicity in mice (H), weight gain in rats	2E-03	--	1E-03	3E-03
			TOLUENE	--	--	--	--	Increased kidney weight (R)	1E+00	--	3E-01	1E+00
			VINYL CHLORIDE	5E-06	--	2E-07	5E-06	Liver cell polymorphism (H)	2E-02	--	1E-03	2E-02
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	3E-01	--	--	3E-01
			Chemical Total	3E-03	--	7E-01	7E-01		2E+02	--	3E+01	2E+02
		Exposure Point Total					7E-01					2E+02
	Exposure Medium Total						7E-01					2E+02
	Shower Vapor	Exposure Unit 8	1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	Hematological and Pulmonary	--	1E+02	--	1E+02
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	8E+00	--	8E+00
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	9E-04	--	9E-04	Liver	--	1E+00	--	1E+00

TABLE 9.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shower Vapor	Exposure Unit 8	2-HEXANONE	--	--	--	--	Peripheral neuropathy	--	2E-02	--	2E-02
			ACETONE	--	--	--	--	Neurological effects	--	5E-03	--	5E-03
			BENZENE	--	8E-03	--	8E-03	Decreased lymphocyte count	--	4E+02	--	4E+02
			BROMODICHLOROMETHANE	--	2E-05	--	2E-05	--	--	--	--	--
			CARBON DISULFIDE	--	--	--	--	Peripheral nervous system dysfunction	--	4E-02	--	4E-02
			CHLOROBENZENE	--	--	--	--	--	--	--	--	--
			CHLOROETHANE	--	--	--	--	Delayed fetal ossification	--	1E-03	--	1E-03
			CHLOROFORM	--	5E-05	--	5E-05	Hepatic effects	--	2E-01	--	2E-01
			ETHYLBENZENE	--	--	--	--	Developmental toxicity	--	3E-01	--	3E-01
			ISOPROPYLBENZENE	--	--	--	--	Increased kidney and adrenal weights	--	2E-02	--	2E-02
			METHYLENE CHLORIDE	--	6E-08	--	6E-08	Hepatic effects	--	1E-03	--	1E-03
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Central nervous system effects	--	2E-01	--	2E-01
			TETRACHLOROETHENE	--	3E-10	--	3E-10	Neurological effects	--	2E-03	--	2E-03
			TOLUENE	--	--	--	--	Neurological effects	--	5E-01	--	5E-01
			VINYL CHLORIDE	--	2E-06	--	2E-06	Liver cell polymorphism	--	2E-02	--	2E-02
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	1E+01	--	1E+01
			Chemical Total	--	9E-03	--	9E-03		--	5E+02	--	5E+02
		Exposure Point Total					9E-03					5E+02
	Exposure Medium Total						9E-03					5E+02
Medium Total							7E-01					7E+02
Receptor Total							7E-01					8E+02
								Receptor HI Total				

Total Risk Across All Media = 7E-01

Total Hazard Across All Media = 8E+02

Total Liver HI Across All Media =	7E+00
Total Kidney HI Across All Media =	4E+00
Total Nervous System Effects HI Across All Media =	3E+01
Total Lymphocyte Effects HI Across All Media =	5E+02
Total Nasal/Respiratory Effects HI Across All Media =	1E+02
Total Ocular Effects HI Across All Media =	7E+00
Total Other Effects HI Across All Media =	1E+02

TABLE 9.12a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	Development, cardiovascular, nervous system	--	--	--	--	
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E-03	--	1E-03	
			ARSENIC	--	2E-09	--	2E-09	Development, cardiovascular, nervous system	--	1E-04	--	1E-04	
			CADMIUM	--	3E-09	--	3E-09	--	--	--	--		
			CHROMIUM	--	1E-07	--	1E-07	--	--	1E-03	--	1E-03	
			COPPER	--	--	--	--	--	--	--	--		
			IRON	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O) PNS (N); CNS (N)	--	7E-03	--	7E-03	
			MERCURY	--	--	--	--		6E-06	--	6E-06		
			VANADIUM	--	--	--	--		--	--	--		
			HIGHLY CHLORINATED PCBs	--	4E-11	--	4E-11		--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--		
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--		
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--		
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--		
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--		
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--		
			CHRYSENE	--	--	--	--	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--		
			DIBENZOFURAN	--	--	--	--	--	--	--	--		
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--		
			PHENANTHRENE	--	--	--	--	--	--	--	--		
			BENZENE	--	8E-10	--	8E-10	Decreased lymphocyte count	--	4E-05	--	4E-05	
			Chemical Total	--	1E-07	--	1E-07		--	9E-03	--	9E-03	
				Exposure Point Total					1E-07				
			Exposure Medium Total					1E-07					9E-03
Medium Total								1E-07					9E-03
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	5E-06	--	6E-06	1E-05	--	4E-01	--	5E-01	9E-01	
			ALUMINUM	--	--	--	--	Neurotoxicity	7E-02	--	--	7E-02	
			ARSENIC	1E-05	--	1E-05	2E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-01	--	3E-01	6E-01	
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-01	--	4E-01	6E-01	
			CHROMIUM	--	--	--	--	--	5E-01	--	--	5E-01	
			COPPER	--	--	--	--	Gastrointestinal effects	4E-02	--	--	4E-02	
			IRON	--	--	--	--	Gastrointestinal effects	2E-01	--	--	2E-01	
			MANGANESE	--	--	--	--	CNS (N)	3E-02	--	--	3E-02	
			MERCURY	--	--	--	--	Autoimmune effects	8E-02	--	--	8E-02	

TABLE 9.12a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-02	--	--	2E-02
			HIGHLY CHLORINATED PCBs	2E-06	--	1E-05	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-01	--	4E+00	4E+00
			ACENAPHTHYLENE	--	--	--	--	--	9E-04	--	5E-03	6E-03
			BENZ(A)ANTHRACENE	5E-05	--	2E-05	7E-05	--	--	--	--	--
			BENZO(A)PYRENE	4E-04	--	1E-04	5E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	5E-05	--	2E-05	7E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-03	--	5E-03	6E-03
			BENZO(K)FLUORANTHENE	2E-06	--	6E-07	2E-06	--	--	--	--	--
			CHRYSENE	5E-07	--	2E-07	7E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-05	--	1E-05	4E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-02	--	9E-02	1E-01
			INDENO(1,2,3-CD)PYRENE	1E-05	--	3E-06	1E-05	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	6E-03	--	3E-02	4E-02
			BENZENE	6E-11	--	--	6E-11	Reduced lymphocyte count	3E-06	--	--	3E-06
	Chemical Total	5E-04	--	2E-04	7E-04		3E+00	--	5E+00	7E+00		
	Exposure Point Total			7E-04			7E+00					
	Exposure Medium Total			7E-04			7E+00					
Medium Total				7E-04			7E+00					
Receptor Total				7E-04			Receptor HI Total 7E+00					

Total Risk Across All Media = 7E-04

Total Hazard Across All Media = 7E+00

Total Kidney HI Across All Media =	6E-01
Total Nervous System Effects HI Across All Media =	7E-01
Total Lymphocyte Effects HI Across All Media =	4E-05
Total Ocular Effects HI Across All Media =	4E+00
Total Other Effects HI Across All Media =	2E+00

TABLE 9.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	Development, cardiovascular, nervous system	--	--	--	--		
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-04	--	2E-04		
			ARSENIC	--	2E-09	--	2E-09	Development, cardiovascular, nervous system	--	3E-05	--	3E-05		
			BARIUM	--	--	--	--	Renal toxicity	--	1E-04	--	1E-04		
			CADMIUM	--	4E-09	--	4E-09	--	--	--	--	--		
			CHROMIUM	--	9E-08	--	9E-08	--	--	2E-04	--	2E-04		
			COPPER	--	--	--	--	--	--	--	--	--		
			IRON	--	--	--	--	--	--	--	--	--		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	9E-04	--	9E-04		
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	6E-06	--	6E-06		
			SILVER	--	--	--	--	--	--	--	--	--		
			THALLIUM	--	--	--	--	--	--	--	--	--		
			VANADIUM	--	--	--	--	--	--	--	--	--		
			HIGHLY CHLORINATED PCBs	--	6E-11	--	6E-11	--	--	--	--	--		
			LESS CHLORINATED PCBs	--	3E-11	--	3E-11	--	--	--	--	--		
			DIELDRIN	--	3E-11	--	3E-11	--	--	--	--	--		
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--		
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--		
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--		
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--		
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--		
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--		
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--		
			CHRYSENE	--	--	--	--	--	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--		
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--		
			HEXACHLORO BENZENE	--	3E-11	--	3E-11	--	--	--	--	--		
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--		
			NAPHTHALENE	--	5E-11	--	5E-11	Nasal/respiratory (P)	--	1E-06	--	1E-06		
			PHENANTHRENE	--	--	--	--	--	--	--	--	--		
			1,2,3-TRICHLORO BENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLORO BENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLORO BENZENE	--	--	--	--	--	--	4E-03	--	4E-03		
			1,4-DICHLORO BENZENE	--	1E-05	--	1E-05	Liver	--	3E-03	--	3E-03		
			BENZENE	--	6E-07	--	6E-07	Decreased lymphocyte count	--	6E-03	--	6E-03		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
			DODECANE	--	--	--	--	--	--	--	--	--		
						Chemical Total	--	1E-05	--	1E-05				1E-02
						Exposure Point Total				1E-05				1E-02
				Exposure Medium Total				1E-05				1E-02		
Medium Total							1E-05				1E-02			

TABLE 9.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	2E-05	--	5E-06	3E-05	Developmental effects	4E-01	--	8E-02	4E-01
			ALUMINUM	--	--	--	--	Neurotoxicity	5E-03	--	--	5E-03
			ARSENIC	4E-06	--	9E-07	5E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	5E-03	2E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-03	--	--	1E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-02	--	8E-03	3E-02
			CHROMIUM	--	--	--	--	--	3E-02	--	--	3E-02
			COPPER	--	--	--	--	Gastrointestinal effects	4E-03	--	--	4E-03
			IRON	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	1E-03	--	--	1E-03
			MERCURY	--	--	--	--	Autoimmune effects	3E-02	--	--	3E-02
			SILVER	--	--	--	--	Argyria (In)	2E-03	--	--	2E-03
			THALLIUM	--	--	--	--	Hematological effects	7E-03	--	--	7E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	9E-07	--	1E-06	2E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	5E-02	--	6E-02	1E-01
			LESS CHLORINATED PCBs	4E-07	--	5E-07	9E-07	Reduced birth weights (W)	7E-03	--	8E-03	1E-02
			DIELDRIN	5E-07	--	--	5E-07	Hepatic (H)	2E-03	--	--	2E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E-03	--	2E-03	4E-03
			ACENAPHTHYLENE	--	--	--	--	--	9E-05	--	9E-05	2E-04
			BENZ(A)ANTHRACENE	2E-06	--	2E-06	3E-06	--	--	--	--	--
			BENZO(A)PYRENE	2E-05	--	2E-05	4E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	1E-06	3E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-04	--	1E-04	2E-04
			BENZO(K)FLUORANTHENE	1E-07	--	1E-07	2E-07	--	--	--	--	--
			CHRYSENE	2E-08	--	2E-08	4E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-06	--	4E-06	7E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-03	--	2E-03	5E-03
			HEXACHLOROBENZENE	4E-07	--	4E-07	8E-07	Hepatic (H)	8E-04	--	7E-04	1E-03
			INDENO(1,2,3-CD)PYRENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	8E-04	--	8E-04	2E-03
			PHENANTHRENE	--	--	--	--	--	4E-04	--	5E-04	9E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	4E-09	--	--	4E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-04	--	--	3E-04
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	6E-05	--	--	6E-05
			1,4-DICHLOROBENZENE	5E-08	--	--	5E-08	Liver	3E-04	--	--	3E-04
			BENZENE	9E-09	--	--	9E-09	Reduced lymphocyte count	9E-05	--	--	9E-05

TABLE 9.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 6	P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	
			Chemical Total	6E-05	--	4E-05	9E-05		6E-01	--	2E-01	7E-01
		Exposure Point Total				9E-05					7E-01	
	Exposure Medium Total				9E-05					7E-01		
Medium Total							9E-05				7E-01	
Ground Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicity	7E-01	--	4E-03	7E-01
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	2E-01	--	5E-03	2E-01
			ARSENIC	2E-04	--	9E-07	2E-04	Hyperpigmentation (In); Vascular (V); PNS (N)	9E-01	--	4E-03	9E-01
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	2E-01	--	2E-02	2E-01
			BERYLLIUM	--	--	--	--	Small intestinal lesions	1E-02	--	8E-03	2E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-02	--	1E-02	7E-02
			CHROMIUM	--	--	--	--	--	6E-01	--	3E-01	9E-01
			COBALT	--	--	--	--	--	--	--	--	--
			COPPER	--	--	--	--	Gastrointestinal effects	6E-02	--	3E-04	6E-02
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	4E-02	--	2E-04	4E-02
			IRON	--	--	--	--	Gastrointestinal effects	2E+00	--	8E-03	2E+00
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	4E-01	--	5E-02	4E-01
			MERCURY	--	--	--	--	Autoimmune effects	2E-01	--	1E-02	2E-01
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	7E-02	--	2E-03	7E-02
			SELENIUM	--	--	--	--	Clinical selenosis	2E-02	--	1E-04	2E-02
			SILVER	--	--	--	--	Argyria (In)	1E-02	--	9E-04	1E-02
			THALLIUM	--	--	--	--	Hematological effects	2E+00	--	1E-02	2E+00
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-01	--	3E-02	2E-01
			ZINC	--	--	--	--	Decreased ESOD (B)	9E-03	--	3E-05	9E-03
			HIGHLY CHLORINATED PCBs	2E-06	--	--	2E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	--	1E-01
			4,4'-DDD	2E-07	--	2E-06	2E-06	--	--	--	--	--
			4,4'-DDT	4E-06	--	5E-05	6E-05	Liver lesions (H)	6E-02	--	7E-01	8E-01
			ALDRIN	7E-06	--	6E-07	7E-06	Liver toxicity (H)	3E-02	--	3E-03	3E-02
			ALPHA-BHC	1E-05	--	--	1E-05	--	--	--	--	--
			ENDOSULFAN II	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	3E-04	--	--	3E-04
			ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	1E-04	--	--	1E-04
			HEPTACHLOR EPOXIDE	1E-06	--	--	1E-06	Increased liver-to-body weight ratio in males and females (H)	2E-02	--	--	2E-02

TABLE 9.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	7E-03	--	--	7E-03
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	9E-02	--	3E-02	1E-01
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	6E+00	--	8E-01	6E+00
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	4E+00	--	--	4E+00
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	5E-01	--	5E-02	6E-01
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	2E+00	--	2E-01	3E+00
			4-CHLORO-3-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			4-METHYLPHENOL	--	--	--	--	--	5E+00	--	4E-01	5E+00
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	5E-02	--	--	5E-02
			ACENAPHTHYLENE	--	--	--	--	--	2E-01	--	--	2E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	1E-02	--	--	1E-02
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	4E-02	--	--	4E-02
			BENZ(A)ANTHRACENE	5E-04	--	6E-03	6E-03	--	--	--	--	--
			BENZO(A)PYRENE	2E-03	--	4E-02	4E-02	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-04	--	4E-03	4E-03	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-03	--	--	5E-03
			BENZO(K)FLUORANTHENE	2E-05	--	--	2E-05	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	2E-06	--	3E-06	4E-06	Increased relative liver weight (H)	1E-02	--	2E-02	4E-02
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	3E-06	--	4E-05	4E-05	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-04	--	8E-03	8E-03	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E+00	--	--	5E+00
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	1E-01	--	6E-01	7E-01
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	1E-01	--	--	1E-01
			HEXACHLOROBUTADIENE	9E-07	--	2E-06	3E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	7E-05	--	2E-03	2E-03	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	5E+00	--	4E+00	9E+00
			NITROBENZENE	--	--	--	--	Hematologic (B), adrenal, renal (R) and hepatic (H) lesions	1E-01	--	--	1E-01
			PHENANTHRENE	--	--	--	--	--	4E-01	--	1E+00	1E+00
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	2E-01	--	9E-03	2E-01
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	9E-02	--	--	9E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	6E-07	--	8E-07	1E-06	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	4E-02	--	5E-02	9E-02

TABLE 9.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	2E-01	--	1E-01	3E-01
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	3E-05	--	2E-05	5E-05	Liver	2E-01	--	1E-01	3E-01
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	3E-04	--	--	3E-04
			ACETONE	--	--	--	--	Nephropathy	2E-03	--	--	2E-03
			BENZENE	4E-03	--	6E-04	4E-03	Reduced lymphocyte count	4E+01	--	6E+00	5E+01
			BROMODICHLOROMETHANE	2E-06	--	2E-07	2E-06	Renal cytomegaly (R)	4E-03	--	3E-04	4E-03
			CARBON DISULFIDE	--	--	--	--	Fetal toxicity/malformations	3E-03	--	6E-04	4E-03
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-01	--	9E-02	3E-01
			CHLOROETHANE	--	--	--	--	--	--	--	--	--
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	4E-02	--	2E-02	6E-02
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	1E-03	--	--	1E-03
			METHYLENE CHLORIDE	7E-08	--	2E-09	7E-08	Liver toxicity (H)	3E-04	--	1E-05	4E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	1E-01	--	5E-02	2E-01
			TETRACHLOROETHENE	2E-06	--	1E-06	3E-06	Hepatotoxicity in mice (H), weight gain in rats	8E-04	--	5E-04	1E-03
			TOLUENE	--	--	--	--	Increased kidney weight (R)	4E-01	--	2E-01	6E-01
			VINYL CHLORIDE	1E-05	--	5E-07	1E-05	Liver cell polymorphism (H)	1E-02	--	5E-04	1E-02
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-01	--	--	1E-01
			Chemical Total			7E-03	--	6E-02	6E-02	8E+01	--	1E+01
	Exposure Point Total			6E-02				9E+01				
	Exposure Medium Total			6E-02				9E+01				
	Shower Vapor	Exposure Unit 8	Exposure Unit 8	1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--
1,2,4-TRICHLOROBENZENE				--	--	--	--	--	--	--	--	
1,2,4-TRIMETHYLBENZENE				--	--	--	--	Hematological and Pulmonary	--	1E+01	--	1E+01
1,2-DICHLOROBENZENE				--	--	--	--	--	1E+00	--	1E+00	
1,3,5-TRIMETHYLBENZENE				--	--	--	--	--	--	--	--	
1,3-DICHLOROBENZENE				--	--	--	--	--	--	--	--	
1,4-DICHLOROBENZENE				--	6E-04	--	6E-04	Liver	--	2E-01	--	2E-01
2-HEXANONE				--	--	--	--	Peripheral neuropathy	--	3E-03	--	3E-03
ACETONE				--	--	--	--	Neurological effects	--	7E-04	--	7E-04
BENZENE				--	5E-03	--	5E-03	Decreased lymphocyte count	--	5E+01	--	5E+01
BROMODICHLOROMETHANE				--	1E-05	--	1E-05	--	--	--	--	
CARBON DISULFIDE				--	--	--	--	Peripheral nervous system dysfunction	--	5E-03	--	5E-03
CHLOROBENZENE				--	--	--	--	--	--	--	--	
CHLOROETHANE				--	--	--	--	Delayed fetal ossification	--	1E-04	--	1E-04
CHLOROFORM				--	3E-05	--	3E-05	Hepatic effects	--	3E-02	--	3E-02
ETHYLBENZENE				--	--	--	--	Developmental toxicity	--	4E-02	--	4E-02

TABLE 9.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shower Vapor	Exposure Unit 8	ISOPROPYLBENZENE	--	--	--	--	Increased kidney and adrenal weights	--	3E-03	--	3E-03
			METHYLENE CHLORIDE	--	4E-08	--	4E-08	Hepatic effects	--	2E-04	--	2E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Central nervous system effects	--	2E-02	--	2E-02
			TETRACHLOROETHENE	--	2E-10	--	2E-10	Neurological effects	--	3E-04	--	3E-04
			TOLUENE	--	--	--	--	Neurological effects	--	7E-02	--	7E-02
			VINYL CHLORIDE	--	6E-07	--	6E-07	Liver cell polymorphism	--	3E-03	--	3E-03
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	2E+00	--	2E+00
		Chemical Total	--	6E-03	--	6E-03		--	7E+01	--	7E+01	
	Exposure Point Total		6E-03		7E+01							
	Exposure Medium Total		6E-03		7E+01							
Medium Total				7E-02		2E+02						
Receptor Total				7E-02		Receptor HI Total 2E+02						

Total Risk Across All Media = 7E-02

Total Hazard Across All Media = 2E+02

Total Liver HI Across All Media =	3E+00
Total Kidney HI Across All Media =	7E-01
Total Nervous System Effects HI Across All Media =	1E+01
Total Lymphocyte Effects HI Across All Media =	1E+02
Total Nasal/Respiratory Effects HI Across All Media =	2E+01
Total Ocular Effects HI Across All Media =	2E-01
Total Other Effects HI Across All Media =	3E+01

TABLE 9.13a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	3E-04	--	3E-04
			ARSENIC	--	3E-09	--	3E-09	Development, cardiovascular, nervous system	--	3E-05	--	3E-05
			CADMIUM	--	4E-09	--	4E-09	--	--	--	--	
			CHROMIUM	--	2E-07	--	2E-07	--	--	3E-04	--	3E-04
			COPPER	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E-03	--	2E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-06	--	2E-06
			VANADIUM	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	6E-11	--	6E-11	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	
			PHENANTHRENE	--	--	--	--	--	--	--	--	
			BENZENE	--	1E-09	--	1E-09	Decreased lymphocyte count	--	1E-05	--	1E-05
			Chemical Total	--	2E-07	--	2E-07		--	--	--	2E-03
		Exposure Point Total					2E-07					2E-03
	Exposure Medium Total					2E-07					2E-03	
Medium Total						2E-07						2E-03
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	1E-06	--	3E-07	2E-06	--	2E-02	--	5E-03	3E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	4E-03	--	--	4E-03
			ARSENIC	3E-06	--	7E-07	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	3E-03	2E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-02	--	4E-03	2E-02
			CHROMIUM	--	--	--	--	--	3E-02	--	--	3E-02
			COPPER	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	4E-03	--	--	4E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-03	--	--	1E-03
			HIGHLY CHLORINATED PCBs	5E-07	--	6E-07	1E-06	--	3E-02	--	4E-02	7E-02
			ACENAPHTHYLENE	--	--	--	--	--	5E-05	--	5E-05	9E-05
			BENZ(A)ANTHRACENE	2E-06	--	2E-06	4E-06	--	--	--	--	--

TABLE 9.13a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
			BENZO(A)PYRENE	1E-05	--	1E-05	3E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-06	--	2E-06	4E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-05	--	6E-05	1E-04
			BENZO(K)FLUORANTHENE	7E-08	--	7E-08	1E-07	--	--	--	--	--
			CHRYSENE	2E-08	--	2E-08	4E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	1E-06	3E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-03	--	9E-04	2E-03
			INDENO(1,2,3-CD)PYRENE	4E-07	--	4E-07	8E-07	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	3E-04	--	3E-04	7E-04
			BENZENE	2E-11	--	--	2E-11	Reduced lymphocyte count	2E-07	--	--	2E-07
			Chemical Total	2E-05	--	2E-05	5E-05		1E-01	--	5E-02	2E-01
		Exposure Point Total					5E-05					2E-01
	Exposure Medium Total						5E-05					2E-01
Medium Total							5E-05					2E-01
Receptor Total							5E-05				Receptor HI Total	2E-01

Total Risk Across All Media = 5E-05

Total Hazard Across All Media = 2E-01

RAGS Table 9 CT Series

TABLE 9.1 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-05	--	--	2E-05	Developmental effects	1E+00	--	--	1E+00		
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	2E-01	--	--	2E-01		
			ARSENIC	1E-06	--	--	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	--	3E-02		
			CHROMIUM	--	--	--	--	--	2E-02	--	--	2E-02		
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	3E-02	--	--	3E-02		
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03		
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	1E+00	--	--	1E+00		
			SELENIUM	--	--	--	--	Clinical selenosis	3E-02	--	--	3E-02		
			VANADIUM	--	--	--	--	Decreased hair cystine	7E-03	--	--	7E-03		
			ZINC	--	--	--	--	Decreased ESOD (B)	1E-02	--	--	1E-02		
			HIGHLY CHLORINATED PCBs	6E-06	--	--	6E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+00	--	--	2E+00		
			LESS CHLORINATED PCBs	5E-06	--	--	5E-06	Reduced birth weights (W)	4E-01	--	--	4E-01		
			4,4-DDD	3E-08	--	--	3E-08	--	--	--	--	--		
			4,4'-DDT	3E-08	--	--	3E-08	Liver lesions (H)	2E-03	--	--	2E-03		
			ALDRIN	3E-07	--	--	3E-07	Liver toxicity (H)	8E-03	--	--	8E-03		
			DELTA-BHC	--	--	--	--	--	--	--	--	--		
			DIELDRIN	5E-07	--	--	5E-07	Hepatic (H)	7E-03	--	--	7E-03		
			HEPTACHLOR EPOXIDE	3E-07	--	--	3E-07	Increased liver-to-body weight ratio in males and females (H)	3E-02	--	--	3E-02		
			BIS(2-ETHYLHEXYL)PHTHALATE	3E-07	--	--	3E-07	Increased relative liver weight (H)	1E-02	--	--	1E-02		
			HEXACHLOROBENZENE	2E-07	--	--	2E-07	Hepatic (H)	2E-03	--	--	2E-03		
			Chemical Total	3E-05	--	--	3E-05		5E+00	--	--	5E+00		
			Exposure Point Total						3E-05					5E+00
			Exposure Medium Total						3E-05					5E+00
		Medium Total						3E-05					5E+00	
Sediment	Surface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	7E-08	--	4E-08	1E-07	Developmental effects	5E-03	--	7E-02	7E-02		
			ARSENIC	7E-08	--	4E-08	1E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-03	--	2E-02	2E-02		
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-03	--	2E-03	7E-03		
			CHROMIUM	--	--	--	--	None Reported (O)	5E-02	--	--	5E-02		
			IRON	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--	4E-03		
			MERCURY	--	--	--	--	Autoimmune effects	2E-02	--	--	2E-02		
			THALLIUM	--	--	--	--	Hematological effects	6E-04	--	--	6E-04		
			VANADIUM	--	--	--	--	Decreased hair cystine	5E-03	--	--	5E-03		

TABLE 9.1 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	HIGHLY CHLORINATED PCBs	1E-08	--	3E-08	4E-08	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-03	--	2E-01	2E-01
			DIELDRIN	2E-09	--	--	2E-09	Hepatic (H)	3E-05	--	--	3E-05
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	1E-05	--	--	1E-05
			HEPTACHLOR EPOXIDE	5E-10	--	--	5E-10	Increased liver-to-body weight ratio in males and females (H)	5E-05	--	--	5E-05
			1-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	6E-04	--	3E-02	3E-02
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-05	--	5E-04	5E-04
			BENZ(A)ANTHRACENE	2E-06	--	9E-06	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	4E-06	--	2E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-07	--	3E-06	4E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-04	--	6E-03	6E-03
			BENZO(K)FLUORANTHENE	2E-08	--	1E-07	1E-07	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	4E-09	--	8E-09	1E-08	Increased relative liver weight (H)	1E-04	--	6E-03	6E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	5E-09	--	3E-08	3E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	6E-07	--	3E-06	4E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	7E-02	7E-02
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-04	--	1E-02	1E-02
			HEXACHLOROBENZENE	1E-09	--	3E-09	4E-09	Hepatic (H)	1E-05	--	5E-04	5E-04
			INDENO(1,2,3-CD)PYRENE	2E-07	--	1E-06	1E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-04	--	1E-02	1E-02
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-04	--	2E-02	2E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	5E-04	--	2E-02	3E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	1E-05	--	--	1E-05
			1,2,4-TRICHLOROBENZENE	--	--	--	--	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	--	--	--	--
			1,2-DICHLOROBENZENE	1E-11	--	--	1E-11	No adverse effects observed (O)	4E-06	--	--	4E-06
			1,3,5-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	--	--	Liver	--	--	--	--
			BENZENE	8E-10	--	--	8E-10	Reduced lymphocyte count	3E-05	--	--	3E-05
			CHLOROBENZENE	1E-09	--	--	1E-09	Histopathologic changes in liver	8E-05	--	--	8E-05
			METHYLENE CHLORIDE	--	--	--	--	Liver toxicity (H)	8E-05	--	--	8E-05
			N-HEXADACANE	2E-11	--	--	2E-11	--	5E-07	--	--	5E-07

TABLE 9.1 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	--	--
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-05	--	--	1E-05
		Chemical Total	7E-06	--	4E-05	4E-05		1E-01	--	5E-01	6E-01	
		Exposure Point Total				4E-05					6E-01	
	Exposure Medium Total				4E-05					6E-01		
Medium Total							4E-05				6E-01	
Soil	Surface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	5E-07	--	3E-07	9E-07	Developmental effects	4E-02	--	3E-02	7E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	6E-04	--	--	6E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	1E-04	--	--	1E-04
			ARSENIC	9E-08	--	6E-08	2E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-03	--	2E-03	4E-03
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-04	--	--	1E-04
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-03	--	2E-03	4E-03
			CHROMIUM	--	--	--	--	--	3E-03	--	--	3E-03
			COPPER	--	--	--	--	Gastrointestinal effects	4E-04	--	--	4E-04
			IRON	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	2E-04	--	--	2E-04
			MERCURY	--	--	--	--	Autoimmune effects	2E-03	--	--	2E-03
			SILVER	--	--	--	--	Argyria (In)	2E-04	--	--	2E-04
			THALLIUM	--	--	--	--	Hematological effects	7E-04	--	--	7E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-04	--	--	2E-04
			HIGHLY CHLORINATED PCBs	2E-08	--	6E-08	8E-08	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-03	--	2E-02	2E-02
			LESS CHLORINATED PCBs	1E-08	--	4E-08	5E-08	Reduced birth weights (W)	1E-03	--	3E-03	4E-03
			DIELDRIN	1E-09	--	--	1E-09	Hepatic (H)	2E-05	--	--	2E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E-04	--	5E-04	6E-04
			ACENAPHTHYLENE	--	--	--	--	--	1E-05	--	4E-05	5E-05
			BENZ(A)ANTHRACENE	9E-08	--	5E-07	6E-07	--	--	--	--	--
			BENZO(A)PYRENE	9E-07	--	5E-06	6E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	8E-08	--	4E-07	5E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	3E-05	--	7E-05	1E-04
			BENZO(K)FLUORANTHENE	7E-09	--	4E-08	4E-08	--	--	--	--	--
			CHRYSENE	9E-10	--	5E-09	6E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-07	--	1E-06	1E-06	--	--	--	--	--

TABLE 9.1 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 1	DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O). Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-04	--	6E-04	8E-04
			FLUORANTHENE	--	--	--	--		6E-05	--	2E-04	2E-04
			HEXACHLOROBENZENE	7E-09	--	2E-08	2E-08		Hepatic (H)	6E-05	--	1E-04
			INDENO(1,2,3-CD)PYRENE	5E-08	--	3E-07	3E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	6E-05	--	2E-04	2E-04
			PHENANTHRENE	--	--	--	--	--	6E-05	--	2E-04	2E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	7E-11	--	--	7E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	2E-05	--	--	2E-05
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	5E-06	--	--	5E-06
			1,4-DICHLOROBENZENE	7E-10	--	--	7E-10	Liver	2E-05	--	--	2E-05
			BENZENE	1E-10	--	--	1E-10	Reduced lymphocyte count	7E-06	--	--	7E-06
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
	Chemical Total			2E-06	--	7E-06	9E-06		6E-02	--	5E-02	1E-01
Exposure Point Total							9E-06					1E-01
Exposure Medium Total							9E-06					1E-01
Medium Total							9E-06					1E-01
Surface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E-05	--	1E-05
			ANTIMONY	--	--	--	--	--	--	--	--	--
			ARSENIC	--	3E-11	--	3E-11	Development, cardiovascular, nervous system	--	2E-06	--	2E-06
			BARIUM	--	--	--	--	Renal toxicity	--	5E-06	--	5E-06
			CADMIUM	--	4E-11	--	4E-11	--	--	--	--	--
			CHROMIUM	--	1E-09	--	1E-09	--	1E-05	--	1E-05	
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	6E-05	--	6E-05
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	3E-07	--	3E-07
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	7E-13	--	7E-13	--	--	--	--	--
			LESS CHLORINATED PCBs	--	4E-13	--	4E-13	--	--	--	--	--
			DIELDRIN	--	4E-14	--	4E-14	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.1 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 1	BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORANTHENE	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	2E-13	--	2E-13	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	4E-13	--	4E-13	Nasal/respiratory (P)	--	5E-08	--	5E-08
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	6E-05	--	6E-05
			1,4-DICHLOROBENZENE	--	3E-08	--	3E-08	Liver	--	4E-05	--	4E-05
			BENZENE	--	2E-09	--	2E-09	Decreased lymphocyte count	--	9E-05	--	9E-05
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
	DODECANE	--	--	--	--	--	--	--	--	--		
		Chemical Total	--	3E-08	--	3E-08		--	3E-04	--	3E-04	
		Exposure Point Total					3E-08					3E-04
		Exposure Medium Total					3E-08					3E-04
Medium Total							3E-08					3E-04
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	5E-04	5E-04
			ARSENIC	--	--	7E-09	7E-09	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	2E-04	2E-04
			CHROMIUM	--	--	--	--	(N)	--	--	3E-03	3E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	1E-04	1E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	1E-03	1E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	8E-05	8E-05
			THALLIUM	--	--	--	--	Hematological effects	--	--	8E-04	8E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	1E-04	1E-04
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	1E-05	1E-05
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	7E-04	7E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	2E-04	2E-04
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	1E-05	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-04	1E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-05	2E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
					BIS(2-ETHYLHEXYL)PHTHALATE	--	--	2E-08	2E-08	Increased relative liver weight (H)	--	--

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SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Water	Surface Water	Exposure Unit 1	CARBAZOLE	--	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	9E-08	9E-08	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	8E-06	8E-06	--	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	7E-02	7E-02	
			PHENANTHRENE	--	--	--	--	--	--	--	3E-03	3E-03	
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	4E-09	4E-09	Liver	--	--	1E-04	1E-04	
			BENZENE	--	--	7E-08	7E-08	Reduced lymphocyte count	--	--	3E-03	3E-03	
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	2E-03	2E-03	
		XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--	--	
		Chemical Total	--	--	2E-04	2E-04		--	--	8E-02	8E-02		
		Exposure Point Total				2E-04					8E-02		
		Exposure Medium Total				2E-04					8E-02		
Medium Total						2E-04					8E-02		
Receptor Total						2E-04				Receptor HI Total	6E+00		

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 6E+00

Total Liver HI Across All Media = 8E-02
Total Kidney HI Across All Media = 6E-02
Total Nervous System Effects HI Across All Media = 1E+00
Total Lymphocyte Effects HI Across All Media = 4E-03
Total Nasal/Respiratory Effects HI Across All Media = 3E-02
Total Ocular Effects HI Across All Media = 2E+00
Total Other Effects HI Across All Media = 2E+00

TABLE 9.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	3E-05	--	--	3E-05	Developmental effects	2E+00	--	--	2E+00		
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	3E-01	--	--	3E-01		
			ARSENIC	2E-06	--	--	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	--	3E-02		
			CHROMIUM	--	--	--	--	--	2E-02	--	--	2E-02		
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	3E-02	--	--	3E-02		
			MANGANESE	--	--	--	--	CNS (N)	3E-03	--	--	3E-03		
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	1E+00	--	--	1E+00		
			SELENIUM	--	--	--	--	Clinical selenosis	3E-02	--	--	3E-02		
			VANADIUM	--	--	--	--	Decreased hair cystine	8E-03	--	--	8E-03		
			ZINC	--	--	--	--	Decreased ESOD (B)	2E-02	--	--	2E-02		
			HIGHLY CHLORINATED PCBs	1E-05	--	--	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+00	--	--	2E+00		
			LESS CHLORINATED PCBs	9E-06	--	--	9E-06	Reduced birth weights (W)	5E-01	--	--	5E-01		
			4,4-DDD	5E-08	--	--	5E-08	--	--	--	--	--		
			4,4'-DDT	5E-08	--	--	5E-08	Liver lesions (H)	2E-03	--	--	2E-03		
			ALDRIN	6E-07	--	--	6E-07	Liver toxicity (H)	1E-02	--	--	1E-02		
			DELTA-BHC	--	--	--	--	--	--	--	--	--		
			DIELDRIN	9E-07	--	--	9E-07	Hepatic (H)	9E-03	--	--	9E-03		
			HEPTACHLOR EPOXIDE	5E-07	--	--	5E-07	Increased liver-to-body weight ratio in males and females (H)	4E-02	--	--	4E-02		
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-07	--	--	5E-07	Increased relative liver weight (H)	1E-02	--	--	1E-02		
			HEXACHLOROBENZENE	3E-07	--	--	3E-07	Hepatic (H)	2E-03	--	--	2E-03		
			Chemical Total	6E-05	--	--	6E-05		6E+00	--	--	6E+00		
			Exposure Point Total				6E-05				6E+00			
			Exposure Medium Total				6E-05				6E+00			
Medium Total				6E-05				6E+00						
Sediment	Surface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	4E-08	--	4E-08	8E-08	Developmental effects	2E-03	--	2E-03	4E-03		
			ARSENIC	4E-08	--	4E-08	8E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	7E-04	--	7E-04	1E-03		
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-03	--	6E-05	2E-03		
			CHROMIUM	--	--	--	--	None Reported (O)	2E-02	--	--	2E-02		
			IRON	--	--	--	--	Gastrointestinal effects	--	--	--	--		
			LEAD	--	--	--	--	--	1E-03	--	--	1E-03		
			MANGANESE	--	--	--	--	CNS (N)	8E-03	--	--	8E-03		
			MERCURY	--	--	--	--	Autoimmune effects	2E-04	--	--	2E-04		
			THALLIUM	--	--	--	--	Hematological effects	2E-03	--	--	2E-03		

TABLE 9.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	VANADIUM	6E-09	--	--	6E-09	Decreased hair cystine	1E-03	--	--	1E-03
			HIGHLY CHLORINATED PCBs	1E-09	--	3E-08	3E-08	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-05	--	6E-03	6E-03
			DIELDRIN	--	--	--	--	Hepatic (H)	5E-06	--	--	5E-06
			ENDRIN KETONE	3E-10	--	--	3E-10	Mild histological lesions in liver (H), occasional convulsions	2E-05	--	--	2E-05
			HEPTACHLOR EPOXIDE	--	--	--	--	Increased liver-to-body weight ratio in males and females (H)	--	--	--	--
			1-METHYLNAPHTHALENE	--	--	--	--	--	2E-04	--	--	2E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	4E-06	--	1E-03	1E-03
			ACENAPHTHYLENE	8E-07	--	--	8E-07	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	2E-05	2E-05
			BENZ(A)ANTHRACENE	2E-06	--	4E-06	5E-06	--	--	--	--	--
			BENZO(A)PYRENE	3E-07	--	8E-06	8E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	1E-06	1E-06	--	4E-05	--	--	4E-05
			BENZO(G,H,I)PERYLENE	1E-08	--	--	1E-08	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	2E-04	2E-04
			BENZO(K)FLUORANTHENE	2E-09	--	5E-08	5E-08	--	6E-05	--	--	6E-05
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	7E-09	7E-09	Increased relative liver weight (H)	--	--	2E-04	2E-04
			CARBAZOLE	3E-09	--	--	3E-09	--	--	--	--	--
			CHRYSENE	3E-07	--	1E-08	3E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	1E-06	1E-06	--	7E-04	--	--	7E-04
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-04	--	2E-03	2E-03
			FLUORANTHENE	8E-10	--	--	8E-10	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	5E-06	--	5E-04	5E-04
			HEXACHLOROBENZENE	1E-07	--	3E-09	1E-07	Hepatic (H)	--	--	2E-05	2E-05
			INDENO(1,2,3-CD)PYRENE	--	--	4E-07	4E-07	--	9E-05	--	--	9E-05
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E-04	--	4E-04	5E-04
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-04	--	5E-04	7E-04
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-08	--	8E-04	8E-04
			1,2,3-TRICHLOROBENZENE	7E-12	--	--	7E-12	--	2E-06	--	--	2E-06
			1,2,4-TRICHLOROBENZENE	--	--	--	--	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	--	--
			1,3,5-TRICHLOROBENZENE	5E-10	--	--	5E-10	--	1E-05	--	--	1E-05
			1,4-DICHLOROBENZENE	9E-10	--	--	9E-10	Liver	3E-05	--	--	3E-05
			BENZENE	--	--	--	--	Reduced lymphocyte count	3E-05	--	--	3E-05
			CHLOROBENZENE	1E-11	--	--	1E-11	Histopathologic changes in liver	2E-07	--	--	2E-07
			METHYLENE CHLORIDE	--	--	--	--	Liver toxicity (H)	--	--	--	--
			N-HEXADACANE	--	--	--	--	--	--	--	--	--
			P-ISOPROPYLTOLUENE	--	--	--	--	--	4E-06	--	--	4E-06

TABLE 9.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 1	TOLUENE	--	--	--	--	Increased kidney weight (R)	5E-06	--	--	5E-06
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total	3E-06	--	2E-05	2E-05		4E-02	--	1E-02	5E-02
		Exposure Point Total				2E-05				5E-02		
	Exposure Medium Total				2E-05				5E-02			
Medium Total						2E-05				5E-02		
Soil	Surface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	3E-07	--	3E-07	6E-07	Developmental effects	2E-02	--	2E-02	3E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	2E-04	--	--	2E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	5E-05	--	--	5E-05
			ARSENIC	6E-08	--	6E-08	1E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-03	--	1E-03	2E-03
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	4E-05	--	--	4E-05
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	8E-04	--	1E-03	2E-03
			CHROMIUM	--	--	--	--	--	1E-03	--	--	1E-03
			COPPER	--	--	--	--	Gastrointestinal effects	2E-04	--	--	2E-04
			IRON	--	--	--	--	Gastrointestinal effects	6E-04	--	--	6E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	7E-05	--	--	7E-05
			MERCURY	--	--	--	--	Autoimmune effects	9E-04	--	--	9E-04
			SILVER	--	--	--	--	Argyria (In)	6E-05	--	--	6E-05
			THALLIUM	--	--	--	--	Hematological effects	3E-04	--	--	3E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	7E-05	--	--	7E-05
								Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-03	--	1E-02	1E-02
								Reduced birth weights (W)	4E-04	--	2E-03	2E-03
								Hepatic (H)	7E-06	--	--	7E-06
								Pulmonary alveolar proteinosis	7E-05	--	3E-04	4E-04
								--	6E-06	--	3E-05	3E-05
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TABLE 9.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 1	INDENO(1,2,3-CD)PYRENE	3E-08	--	1E-07	1E-07	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-05	--	1E-04	1E-04
			PHENANTHRENE	--	--	--	--	--	2E-05	--	1E-04	1E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	4E-11	--	--	4E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	9E-06	--	--	9E-06
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	2E-06	--	--	2E-06
			1,4-DICHLOROBENZENE	4E-10	--	--	4E-10	Liver	9E-06	--	--	9E-06
			BENZENE	8E-11	--	--	8E-11	Reduced lymphocyte count	3E-06	--	--	3E-06
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
		DODECANE	--	--	--	--	--	--	--	--	--	
		Chemical Total	1E-06	--	3E-06	4E-06		2E-02	--	3E-02	6E-02	
		Exposure Point Total				4E-06					6E-02	
		Exposure Medium Total				4E-06					6E-02	
Medium Total						4E-06					6E-02	
Surface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	9E-06	--	9E-06
			ANTIMONY	--	--	--	--	--	--	--	--	--
			ARSENIC	--	3E-11	--	3E-11	Development, cardiovascular, nervous system	--	1E-06	--	1E-06
			BARIUM	--	--	--	--	Renal toxicity	--	4E-06	--	4E-06
			CADMIUM	--	4E-11	--	4E-11	--	--	--	--	--
			CHROMIUM	--	1E-09	--	1E-09	--	--	7E-06	--	7E-06
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	4E-05	--	4E-05
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-07	--	2E-07
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	7E-13	--	7E-13	--	--	--	--	--
			LESS CHLORINATED PCBs	--	4E-13	--	4E-13	--	--	--	--	--
			DIELDRIN	--	4E-14	--	4E-14	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORANTHENE	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	2E-13	--	2E-13	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--

TABLE 9.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 1	NAPHTHALENE	--	4E-13	--	4E-13	Nasal/respiratory (P)	--	3E-08	--	3E-08
			PHENANTHRENE	--	--	--	--	--	--	--	--	
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	
			1,2-DICHLOROBENZENE	--	--	--	--	--	4E-05	--	4E-05	
			1,4-DICHLOROBENZENE	--	3E-08	--	3E-08	Liver	--	3E-05	--	3E-05
			BENZENE	--	2E-09	--	2E-09	Decreased lymphocyte count	--	6E-05	--	6E-05
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	
			DODECANE	--	--	--	--	--	--	--	--	
		Chemical Total	--	3E-08	--	3E-08		--	2E-04	--	2E-04	
	Exposure Point Total			3E-08					2E-04			
	Exposure Medium Total			3E-08					2E-04			
Medium Total						3E-08				2E-04		
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	4E-04	4E-04
			ARSENIC	--	--	9E-09	9E-09	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	2E-04	2E-04
			CHROMIUM	--	--	--	--	--	--	--	2E-03	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	1E-04	1E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	1E-03	1E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	6E-05	6E-05
			THALLIUM	--	--	--	--	Hematological effects	--	--	7E-04	7E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	1E-04	1E-04
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	1E-05	1E-05
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	6E-04	6E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	2E-04	2E-04
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	7E-06	7E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	6E-05	6E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	9E-06	9E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	2E-08	2E-08	Increased relative liver weight (H)	--	--	7E-04	7E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	5E-08	5E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	4E-06	4E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	6E-02	6E-02
			PHENANTHRENE	--	--	--	--	--	--	--	2E-03	2E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--

TABLE 9.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Trespasser
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	6E-09	6E-09	Liver	--	--	1E-04	1E-04
			BENZENE	--	--	8E-08	8E-08	Reduced lymphocyte count	--	--	3E-03	3E-03
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	2E-03	2E-03
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total	--	--	8E-05	8E-05		--	--	7E-02	7E-02
		Exposure Point Total					8E-05					7E-02
	Exposure Medium Total						8E-05					7E-02
Medium Total							8E-05					7E-02
Receptor Total							2E-04					6E+00

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 6E+00

Total Liver HI Across All Media = 7E-02
 Total Kidney HI Across All Media = 7E-03
 Total Nervous System Effects HI Across All Media = 1E+00
 Total Lymphocyte Effects HI Across All Media = 3E-03
 Total Nasal/Respiratory Effects HI Across All Media = 1E-03
 Total Ocular Effects HI Across All Media = 2E+00
 Total Other Effects HI Across All Media = 3E+00

TABLE 9.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-08	--	4E-09	3E-08	Developmental effects	1E+03	--	2E+02	1E+03
			ARSENIC	1E-08	--	2E-09	1E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-04	--	4E-05	2E-04
			BARIIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-05	--	--	1E-05
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-05	--	6E-06	3E-05
			CHROMIUM	--	--	--	--	None Reported (O)	3E-04	--	--	3E-04
			IRON	--	--	--	--	Gastrointestinal effects	3E-04	--	--	3E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	5E-05	--	--	5E-05
			MERCURY	--	--	--	--	Autoimmune effects	3E-04	--	--	3E-04
			THALLIUM	--	--	--	--	Hematological effects	1E-04	--	--	1E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-05	--	--	3E-05
			HIGHLY CHLORINATED PCBs	3E-09	--	3E-09	7E-09	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	7E-04	--	6E-04	1E-03
			DELTA-BHC	--	--	--	--	--	--	--	--	--
			DIELDRIN	5E-10	--	--	5E-10	Hepatic (H)	5E-06	--	--	5E-06
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	--	--	--	--
			HEPTACHLOR EPOXIDE	--	--	--	--	Increased liver-to-body weight ratio in males and females (H)	--	--	--	--
			1-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E-02	--	9E-03	2E-02
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	1E-04	--	1E-04	3E-04
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	4E-04	--	3E-04	7E-04
			ANTHRACENE	--	--	--	--	No observed effects (O)	2E-05	--	2E-05	4E-05
			BENZ(A)ANTHRACENE	3E-07	--	3E-07	6E-07	--	--	--	--	--
			BENZO(A)PYRENE	8E-07	--	7E-07	1E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-07	--	1E-07	2E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-05	--	1E-05	3E-05
			BENZO(K)FLUORANTHENE	4E-09	--	4E-09	8E-09	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	--	--	Increased relative liver weight (H)	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	3E-09	--	2E-09	5E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-07	--	1E-07	3E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-02	--	1E-02	2E-02
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-04	--	3E-04	6E-04
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	4E-04	--	3E-04	7E-04
			HEXACHLOROBENZENE	3E-10	--	2E-10	6E-10	Hepatic (H)	2E-06	--	1E-06	3E-06
			INDENO(1,2,3-CD)PYRENE	4E-08	--	3E-08	8E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	6E-03	--	5E-03	1E-02
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-03	--	1E-03	2E-03

TABLE 9.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-04	--	2E-04	5E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	0E+00
			1,2,4-TRICHLOROBENZENE	3E-12	--	--	3E-12	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	7E-07	--	--	7E-07
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	--	--
			1,3,5-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	2E-10	--	--	2E-10	Liver	4E-06	--	--	4E-06
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	2E-04	--	--	2E-04
			BENZENE	9E-09	--	--	9E-09	Reduced lymphocyte count	3E-04	--	--	3E-04
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	1E-05	--	--	1E-05
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	2E-05	--	--	2E-05
			METHYLENE CHLORIDE	2E-11	--	--	2E-11	Liver toxicity (H)	3E-07	--	--	3E-07
			N-HEXADACANE	--	--	--	--	--	--	--	--	--
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	1E-05	--	--	1E-05
		TOLUENE	--	--	--	--	Increased kidney weight (R)	6E-05	--	--	6E-05	
XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	6E-05	--	--	6E-05			
		Chemical Total	1E-06		1E-06	3E-06		1E+03		2E+02	1E+03	
		Exposure Point Total				3E-06					1E+03	
	Exposure Medium Total					3E-06					1E+03	
Medium Total						3E-06					1E+03	
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-07	--	4E-08	2E-07	Developmental effects	1E-02	--	2E-03	1E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	1E-04	--	--	1E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	3E-05	--	--	3E-05
			ARSENIC	4E-08	--	8E-09	5E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	7E-04	--	1E-04	9E-04
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	5E-05	--	--	5E-05
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	4E-04	--	1E-04	5E-04
			CHROMIUM	--	--	--	--	--	7E-04	--	--	7E-04
			COPPER	--	--	--	--	Gastrointestinal effects	9E-05	--	--	9E-05
			IRON	--	--	--	--	Gastrointestinal effects	4E-04	--	--	4E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	4E-05	--	--	4E-05
			MERCURY	--	--	--	--	Autoimmune effects	8E-04	--	--	8E-04
			SILVER	--	--	--	--	Argyria (In)	6E-05	--	--	6E-05
			THALLIUM	--	--	--	--	Hematological effects	3E-04	--	--	3E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	4E-05	--	--	4E-05
								Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-03	--	3E-03	5E-03
			HIGHLY CHLORINATED PCBs	1E-08	--	1E-08	3E-08	Reduced birth weights (W)	8E-04	--	8E-04	2E-03
			LESS CHLORINATED PCBs	2E-08	--	1E-08	3E-08	Hepatic (H)	4E-06	--	--	4E-06
			DIELDRIN	4E-10	--	--	4E-10	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	3E-06	--	2E-06	5E-06
			2,4-DIMETHYLPHENOL	--	--	--	--	Pulmonary alveolar proteinosis	1E-03	--	1E-03	2E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Decreased body weight and neurotoxicity	4E-06	--	2E-06	6E-06
			3&4-METHYLPHENOL	--	--	--	--					

TABLE 9.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	2E-05	--	2E-05	5E-05
			ACENAPHTHYLENE	--	--	--	--	--	3E-05	--	2E-05	5E-05
			ANTHRACENE	--	--	--	--	No observed effects (O)	9E-06	--	8E-06	2E-05
			BENZ(A)ANTHRACENE	2E-07	--	1E-07	3E-07	--	--	--	--	--
			BENZO(A)PYRENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-07	--	1E-07	3E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-05	--	1E-05	2E-05
			BENZO(K)FLUORANTHENE	7E-09	--	6E-09	1E-08	--	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	1E-09	--	1E-09	3E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-07	--	1E-07	2E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	1E-03	3E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	1E-04	--	1E-04	2E-04
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	7E-05	--	5E-05	1E-04
			HEXACHLOROBENZENE	3E-09	--	2E-09	5E-09	Hepatic (H)	2E-05	--	1E-05	3E-05
			INDENO(1,2,3-CD)PYRENE	4E-08	--	3E-08	7E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E-03	--	8E-04	2E-03
			PHENANTHRENE	--	--	--	--	--	3E-04	--	2E-04	5E-04
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-04	--	1E-04	3E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	1E-10	--	--	1E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-05	--	--	3E-05
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	2E-05	--	--	2E-05
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	2E-09	--	--	2E-09	Liver	3E-05	--	--	3E-05
			BENZENE	1E-09	--	--	1E-09	Reduced lymphocyte count	4E-05	--	--	4E-05
			BROMOMETHANE	--	--	--	--	Epithelial hyperplasia of the forestomach	1E-05	--	--	1E-05
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	8E-06	--	--	8E-06
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	5E-06	--	--	5E-06
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	4E-06	--	--	4E-06
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	2E-06	--	2E-06	4E-06		2E-02	--	9E-03	3E-02
		Exposure Point Total					4E-06					3E-02
	Exposure Medium Total						4E-06					3E-02
Medium Total							4E-06					3E-02
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E-02	--	1E-02
			ANTIMONY	--	--	--	--	--	--	--	--	--
			ARSENIC	--	6E-08	--	6E-08	Development, cardiovascular, nervous system	--	2E-03	--	2E-03

TABLE 9.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	CADMIUM	--	4E-08	--	4E-08	--	--	--	--	--
			CHROMIUM	--	1E-06	--	1E-06	--	--	9E-03	--	9E-03
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O) PNS (N); CNS (N)	--	6E-02	--	6E-02
			MERCURY	--	--	--	--		4E-04	--	4E-04	
			SILVER	--	--	--	--		--	--	--	
			THALLIUM	--	--	--	--		--	--	--	
			VANADIUM	--	--	--	--		--	--	--	
			HIGHLY CHLORINATED PCBs	--	2E-09	--	2E-09		--	--	--	--
			LESS CHLORINATED PCBs	--	2E-09	--	2E-09		--	--	--	--
			DIELDRIN	--	5E-11	--	5E-11		--	--	--	--
			2,4-DIMETHYLPHENOL	--	--	--	--		--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--		--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	--	--	--	--	
			ACENAPHTHENE	--	--	--	--	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	
			ANTHRACENE	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			CARBAZOLE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	
			FLUORANTHENE	--	--	--	--	--	--	--	--	
			FLUORENE	--	--	--	--	--	--	--	--	
			HEXACHLOROBENZENE	--	4E-10	--	4E-10	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	
			NAPHTHALENE	--	4E-08	--	4E-08	Nasal/respiratory (P)	--	3E-03	--	3E-03
			PHENANTHRENE	--	--	--	--		--	--	--	--
			PYRENE	--	--	--	--		--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	
			1,4-DICHLOROBENZENE	--	2E-07	--	2E-07	Liver	--	1E-04	--	1E-04
			BENZENE	--	4E-08	--	4E-08	Decreased lymphocyte count	--	1E-03	--	1E-03
			BROMOMETHANE	--	--	--	--	Nasal lesions and membrane degeneration	--	1E-03	--	1E-03
			TOLUENE	--	--	--	--	Neurological effects	--	1E-05	--	1E-05
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	3E-02	--	3E-02
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total			--	2E-06	--	2E-06			
	Exposure Point Total											1E-01
	Exposure Medium Total											1E-01
Medium Total											1E-01	

TABLE 9.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shallow Ground Water	Exposure Unit 1	ALUMINUM	--	--	--	--	Neurotoxicity	--	--	2E-05	2E-05
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	2E-04	2E-04
			ARSENIC	--	--	6E-09	6E-09	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	1E-04	1E-04
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	1E-03	1E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	4E-04	4E-04
			CHROMIUM	--	--	--	--	--	--	--	2E-03	2E-03
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	--	--	7E-06	7E-06
			IRON	--	--	--	--	Gastrointestinal effects	--	--	8E-05	8E-05
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	1E-03	1E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	3E-04	3E-04
			SILVER	--	--	--	--	Argyria (In)	--	--	4E-05	4E-05
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	2E-04	2E-04
			HIGHLY CHLORINATED PCBs	--	--	--	--	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	--	--	--	--
			4,4'-DDT	--	--	3E-09	3E-09	Liver lesions (H)	--	--	1E-04	1E-04
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	--	--	--	--
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response	--	--	6E-07	6E-07
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	3E-06	3E-06
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	--	--	2E-06	2E-06
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	5E-06	5E-06
			4-METHYLPHENOL	--	--	--	--	--	--	--	2E-06	2E-06
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ANTHRACENE	--	--	--	--	No observed effects (O)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	1E-08	1E-08	--	--	--	--	--
			BENZO(A)PYRENE	--	--	2E-07	2E-07	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	1E-08	1E-08	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	1E-11	1E-11	Increased relative liver weight (H)	--	--	4E-07	4E-07
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	1E-10	1E-10	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	8E-08	8E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	--	--	1E-05	1E-05
			FLUORENE	--	--	--	--	Decreased RBC (B); packed cell volumen and hemoglobin (B)	--	--	--	--
			HEXACHLOROBUTADIENE	--	--	7E-12	7E-12	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	2E-08	2E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	7E-05	7E-05

TABLE 9.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Ground Water	Shallow Ground Water	Exposure Unit 1	PHENANTHRENE	--	--	--	--	--	--	--	3E-05	3E-05		
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	--	--	3E-07	3E-07		
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--		
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLOROBENZENE	--	--	1E-11	1E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	--	--	2E-06	2E-06		
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	3E-06	3E-06		
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,4-DICHLOROBENZENE	--	--	1E-10	1E-10	Liver	--	--	3E-06	3E-06		
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	--	--	--	--		
			BENZENE	--	--	6E-10	6E-10	Reduced lymphocyte count	--	--	2E-05	2E-05		
			BROMODICHLOROMETHANE	--	--	1E-13	1E-13	Renal cytomegaly (R)	--	--	9E-10	9E-10		
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	--	--	2E-06	2E-06		
			CHLOROFORM	--	--	--	--	Moderate/marked fatty cyst formation in the liver and elevated SGPT	--	--	8E-09	8E-09		
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	--	--	2E-07	2E-07		
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats	--	--	--	--		
			METHYLENE CHLORIDE	--	--	5E-13	5E-13	Liver toxicity (H)	--	--	8E-09	8E-09		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--		
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	--	--	3E-07	3E-07		
			TETRACHLOROETHENE	--	--	6E-12	6E-12	Hepatotoxicity in mice (H), weight gain in rats	--	--	9E-09	9E-09		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	4E-06	4E-06		
			VINYL CHLORIDE	--	--	4E-12	4E-12	Liver cell polymorphism (H)	--	--	1E-08	1E-08		
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--		
						Chemical Total	--	--	3E-07	3E-07		--	--	7E-03
				Exposure Point Total					3E-07					7E-03
				Exposure Medium Total					3E-07					7E-03
Medium Total								3E-07					7E-03	
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	2E-04	2E-04		
			ARSENIC	--	--	3E-09	3E-09	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	6E-05	6E-05		
			CHROMIUM	--	--	--	--	--	--	--	8E-04	8E-04		
			IRON	--	--	--	--	--	--	--	4E-05	4E-05		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	CNS (N)	--	--	4E-04	4E-04		
			MERCURY	--	--	--	--	Autoimmune effects	--	--	2E-05	2E-05		
			THALLIUM	--	--	--	--	Hematological effects	--	--	2E-04	2E-04		
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	3E-05	3E-05		
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	4E-06	4E-06		
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	2E-04	2E-04		
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--		
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	5E-05	5E-05		
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--		
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--		

TABLE 9.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	1E-06	1E-06	--	--	--	--	
			BENZO(A)PYRENE	--	--	1E-05	1E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	2E-06	2E-06	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	4E-09	4E-09	Increased relative liver weight (H)	--	--	1E-04	1E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	9E-09	9E-09	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	8E-07	8E-07	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	1E-02	1E-02
			PHENANTHRENE	--	--	--	--	--	--	--	4E-04	4E-04
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	1E-09	1E-09	Liver	--	--	3E-05	3E-05
			BENZENE	--	--	2E-08	2E-08	Reduced lymphocyte count	--	--	9E-04	9E-04
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	4E-04	4E-04
	XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--		
Chemical Total			--	--	1E-05	1E-05	2E-02			2E-02		
Exposure Point Total			1E-05				2E-02					
Exposure Medium Total			1E-05				2E-02					
Medium Total			1E-05				2E-02					
Receptor Total			2E-05				Receptor HI Total 1E+03					

Total Risk Across All Media = 2E-05

Total Hazard Across All Media = 1E+03

TABLE 9.3a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	1E-08	--	2E-09	1E-08	Developmental effects	6E-04	--	1E-04	7E-04
			ALUMINUM	--	--	--	--	Neurotoxicity	1E-04	--	--	1E-04
			ARSENIC	2E-08	--	4E-09	3E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	4E-04	--	8E-05	5E-04
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-04	--	9E-05	4E-04
			CHROMIUM	--	--	--	--	--	1E-03	--	--	1E-03
			COPPER	--	--	--	--	Gastrointestinal effects	6E-05	--	--	6E-05
			IRON	--	--	--	--	Gastrointestinal effects	4E-04	--	--	4E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	5E-05	--	--	5E-05
			MERCURY	--	--	--	--	Autoimmune effects	1E-04	--	--	1E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-05	--	--	3E-05
			HIGHLY CHLORINATED PCBs	5E-09	--	4E-09	9E-09	--	9E-04	--	8E-04	2E-03
			LESS CHLORINATED PCBs	1E-10	--	1E-10	3E-10	--	8E-06	--	7E-06	2E-05
			ACENAPHTHYLENE	--	--	--	--	--	2E-06	--	1E-06	3E-06
			BENZ(A)ANTHRACENE	2E-08	--	1E-08	3E-08	--	--	--	--	--
			BENZO(A)PYRENE	1E-07	--	1E-07	2E-07	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-08	--	1E-08	3E-08	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-06	--	1E-06	3E-06
			BENZO(K)FLUORANTHENE	6E-10	--	5E-10	1E-09	--	--	--	--	--
			CHRYSENE	2E-10	--	1E-10	3E-10	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-08	--	1E-08	2E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-05	--	2E-05	5E-05
			INDENO(1,2,3-CD)PYRENE	3E-09	--	3E-09	6E-09	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-06	--	1E-06	3E-06
			PHENANTHRENE	--	--	--	--	--	9E-06	--	8E-06	2E-05
			BENZENE	2E-13	--	--	2E-13	Reduced lymphocyte count	9E-09	--	--	9E-09
			Chemical Total	2E-07	--	2E-07	4E-07		4E-03	--	1E-03	5E-03
		Exposure Point Total					4E-07					5E-03
	Exposure Medium Total						4E-07					5E-03
Medium Total							4E-07					5E-03
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	4E-06	--	4E-06
			ARSENIC	--	1E-11	--	1E-11	Development, cardiovascular, nervous system	--	4E-07	--	4E-07
			CADMIUM	--	1E-11	--	1E-11	--	--	--	--	--
			CHROMIUM	--	8E-10	--	8E-10	--	--	5E-06	--	5E-06
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E-05	--	2E-05
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-08	--	2E-08
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-13	--	2E-13	--	--	--	--	--

TABLE 9.3a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	LESS CHLORINATED PCBs	--	8E-15	--	8E-15	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	3E-14	--	3E-14	Nasal/respiratory (P)	--	2E-09	--	2E-09
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	8E-12	--	8E-12	Decreased lymphocyte count	--	3E-07	--	3E-07
	Chemical Total	--	8E-10	--	8E-10		--	3E-05	--	3E-05		
Exposure Point Total			8E-10			3E-05						
Exposure Medium Total			8E-10			3E-05						
Medium Total			8E-10			3E-05						
Ground Water	Shallow Ground Water	Exposure Unit 9	ALUMINUM	--	--	--	--	Neurotoxicity	--	--	2E-04	2E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	5E-04	5E-04
			ARSENIC	--	--	2E-08	2E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	3E-04	3E-04
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	3E-04	3E-04
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	2E-03	2E-03
			CHROMIUM	--	--	--	--	--	--	--	2E-02	2E-02
			COPPER	--	--	--	--	Gastrointestinal effects	--	--	4E-05	4E-05
			IRON	--	--	--	--	Gastrointestinal effects	--	--	4E-04	4E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	1E-03	1E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	5E-04	5E-04
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	--	--	8E-05	8E-05
			SELENIUM	--	--	--	--	Clinical selenosis	--	--	1E-05	1E-05
			THALLIUM	--	--	--	--	Hematological effects	--	--	1E-03	1E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	2E-03	2E-03
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	5E-06	5E-06
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	2E-06	2E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	3E-05	3E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	4E-06	4E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.3a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Utility Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shallow Ground Water	Exposure Unit 9	BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	4E-09	4E-09	Increased relative liver weight (H)	--	--	1E-04	1E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	2E-08	2E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	1E-03	1E-03
			PHENANTHRENE	--	--	--	--	--	--	--	2E-04	2E-04
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	5E-11	5E-11	Liver	--	--	1E-06	1E-06
			BENZENE	--	--	5E-10	5E-10	Reduced lymphocyte count	--	--	2E-05	2E-05
			Chemical Total	--	--	4E-05	4E-05		--	--	3E-02	3E-02
		Exposure Point Total					4E-05					3E-02
	Exposure Medium Total						4E-05					3E-02
Medium Total							4E-05					3E-02
Receptor Total							4E-05				Receptor HI Total	4E-02

Total Risk Across All Media = 4E-05

Total Hazard Across All Media = 4E-02

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-07	--	1E-07	3E-07	Developmental effects	9E-02	--	5E-02	1E-01
			ARSENIC	1E-07	--	5E-08	1E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	8E-03	2E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-03	--	--	1E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-03	--	1E-03	3E-03
			CHROMIUM	--	--	--	--	None Reported (O)	2E-02	--	--	2E-02
			IRON	--	--	--	--	Gastrointestinal effects	3E-02	--	--	3E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--	4E-03
			MERCURY	--	--	--	--	Autoimmune effects	3E-02	--	--	3E-02
			THALLIUM	--	--	--	--	Hematological effects	1E-02	--	--	1E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	3E-08	--	8E-08	1E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-02	--	1E-01	2E-01
			DELTA-BHC	--	--	--	--	--	--	--	--	--
			DIELDRIN	5E-09	--	--	5E-09	Hepatic (H)	4E-04	--	--	4E-04
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	--	--	--	--
			HEPTACHLOR EPOXIDE	--	--	--	--	Increased liver-to-body weight ratio in males and females (H)	--	--	--	--
			1-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	9E-01	--	2E+00	3E+00
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	1E-02	--	3E-02	4E-02
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-02	--	7E-02	1E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	2E-03	--	4E-03	6E-03
			BENZ(A)ANTHRACENE	3E-06	--	7E-06	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	7E-06	--	2E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	2E-06	4E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-03	--	3E-03	5E-03
			BENZO(K)FLUORANTHENE	4E-08	--	9E-08	1E-07	NA	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	--	--	Increased relative liver weight (H)	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	2E-08	--	5E-08	8E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	3E-06	4E-06	--	--	--	--	--

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E+00	--	2E+00	3E+00
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-02	--	6E-02	8E-02
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	3E-02	--	6E-02	9E-02
			HEXACHLOROBENZENE	3E-09	--	6E-09	9E-09	Hepatic (H)	2E-04	--	3E-04	5E-04
			INDENO(1,2,3-CD)PYRENE	4E-07	--	9E-07	1E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	5E-01	--	1E+00	2E+00
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	9E-02	--	2E-01	3E-01
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-02	--	6E-02	8E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	3E-11	--	--	3E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	6E-05	--	--	6E-05
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRICHLOROBENZENE	--	--	--	--	NA	--	--	--	--
			1,4-DICHLOROBENZENE	2E-09	--	--	2E-09	Liver	4E-04	--	--	4E-04
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	2E-02	--	--	2E-02
			BENZENE	8E-08	--	--	8E-08	Reduced lymphocyte count	3E-02	--	--	3E-02
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	1E-03	--	--	1E-03
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	2E-03	--	--	2E-03
			METHYLENE CHLORIDE	2E-10	--	--	2E-10	Liver toxicity (H)	2E-05	--	--	2E-05
			N-HEXADACANE	--	--	--	--	--	--	--	--	--
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	9E-04	--	--	9E-04
			TOLUENE	--	--	--	--	Increased kidney weight (R)	5E-03	--	--	5E-03
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	5E-03	--	--	5E-03
			Chemical Total	1E-05	--	3E-05	4E-05		3E+00	--	6E+00	9E+00
		Exposure Point Total					4E-05					9E+00
	Exposure Medium Total						4E-05					9E+00
Medium Total							4E-05					9E+00
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-06	--	5E-08	2E-06	Developmental effects	8E-01	--	3E-02	9E-01
			ALUMINUM	--	--	--	--	Neurotoxicology	1E-02	--	--	1E-02
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	3E-03	--	--	3E-03
			ARSENIC	4E-07	--	1E-08	4E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	6E-02	--	2E-03	6E-02

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	4E-03	--	--	4E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-02	--	1E-03	3E-02
			CHROMIUM	--	--	--	--	--	5E-02	--	--	5E-02
			COPPER	--	--	--	--	Gastrointestinal effects	7E-03	--	--	7E-03
			IRON	--	--	--	--	Gastrointestinal effects	3E-02	--	--	3E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--	4E-03
			MERCURY	--	--	--	--	Autoimmune effects	6E-02	--	--	6E-02
			SILVER	--	--	--	--	Argyria (In)	5E-03	--	--	5E-03
			THALLIUM	--	--	--	--	Hematological effects	3E-02	--	--	3E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	4E-03	--	--	4E-03
			HIGHLY CHLORINATED PCBs	1E-07	--	2E-08	1E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-01	--	3E-02	3E-01
			LESS CHLORINATED PCBs	1E-07	--	2E-08	2E-07	Reduced birth weights (W)	7E-02	--	1E-02	8E-02
			DIELDRIN	4E-09	--	--	4E-09	Hepatic (H)	3E-04	--	--	3E-04
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	3E-04	--	3E-05	3E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E-01	--	1E-02	1E-01
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	3E-04	--	3E-05	3E-04
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	2E-03	--	3E-04	2E-03
			ACENAPHTHYLENE	--	--	--	--	--	2E-03	--	3E-04	2E-03
			ANTHRACENE	--	--	--	--	No observed effects (O)	8E-04	--	1E-04	9E-04
			BENZ(A)ANTHRACENE	2E-06	--	2E-07	2E-06	--	--	--	--	--
			BENZO(A)PYRENE	1E-05	--	1E-06	1E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	2E-07	2E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-03	--	1E-04	1E-03
			BENZO(K)FLUORANTHENE	6E-08	--	8E-09	7E-08	--	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	1E-08	--	2E-09	2E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-07	1E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-01	--	2E-02	2E-01
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	1E-02	--	1E-03	1E-02
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	6E-03	--	6E-04	6E-03
			HEXACHLOROBENZENE	3E-08	--	3E-09	3E-08	Hepatic (H)	1E-03	--	1E-04	2E-03
			INDENO(1,2,3-CD)PYRENE	4E-07	--	5E-08	4E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	8E-02	--	1E-02	9E-02

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	PHENANTHRENE	--	--	--	--	--	2E-02	--	3E-03	3E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	1E-02	--	1E-03	1E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	1E-09	--	--	1E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	2E-03	--	--	2E-03
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	2E-03	--	--	2E-03
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	1E-08	--	--	1E-08	Liver	3E-03	--	--	3E-03
			BENZENE	1E-08	--	--	1E-08	Reduced lymphocyte count	4E-03	--	--	4E-03
			BROMOMETHANE	--	--	--	--	Epithelial hyperplasia of the forestomach	9E-04	--	--	9E-04
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	7E-04	--	--	7E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	5E-04	--	--	5E-04
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	4E-04	--	--	4E-04
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	2E-05	--	2E-06	2E-05		2E+00	--	1E-01	2E+00
	Exposure Point Total				2E-05					2E+00		
	Exposure Medium Total				2E-05					2E+00		
Medium Total				2E-05					2E+00			
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	4E-01	--	4E-01
			ANTIMONY	--	--	--	--	--	--	--	--	--
			ARSENIC	--	2E-07	--	2E-07	Development, cardiovascular, nervous system	--	6E-02	--	6E-02
			CADMIUM	--	1E-07	--	1E-07	--	--	--	--	--
			CHROMIUM	--	4E-06	--	4E-06	--	--	3E-01	--	3E-01
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E+00	--	2E+00
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	1E-02	--	1E-02
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	6E-09	--	6E-09	--	--	--	--	--
			LESS CHLORINATED PCBs	--	6E-09	--	6E-09	--	--	--	--	--
			DIELDRIN	--	2E-10	--	2E-10	--	--	--	--	--
			2,4-DIMETHYLPHENOL	--	--	--	--	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	3&4-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORANTHENE	--	--	--	--	--	--	--	--	--
			FLUORENE	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	1E-09	--	1E-09	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	1E-07	--	1E-07	Nasal/respiratory (P)	--	8E-02	--	8E-02
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			PYRENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	5E-07	--	5E-07	Liver	--	4E-03	--	4E-03
			BENZENE	--	1E-07	--	1E-07	Decreased lymphocyte count	--	4E-02	--	4E-02
			BROMOMETHANE	--	--	--	--	Nasal lesions and membrane degeneration	--	3E-02	--	3E-02
			TOLUENE	--	--	--	--	Neurological effects	--	4E-04	--	4E-04
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	7E-01	--	7E-01
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	--	5E-06	--	5E-06	--	--	3E+00	--	3E+00
		Exposure Point Total										
	Exposure Medium Total											
Medium Total												
Ground Water	Shallow Ground Water	Exposure Unit 1	ALUMINUM	--	--	--	--	Neurotoxicology	--	--	5E-04	5E-04
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	5E-03	5E-03
			ARSENIC	--	--	2E-08	2E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	3E-03	3E-03
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	3E-02	3E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	1E-02	1E-02
			CHROMIUM	--	--	--	--	--	--	--	6E-02	6E-02
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	--	--	2E-04	2E-04

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shallow Ground Water	Exposure Unit 1	IRON	--	--	--	--	Gastrointestinal effects	--	--	2E-03	2E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	3E-02	3E-02
			MERCURY	--	--	--	--	Autoimmune effects	--	--	8E-03	8E-03
			SILVER	--	--	--	--	Argyria (In)	--	--	1E-03	1E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	6E-03	6E-03
			HIGHLY CHLORINATED PCBs	--	--	--	--	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	--	--	--	--
			4,4'-DDT	--	--	7E-09	7E-09	Liver lesions (H)	--	--	3E-03	3E-03
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	--	--	--	--
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	--	--	1E-05	1E-05
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	7E-05	7E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	--	--	4E-05	4E-05
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	1E-04	1E-04
			4-METHYLPHENOL	--	--	--	--	--	--	--	6E-05	6E-05
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			ANTHRACENE	--	--	--	--	No observed effects (O)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	3E-08	3E-08	--	--	--	--	--
			BENZO(A)PYRENE	--	--	5E-07	5E-07	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	4E-08	4E-08	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	4E-11	4E-11	Increased relative liver weight (H)	--	--	1E-05	1E-05
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	3E-10	3E-10	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	2E-07	2E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	--	--	3E-04	3E-04
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			HEXACHLOROBUTADIENE	--	--	2E-11	2E-11	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	5E-08	5E-08	--	--	--	--	--

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shallow Ground Water	Exposure Unit 1	NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	2E-03	2E-03
			PHENANTHRENE	--	--	--	--	--	--	--	6E-04	6E-04
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	--	--	7E-06	7E-06
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	3E-11	3E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	--	--	6E-05	6E-05
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	--	--	7E-05	7E-05
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	4E-10	4E-10	Liver	--	--	7E-05	7E-05
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	--	--	--	--
			BENZENE	--	--	2E-09	2E-09	Reduced lymphocyte count	--	--	5E-04	5E-04
			BROMODICHLOROMETHANE	--	--	4E-13	4E-13	Renal cytomegaly (R)	--	--	2E-08	2E-08
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	--	--	4E-05	4E-05
			CHLOROFORM	--	--	--	--	Moderate/marked fatty cyst formation in the liver and elevated SGPT	--	--	2E-07	2E-07
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	--	--	6E-06	6E-06
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	--	--	--	--
			METHYLENE CHLORIDE	--	--	1E-12	1E-12	Liver toxicity (H)	--	--	2E-07	2E-07
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	--	--	7E-06	7E-06
			TETRACHLOROETHENE	--	--	2E-11	2E-11	Hepatotoxicity in mice (H), weight gain in rats	--	--	2E-07	2E-07
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	9E-05	9E-05
			VINYL CHLORIDE	--	--	1E-11	1E-11	Liver cell polymorphism (H)	--	--	4E-07	4E-07
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total	--	--	9E-07	9E-07		--	--	2E-01	2E-01
		Exposure Point Total					9E-07					2E-01
	Exposure Medium Total						9E-07					2E-01
Medium Total							9E-07					2E-01

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	4E-03	4E-03
			ARSENIC	--	--	9E-09	9E-09	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	1E-03	1E-03
			CHROMIUM	--	--	--	--	--	--	--	2E-02	2E-02
			IRON	--	--	--	--	Gastrointestinal effects	--	--	1E-03	1E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	9E-03	9E-03
			MERCURY	--	--	--	--	Autoimmune effects	--	--	6E-04	6E-04
			THALLIUM	--	--	--	--	Hematological effects	--	--	6E-03	6E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	9E-04	9E-04
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	9E-05	9E-05
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	4E-03	4E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	1E-03	1E-03
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	3E-06	3E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	3E-05	3E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	4E-06	4E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	1E-08	1E-08	Increased relative liver weight (H)	--	--	3E-03	3E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	3E-08	3E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	2E-06	2E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	3E-01	3E-01
			PHENANTHRENE	--	--	--	--	--	--	--	1E-02	1E-02
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	3E-09	3E-09	Liver	--	--	6E-04	6E-04
			BENZENE	--	--	7E-08	7E-08	Reduced lymphocyte count	--	--	2E-02	2E-02
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--

TABLE 9.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	1E-02	1E-02
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
			Chemical Total	--	--	4E-05	4E-05		--	--	4E-01	4E-01
		Exposure Point Total					4E-05					4E-01
	Exposure Medium Total						4E-05					4E-01
Medium Total							4E-05					4E-01
Receptor Total							1E-04					Receptor HI Total 1E+01

Total Risk Across All Media = 1E-04

Total Hazard Across All Media = 1E+01

Total Liver HI Across All Media =	2E-01
Total Kidney HI Across All Media =	6E-01
Total Nervous System Effects HI Across All Media =	2E+00
Total Lymphocyte Effects HI Across All Media =	9E-02
Total Nasal/Respiratory Effects HI Across All Media =	3E+00
Total Ocular Effects HI Across All Media =	5E-01
Total Other Effects HI Across All Media =	8E+00

TABLE 9.4a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	1E-07	--	3E-09	1E-07	Developmental effects	5E-02	--	2E-03	5E-02
			ALUMINUM	--	--	--	--	Neurotoxicology	9E-03	--	--	9E-03
			ARSENIC	2E-07	--	6E-09	2E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	1E-03	3E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-02	--	1E-03	3E-02
			CHROMIUM	--	--	--	--	--	8E-02	--	--	8E-02
			COPPER	--	--	--	--	Gastrointestinal effects	5E-03	--	--	5E-03
			IRON	--	--	--	--	Gastrointestinal effects	3E-02	--	--	3E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--	4E-03
			MERCURY	--	--	--	--	Autoimmune effects	1E-02	--	--	1E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-03	--	--	3E-03
			HIGHLY CHLORINATED PCBs	4E-08	--	6E-09	5E-08	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	8E-02	--	1E-02	9E-02
			LESS CHLORINATED PCBs	1E-09	--	2E-10	2E-09	Reduced birth weights (W)	7E-04	--	9E-05	8E-04
			ACENAPHTHYLENE	--	--	--	--	--	1E-04	--	2E-05	1E-04
			BENZ(A)ANTHRACENE	1E-07	--	2E-08	2E-07	--	--	--	--	--
			BENZO(A)PYRENE	1E-06	--	1E-07	1E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-07	--	2E-08	2E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-04	--	2E-05	1E-04
			BENZO(K)FLUORANTHENE	5E-09	--	7E-10	6E-09	--	--	--	--	--
			CHRYSENE	2E-09	--	2E-10	2E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-07	--	1E-08	1E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	2E-04	3E-03
			INDENO(1,2,3-CD)PYRENE	3E-08	--	4E-09	4E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E-04	--	2E-05	2E-04
			PHENANTHRENE	--	--	--	--	--	8E-04	--	1E-04	9E-04
			BENZENE	2E-12	--	--	2E-12	Reduced lymphocyte count	7E-07	--	--	7E-07
			Chemical Total	2E-06	--	2E-07	2E-06		3E-01	--	1E-02	3E-01
		Exposure Point Total					2E-06					3E-01
	Exposure Medium Total						2E-06					3E-01
Medium Total							2E-06					3E-01
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E-04	--	1E-04
			ARSENIC	--	4E-11	--	4E-11	Development, cardiovascular, nervous system	--	1E-05	--	1E-05
			CADMIUM	--	4E-11	--	4E-11	--	--	--	--	--
			CHROMIUM	--	2E-09	--	2E-09	--	--	1E-04	--	1E-04

TABLE 9.4a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	COPPER	--	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--		
			LEAD	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	6E-04	--	6E-04	
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	6E-07	--	6E-07	
			VANADIUM	--	--	--	--	--	--	--	--		
			HIGHLY CHLORINATED PCBs	--	7E-13	--	7E-13	--	--	--	--		
			LESS CHLORINATED PCBs	--	2E-14	--	2E-14	--	--	--	--		
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--		
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--		
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--		
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--		
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--		
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--		
			CHRYSENE	--	--	--	--	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--		
			DIBENZOFURAN	--	--	--	--	--	--	--	--		
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--		
			NAPHTHALENE	--	8E-14	--	8E-14	Nasal/respiratory (P)	--	6E-08	--	6E-08	
			PHENANTHRENE	--	--	--	--	--	--	--	--		
			BENZENE	--	2E-11	--	2E-11	Decreased lymphocyte count	--	7E-06	--	7E-06	
			Chemical Total	--	3E-09	--	3E-09		--	9E-04	--	9E-04	
		Exposure Point Total							3E-09				
	Exposure Medium Total							3E-09					9E-04
Medium Total								3E-09					9E-04
Ground Water	Shallow Ground Water	Exposure Unit 9	ALUMINUM	--	--	--	--	Neurotoxicology	--	--	5E-03	5E-03	
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	1E-02	1E-02	
			ARSENIC	--	--	5E-08	5E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	8E-03	8E-03	
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	--	--	8E-03	8E-03	
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	--	--	6E-02	6E-02	
			CHROMIUM	--	--	--	--	--	--	--	6E-01	6E-01	
			COPPER	--	--	--	--	Gastrointestinal effects	--	--	1E-03	1E-03	
			IRON	--	--	--	--	Gastrointestinal effects	--	--	9E-03	9E-03	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	CNS (N)	--	--	3E-02	3E-02	
			MERCURY	--	--	--	--	Autoimmune effects	--	--	1E-02	1E-02	
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	--	--	2E-03	2E-03	
			SELENIUM	--	--	--	--	Clinical selenosis	--	--	3E-04	3E-04	
			THALLIUM	--	--	--	--	Hematological effects	--	--	4E-02	4E-02	
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	4E-02	4E-02	
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	1E-04	1E-04	
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--	

TABLE 9.4a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shallow Ground Water	Exposure Unit 9	ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--		--	--	--	--
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	4E-06	4E-06		--	--	--	--
			BENZO(A)PYRENE	--	--	8E-05	8E-05		--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	1E-05	1E-05		--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--		--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--		--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	1E-08	1E-08	Increased relative liver weight (H)	--	--	3E-03	3E-03
			CARBAZOLE	--	--	--	--		--	--	--	--
			CHRYSENE	--	--	4E-08	4E-08		--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	3E-02	3E-02
			PHENANTHRENE	--	--	--	--		--	--	4E-03	4E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	1E-10	1E-10	Liver	--	--	3E-05	3E-05
			BENZENE	--	--	1E-09	1E-09	Reduced lymphocyte count	--	--	4E-04	4E-04
				--	--	1E-04		--	--	8E-01	8E-01	
		Exposure Point Total				1E-04				8E-01		
		Exposure Medium Total				1E-04				8E-01		
Medium Total						1E-04				8E-01		
Receptor Total						1E-04				Receptor HI Total	1E+00	

Total Risk Across All Media = 1E-04

Total Hazard Across All Media = 1E+00

TABLE 9.5 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Surveillance Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 2	2,3,7,8-TCDD Equivalent	1E-06	--	9E-09	1E-06	--	6E-02	--	5E-04	6E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	1E-03	--	--	1E-03
			ARSENIC	3E-07	--	2E-09	3E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	4E-03	--	3E-05	4E-03
			BARIIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney	3E-04	--	--	3E-04
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	6E-03	--	6E-05	7E-03
			CHROMIUM	--	--	--	--	--	6E-03	--	--	6E-03
			COPPER	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03
			IRON	--	--	--	--	Gastrointestinal effects	3E-03	--	--	3E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	3E-04	--	--	3E-04
			MERCURY	--	--	--	--	Autoimmune effects	7E-03	--	--	7E-03
			SILVER	--	--	--	--	Argyria (In)	6E-04	--	--	6E-04
			THALLIUM	--	--	--	--	Hematological effects	1E-03	--	--	1E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-04	--	--	3E-04
			HIGHLY CHLORINATED PCBs	6E-08	--	2E-09	6E-08	--	1E-02	--	4E-04	1E-02
			LESS CHLORINATED PCBs	3E-08	--	9E-10	3E-08	--	1E-03	--	5E-05	2E-03
			DIELDRIN	6E-08	--	--	6E-08	--	6E-04	--	--	6E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Hepatic (H)	6E-04	--	--	6E-04
			ACENAPHTHYLENE	--	--	--	--	Pulmonary alveolar proteinosis	6E-04	--	2E-05	6E-04
			BENZ(A)ANTHRACENE	2E-08	--	7E-10	2E-08	--	2E-05	--	6E-07	2E-05
			BENZO(A)PYRENE	2E-07	--	8E-09	2E-07	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-08	--	1E-09	3E-08	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	6E-06	--	2E-07	6E-06
			BENZO(K)FLUORANTHENE	2E-09	--	5E-11	2E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	5E-08	--	2E-09	5E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E-04	--	1E-05	4E-04
			HEXACHLOROBENZENE	3E-08	--	7E-10	3E-08	Hepatic (H)	2E-04	--	4E-06	2E-04
			INDENO(1,2,3-CD)PYRENE	1E-08	--	5E-10	1E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-04	--	6E-06	2E-04
			PHENANTHRENE	--	--	--	--	--	6E-05	--	2E-06	6E-05
			1,2,3TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	3E-10	--	--	3E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	7E-05	--	--	7E-05
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	3E-05	--	--	3E-05
			1,4-DICHLOROBENZENE	4E-09	--	--	4E-09	Liver	7E-05	--	--	7E-05
			BENZENE	7E-10	--	--	7E-10	Reduced lymphocyte count	2E-05	--	--	2E-05
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			Chemical Total	2E-06	--	3E-08	2E-06		1E-01	--	1E-03	1E-01
		Exposure Point Total					2E-06					1E-01
	Exposure Medium Total						2E-06					1E-01
Medium Total							2E-06					1E-01

TABLE 9.5 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Surveillance Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Surface Soil	Outdoor Air	Exposure Unit 2	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--			
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	--	--	2E-06			
			ARSENIC	--	7E-12	--	7E-12	Development, cardiovascular, nervous system	--	2E-06	--	3E-07			
			BARIUM	--	--	--	--	Renal toxicity	--	3E-07	--	1E-06			
			CADMIUM	--	2E-11	--	2E-11	--	--	--	--	--			
			CHROMIUM	--	3E-10	--	3E-10	--	--	1E-06	--	2E-06			
			COPPER	--	--	--	--	--	--	--	--	--			
			IRON	--	--	--	--	--	--	--	--	--			
			LEAD	--	--	--	--	--	--	--	--	--			
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	--	--	--			
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	1E-05	--	1E-05			
			SILVER	--	--	--	--	--	--	7E-08	--	7E-08			
			THALLIUM	--	--	--	--	--	--	--	--	--			
			VANADIUM	--	--	--	--	--	--	--	--	--			
			HIGHLY CHLORINATED PCBs	--	2E-13	--	2E-13	--	--	--	--	--			
			LESS CHLORINATED PCBs	--	8E-14	--	8E-14	--	--	--	--	--			
			DIELDRIN	--	2E-13	--	2E-13	--	--	--	--	--			
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--			
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--			
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--			
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--			
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--			
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--			
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--			
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--			
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--			
			HEXACHLOROBENZENE	--	9E-14	--	9E-14	--	--	--	--	--			
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--			
			NAPHTHALENE	--	2E-13	--	2E-13	Nasal/respiratory (P)	--	--	--	1E-08			
			PHENANTHRENE	--	--	--	--	--	--	--	--	--			
			1,2,3TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--			
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--			
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	7E-05	--	7E-05			
			1,4-DICHLOROBENZENE	--	3E-08	--	3E-08	Liver	--	3E-05	--	3E-05			
			BENZENE	--	2E-09	--	2E-09	Decreased lymphocyte count	--	6E-05	--	6E-05			
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--			
					Chemical Total	--	3E-08	--	3E-08		--	2E-04	--	2E-04	
					Exposure Point Total					3E-08					2E-04
					Exposure Medium Total					3E-08					2E-04
					Medium Total					3E-08					2E-04
			Receptor Total					2E-06					1E-01		

Total Risk Across All Media = 2E-06

Total Hazard Across All Media = 1E-01

TABLE 9.6 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Ditch Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Sediment	Outdoor Air	Exposure Unit 3	BENZENE	--	2E-09	--	2E-09	Decreased lymphocyte count	--	6E-05	--	6E-05
			Chemical Total	--	2E-09	--	2E-09		--	6E-05	--	6E-05
			Exposure Point Total				2E-09					6E-05
		Exposure Medium Total				2E-09					6E-05	
Medium Total							2E-09					6E-05
Sediment	Surface Sediment	Exposure Unit 3	2,3,7,8-TCDD Equivalent	1E-08	--	7E-10	1E-08	--	6E-04	--	4E-05	6E-04
			ARSENIC	5E-08	--	3E-09	5E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	8E-04	--	5E-05	9E-04
			CHROMIUM	--	--	--	--	--	3E-03	--	--	3E-03
			IRON	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03
			MANGANESE	--	--	--	--	CNS (N)	1E-04	--	--	1E-04
			MERCURY	--	--	--	--	Autoimmune effects	2E-04	--	--	2E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-04	--	--	1E-04
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E-04	--	7E-05	3E-04
			ACENAPHTHYLENE	--	--	--	--	--	8E-06	--	2E-06	1E-05
			BENZ(A)ANTHRACENE	8E-09	--	2E-09	1E-08	--	--	--	--	--
			BENZO(A)PYRENE	8E-08	--	2E-08	1E-07	--	--	--	--	--
			BENZO(B)FLUORANTHENE	9E-09	--	2E-09	1E-08	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	3E-06	--	7E-07	3E-06
			DIBENZ(A,H)ANTHRACENE	6E-09	--	2E-09	7E-09	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E-04	--	5E-05	3E-04
			INDENO(1,2,3-CD)PYRENE	6E-09	--	2E-09	7E-09	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-04	--	5E-05	2E-04
			PHENANTHRENE	--	--	--	--	--	2E-05	--	6E-06	3E-05
			BENZENE	4E-10	--	--	4E-10	Reduced lymphocyte count	1E-05	--	--	1E-05
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
				Chemical Total	2E-07	--	3E-08	2E-07		7E-03	--	3E-04
			Exposure Point Total				2E-07					8E-03
			Exposure Medium Total				2E-07					8E-03
Medium Total							2E-07				8E-03	
Surface Water	Surface Water	Exposure Unit 3	CHROMIUM	--	--	--	--	--	--	--	2E-03	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	1E-05	1E-05
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	--	--	1E-07	1E-07
			MERCURY	--	--	--	--	Autoimmune effects	--	--	6E-05	6E-05
			VANADIUM	--	--	--	--	Decreased hair cystine	--	--	8E-05	8E-05
			ZINC	--	--	--	--	Decreased ESOD (B)	--	--	1E-05	1E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	1E-04	1E-04
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.6 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Ditch Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	1E-02	1E-02
			PHENANTHRENE	--	--	--	--	--	--	--	4E-04	4E-04
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	--	4E-08	4E-08	Reduced lymphocyte count	--	--	1E-03	1E-03
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	3E-04	3E-04
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
	Chemical Total	--	--	--	4E-08		--	--	1E-02	1E-02		
		Exposure Point Total				4E-08				1E-02		
	Exposure Medium Total				4E-08					1E-02		
Medium Total						4E-08				1E-02		
Receptor Total						2E-07		Receptor HI Total			2E-02	

Total Risk Across All Media = 2E-07

Total Hazard Across All Media = 2E-02

TABLE 9.7 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Surface Soil	Outdoor Air	Exposure Unit 4	ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-04	--	2E-04			
			ARSENIC	--	6E-10	--	6E-10	Development, cardiovascular, nervous system	--	2E-05	--	2E-05			
			BARIUM	--	--	--	--	Renal toxicity	--	5E-05	--	5E-05			
			CHROMIUM	--	2E-09	--	2E-09	--	--	2E-05	--	2E-05			
			IRON	--	--	--	--	--	--	--	--	--			
			LEAD	--	--	--	--	--	--	--	--	--			
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	6E-04	--	6E-04			
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	3E-07	--	3E-07			
			VANADIUM	--	--	--	--	--	--	--	--	--			
			HIGHLY CHLORINATED PCBs	--	4E-13	--	4E-13	--	--	--	--	--			
			LESS CHLORINATED PCBs	--	2E-14	--	2E-14	--	--	--	--	--			
			DIELDRIN	--	2E-12	--	2E-12	--	--	--	--	--			
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--			
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--			
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--			
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--			
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--			
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--			
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--			
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--			
			PHENANTHRENE	--	--	--	--	--	--	--	--	--			
			BENZENE	--	3E-11	--	3E-11	Decreased lymphocyte count	--	1E-06	--	1E-06			
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--			
			Chemical Total			--	3E-09	--	3E-09				--	9E-04	9E-04
			Exposure Point Total							3E-09					9E-04
	Exposure Medium Total							3E-09					9E-04		
Medium Total							3E-09					9E-04			
Soil	Surface Soil	Exposure Unit 4	ALUMINUM	--	--	--	--	Neurotoxicity	6E-03	--	--	6E-03			
			ARSENIC	2E-06	--	1E-07	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	2E-03	3E-02			
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	9E-04	--	--	9E-04			
			CHROMIUM	--	--	--	--	--	4E-03	--	--	4E-03			
			IRON	--	--	--	--	Gastrointestinal effects	2E-02	--	--	2E-02			
			LEAD	--	--	--	--	--	--	--	--	--			
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03			
			MERCURY	--	--	--	--	Autoimmune effects	2E-03	--	--	2E-03			
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-03	--	--	1E-03			
			HIGHLY CHLORINATED PCBs	1E-08	--	3E-09	1E-08	--	2E-03	--	6E-04	2E-03			
			LESS CHLORINATED PCBs	5E-10	--	2E-10	7E-10	--	3E-05	--	9E-06	4E-05			
			DIELDRIN	7E-08	--	--	7E-08	Hepatic (H)	6E-04	--	--	6E-04			
			ACENAPHTHYLENE	--	--	--	--	--	4E-06	--	1E-06	5E-06			
			BENZ(A)ANTHRACENE	2E-08	--	7E-09	3E-08	--	--	--	--	--			
			BENZO(A)PYRENE	2E-07	--	7E-08	3E-07	--	--	--	--	--			

TABLE 9.7 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
			BENZO(B)FLUORANTHENE	3E-08	--	9E-09	4E-08	--	--	--	--	--
Soil	Surface Soil	Exposure Unit 4	BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-06	--	2E-06	7E-06
			BENZO(K)FLUORANTHENE	2E-09	--	6E-10	2E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	8E-08	--	2E-08	1E-07	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	2E-08	--	5E-09	2E-08	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	1E-05	--	3E-06	1E-05
			BENZENE	5E-12	--	--	5E-12	Reduced lymphocyte count	2E-07	--	--	2E-07
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			Chemical Total	2E-06	--	2E-07	2E-06	--	6E-02	--	3E-03	7E-02
		Exposure Point Total					2E-06					7E-02
	Exposure Medium Total						2E-06					7E-02
Medium Total							2E-06					7E-02
Receptor Total							2E-06					7E-02
											Receptor HI Total	7E-02

Total Risk Across All Media = 2E-06

Total Hazard Across All Media = 7E-02

TABLE 9.7a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	3E-05	--	3E-05
			ARSENIC	--	1E-10	--	1E-10	Development, cardiovascular, nervous system	--	4E-06	--	4E-06
			CADMIUM	--	1E-10	--	1E-10	--	--	--	--	--
			CHROMIUM	--	6E-09	--	6E-09	--	--	4E-05	--	4E-05
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E-04	--	2E-04
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-07	--	2E-07
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-12	--	2E-12	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	3E-11	--	3E-11	Decreased lymphocyte count	--	1E-06	--	1E-06
			Chemical Total	--	6E-09	--	6E-09		--	3E-04	--	3E-04
		Exposure Point Total			6E-09				3E-04			
	Exposure Medium Total			6E-09				3E-04				
Medium Total			6E-09				3E-04					
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	4E-07	--	3E-08	4E-07	--	2E-02	--	1E-03	2E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	3E-03	--	--	3E-03
			ARSENIC	8E-07	--	5E-08	8E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	9E-04	1E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-02	--	1E-03	1E-02
			CHROMIUM	--	--	--	--	--	3E-02	--	--	3E-02
			COPPER	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	4E-03	--	--	4E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-03	--	--	1E-03
			HIGHLY CHLORINATED PCBs	2E-07	--	5E-08	2E-07	--	3E-02	--	1E-02	4E-02
			ACENAPHTHYLENE	--	--	--	--	--	4E-05	--	1E-05	6E-05
			BENZ(A)ANTHRACENE	6E-07	--	2E-07	7E-07	--	--	--	--	--

TABLE 9.7a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	BENZO(A)PYRENE	4E-06	--	1E-06	5E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-07	--	2E-07	7E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-05	--	1E-05	6E-05
			BENZO(K)FLUORANTHENE	2E-08	--	6E-09	3E-08	--	--	--	--	--
			CHRYSENE	6E-09	--	2E-09	7E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-07	--	1E-07	5E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-03	--	2E-04	1E-03
			INDENO(1,2,3-CD)PYRENE	1E-07	--	3E-08	1E-07	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	3E-04	--	9E-05	4E-04
			BENZENE	4E-12	--	--	4E-12	Reduced lymphocyte count	2E-07	--	--	2E-07
			Chemical Total	7E-06	--	2E-06	9E-06		1E-01	--	1E-02	1E-01
		Exposure Point Total					9E-06					1E-01
	Exposure Medium Total						9E-06					1E-01
Medium Total							9E-06					1E-01
Receptor Total							9E-06				Receptor HI Total	1E-01

Total Risk Across All Media = 9E-06

Total Hazard Across All Media = 1E-01

TABLE 9.8 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Surface Soil	Outdoor Air	Exposure Unit 5	ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	9E-04	--	9E-04			
			ANTIMONY	--	--	--	--	--	--	--	--	--			
			ARSENIC	--	5E-09	--	5E-09	Development, cardiovascular, nervous system	--	2E-04	--	2E-04			
			CHROMIUM	--	4E-08	--	4E-08	--	--	2E-04	--	2E-04			
			IRON	--	--	--	--	--	--	--	--	--			
			LEAD	--	--	--	--	--	--	--	--	--			
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	4E-03	--	4E-03			
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	5E-06	--	5E-06			
			THALLIUM	--	--	--	--	--	--	--	--	--			
			VANADIUM	--	--	--	--	--	--	--	--	--			
			HIGHLY CHLORINATED PCBs	--	3E-10	--	3E-10	--	--	--	--	--			
			ENDOSULFAN SULFATE	--	--	--	--	--	--	--	--	--			
			ENDRIN ALDEHYDE	--	--	--	--	--	--	--	--	--			
			3&4-METHYLPHENOL	--	--	--	--	--	--	--	--	--			
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--			
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--			
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--			
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--			
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--			
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--			
			CHRYSENE	--	--	--	--	--	--	--	--	--			
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--			
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--			
			FLUORANTHENE	--	--	--	--	--	--	--	--	--			
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--			
			NAPHTHALENE	--	2E-11	--	2E-11	Nasal/respiratory (P)	--	2E-06	--	2E-06			
			PHENANTHRENE	--	--	--	--	--	--	--	--	--			
			BENZENE	--	1E-09	--	1E-09	Decreased lymphocyte count	--	5E-05	--	5E-05			
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--			
			Chemical Total			--	4E-08	--	4E-08			--	5E-03	--	5E-03
			Exposure Point Total							4E-08					5E-03
			Exposure Medium Total							4E-08					5E-03
	Medium Total							4E-08					5E-03		
Soil	Surface Soil	Exposure Unit 5	ALUMINUM	--	--	--	--	Neurotoxicology	3E-03	--	--	3E-03			
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	2E-03	--	--	2E-03			
			ARSENIC	1E-06	--	2E-07	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	4E-03	3E-02			
			CHROMIUM	--	--	--	--	--	5E-03	--	--	5E-03			
			IRON	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02			
			LEAD	--	--	--	--	--	--	--	--	--			
			MANGANESE	--	--	--	--	CNS (N)	1E-03	--	--	1E-03			
			MERCURY	--	--	--	--	Autoimmune effects	3E-03	--	--	3E-03			
			THALLIUM	--	--	--	--	Hematological effects	5E-03	--	--	5E-03			
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-03	--	--	1E-03			
			HIGHLY CHLORINATED PCBs	7E-07	--	6E-07	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	1E-01	2E-01			

TABLE 9.8 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 5	ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	9E-06	--	--	9E-06
			ENDRIN ALDEHYDE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	1E-04	--	--	1E-04
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	4E-07	--	2E-07	6E-07
			ACENAPHTHYLENE	--	--	--	--	--	2E-04	--	2E-04	4E-04
			BENZ(A)ANTHRACENE	1E-06	--	1E-06	3E-06	--	--	--	--	--
			BENZO(A)PYRENE	1E-05	--	1E-05	3E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	4E-04	--	4E-04	8E-04
			BENZO(K)FLUORANTHENE	2E-07	--	1E-07	3E-07	--	--	--	--	--
			CHRYSENE	1E-08	--	1E-08	3E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-06	--	3E-06	7E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-03	--	2E-03	4E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	8E-04	--	7E-04	2E-03
			INDENO(1,2,3-CD)PYRENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	2E-04	--	1E-04	3E-04
			PHENANTHRENE	--	--	--	--	--	6E-04	--	5E-04	1E-03
			BENZENE	2E-11	--	--	2E-11	Reduced lymphocyte count	9E-07	--	--	9E-07
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			Chemical Total	2E-05	--	2E-05	4E-05		2E-01	--	1E-01	3E-01
		Exposure Point Total					4E-05					3E-01
	Exposure Medium Total						4E-05					3E-01
Medium Total							4E-05					3E-01
Receptor Total							4E-05				Receptor HI Total	3E-01

Total Risk Across All Media = 4E-05

Total Hazard Across All Media = 3E-01

TABLE 9.9 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 7	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	3E-04	--	3E-04
			ANTIMONY	--	--	--	--	--	--	--	--	--
			ARSENIC	--	1E-09	--	1E-09	Development, cardiovascular, nervous system	--	4E-05	--	4E-05
			BARIUM	--	--	--	--	Renal toxicity	--	1E-04	--	1E-04
			CADMIUM	--	1E-09	--	1E-09	--	--	--	--	--
			CHROMIUM	--	3E-08	--	3E-08	--	--	2E-04	--	2E-04
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-03	--	1E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	7E-06	--	7E-06
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-11	--	2E-11	--	--	--	--	--
			LESS CHLORINATED PCBs	--	1E-11	--	1E-11	--	--	--	--	--
			DIELDRIN	--	1E-11	--	1E-11	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			FLUORANTHENE	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	1E-11	--	1E-11	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	2E-11	--	2E-11	Nasal/respiratory (P)	--	1E-06	--	1E-06
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	2E-03	--	2E-03
			1,4-DICHLOROBENZENE	--	2E-06	--	2E-06	Liver	--	1E-03	--	1E-03
			BENZENE	--	9E-08	--	9E-08	Decreased lymphocyte count	--	3E-03	--	3E-03
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
						Chemical Total	--	2E-06	--	2E-06		
				Exposure Point Total				2E-06				8E-03
			Exposure Medium Total				2E-06				8E-03	
			Medium Total				2E-06				8E-03	

TABLE 9.9 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 7	2,3,7,8-TCDD Equivalent	4E-06	--	8E-07	5E-06	Developmental effects	2E-01	--	4E-02	3E-01
			ALUMINUM	--	--	--	--	Neurotoxicology	3E-03	--	--	3E-03
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol	8E-04	--	--	8E-04
			ARSENIC	8E-07	--	2E-07	9E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	3E-03	2E-02
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	6E-04	--	--	6E-04
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-02	--	3E-03	2E-02
			CHROMIUM	--	--	--	--	--	1E-02	--	--	1E-02
			COPPER	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	8E-03	--	--	8E-03
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	9E-04	--	--	9E-04
			MERCURY	--	--	--	--	Autoimmune effects	1E-02	--	--	1E-02
			SILVER	--	--	--	--	Argyria (In)	1E-03	--	--	1E-03
			THALLIUM	--	--	--	--	Hematological effects	4E-03	--	--	4E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-03	--	--	1E-03
			HIGHLY CHLORINATED PCBs	2E-07	--	1E-07	3E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-02	--	3E-02	6E-02
			LESS CHLORINATED PCBs	8E-08	--	7E-08	2E-07	Reduced birth weights (W)	4E-03	--	4E-03	8E-03
			DIELDRIN	1E-07	--	--	1E-07	Hepatic (H)	1E-03	--	--	1E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E-03	--	9E-04	2E-03
			ACENAPHTHYLENE	--	--	--	--	--	9E-05	--	8E-05	2E-04
			BENZ(A)ANTHRACENE	7E-07	--	6E-07	1E-06	--	--	--	--	--
			BENZO(A)PYRENE	7E-06	--	6E-06	1E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	5E-07	--	4E-07	9E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-04	--	1E-04	3E-04
			BENZO(K)FLUORANTHENE	6E-08	--	5E-08	1E-07	--	--	--	--	--
			CHRYSENE	7E-09	--	6E-09	1E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			DIBENZOFURAN	1E-06	--	--	1E-06	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	1E-03	3E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	4E-04	--	4E-04	8E-04
			HEXACHLOROBENZENE	7E-08	--	5E-08	1E-07	Hepatic (H)	4E-04	--	3E-04	7E-04
			INDENO(1,2,3-CD)PYRENE	4E-07	--	4E-07	8E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	4E-04	--	3E-04	7E-04
			PHENANTHRENE	--	--	--	--	--	4E-04	--	3E-04	7E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	6E-10	--	--	6E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1E-04	--	--	1E-04
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	3E-05	--	--	3E-05

TABLE 9.9 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Soil	Surface Soil	Exposure Unit 7	1,4-DICHLOROBENZENE	7E-09	--	--	7E-09	Liver	1E-04	--	--	1E-04		
			BENZENE	1E-09	--	--	1E-09	Reduced lymphocyte count	4E-05	--	--	4E-05		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--			
			DODECANE	--	--	--	--	--	--	--	--			
			Chemical Total	2E-05	--	1E-05	3E-05		3E-01	--	9E-02	4E-01		
		Exposure Point Total									4E-01			
	Exposure Medium Total											4E-01		
Medium Total												4E-01		
Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicology	4E-01	--	--	4E-01		
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol	9E-02	--	--	9E-02		
			ARSENIC	3E-05	--	--	3E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-01	--	--	5E-01		
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-01	--	--	1E-01		
			BERYLLIUM	--	--	--	--	Small intestinal lesions	7E-03	--	--	7E-03		
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	3E-02	--	--	3E-02		
			CHROMIUM	--	--	--	--	--	4E-01	--	--	4E-01		
			COBALT	--	--	--	--	--	--	--	--	--		
			COPPER	--	--	--	--	Gastrointestinal effects	4E-02	--	--	4E-02		
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	3E-02	--	--	3E-02		
			IRON	--	--	--	--	Gastrointestinal effects	1E+00	--	--	1E+00		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	CNS (N)	2E-01	--	--	2E-01		
			MERCURY	--	--	--	--	Autoimmune effects	1E-01	--	--	1E-01		
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	4E-02	--	--	4E-02		
			SELENIUM	--	--	--	--	Clinical selenosis	1E-02	--	--	1E-02		
			SILVER	--	--	--	--	Argyria (In)	7E-03	--	--	7E-03		
			THALLIUM	--	--	--	--	Hematological effects	1E+00	--	--	1E+00		
			VANADIUM	--	--	--	--	Decreased hair cystine	8E-02	--	--	8E-02		
			ZINC	--	--	--	--	Decreased ESOD (B)	6E-03	--	--	6E-03		
					HIGHLY CHLORINATED PCBs	3E-07	--	--	3E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-02	--	--	6E-02
					4,4'-DDD	5E-08	--	--	5E-08	--	--	--	--	
					4,4'-DDT	8E-07	--	--	8E-07	Liver lesions (H)	4E-02	--	--	4E-02
					ALDRIN	1E-06	--	--	1E-06	Liver toxicity (H)	2E-02	--	--	2E-02

TABLE 9.9 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Water	Potable Water	Exposure Unit 8	ALPHA-BHC	3E-06	--	--	3E-06	--	--	--	--	--
			ENDOSULFAN II	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	2E-04	--	--	2E-04
			ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	6E-05	--	--	6E-05
			HEPTACHLOR EPOXIDE	2E-07	--	--	2E-07	Increased liver-to-body weight ratio in males and females (H)	1E-02	--	--	1E-02
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	4E-03	--	--	4E-03
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response	6E-02	--	--	6E-02
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	3E+00	--	--	3E+00
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E+00	--	--	3E+00
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	3E-01	--	--	3E-01
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	1E+00	--	--	1E+00
			4-CHLORO-3-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			4-METHYLPHENOL	--	--	--	--	--	3E+00	--	--	3E+00
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	3E-02	--	--	3E-02
			ACENAPHTHYLENE	--	--	--	--	--	1E-01	--	--	1E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	6E-03	--	--	6E-03
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	3E-02	--	--	3E-02
			BENZ(A)ANTHRACENE	9E-05	--	--	9E-05	--	--	--	--	--
			BENZO(A)PYRENE	3E-04	--	--	3E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-05	--	--	3E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	3E-03	--	--	3E-03
			BENZO(K)FLUORANTHENE	3E-06	--	--	3E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	3E-07	--	--	3E-07	Increased relative liver weight (H)	9E-03	--	--	9E-03
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	6E-07	--	--	6E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	5E-05	--	--	5E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E+00	--	--	3E+00
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	7E-02	--	--	7E-02
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	7E-02	--	--	7E-02
			HEXACHLOROBUTADIENE	2E-07	--	--	2E-07	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	1E-05	--	--	1E-05	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	3E+00	--	--	3E+00
			NITROBENZENE	--	--	--	--	Hematologic (B), adrenal, renal (R) and hepatic (H) lesions	9E-02	--	--	9E-02
			PHENANTHRENE	--	--	--	--	--	2E-01	--	--	2E-01

TABLE 9.9 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient								
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total				
Water	Potable Water	Exposure Unit 8	PHENOL	--	--	--	--	Decreased maternal weight gain (W)	1E-01	--	--	1E-01				
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	6E-02	--	--	6E-02				
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--				
			1,2,4-TRICHLOROBENZENE	1E-07	--	--	1E-07	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	2E-02	--	--	2E-02				
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--				
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	1E-01	--	--	1E-01				
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--				
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--				
			1,4-DICHLOROBENZENE	6E-06	--	--	6E-06	Liver	1E-01	--	--	1E-01				
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	2E-04	--	--	2E-04				
			ACETONE	--	--	--	--	Nephropathy	1E-03	--	--	1E-03				
			BENZENE	7E-04	--	--	7E-04	Reduced lymphocyte count	2E+01	--	--	2E+01				
			BROMODICHLOROMETHANE	4E-07	--	--	4E-07	Renal cytomegaly (R)	3E-03	--	--	3E-03				
			CARBON DISULFIDE	--	--	--	--	Fetal toxicity/malformations	2E-03	--	--	2E-03				
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-01	--	--	2E-01				
			CHLOROETHANE	--	--	--	--	--	--	--	--	--				
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	3E-02	--	--	3E-02				
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	7E-04	--	--	7E-04				
			METHYLENE CHLORIDE	1E-08	--	--	1E-08	Liver toxicity (H)	2E-04	--	--	2E-04				
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--				
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--				
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	7E-02	--	--	7E-02				
			TETRACHLOROETHENE	4E-07	--	--	4E-07	Hepatotoxicity in mice (H), weight gain in rats	5E-04	--	--	5E-04				
			TOLUENE	--	--	--	--	Increased kidney weight (R)	3E-01	--	--	3E-01				
			VINYL CHLORIDE	2E-06	--	--	2E-06	Liver cell polymorphism (H)	6E-03	--	--	6E-03				
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	9E-02	--	--	9E-02				
			Chemical Total				1E-03	--	--	1E-03		5E+01	--	--	5E+01	
			Exposure Point Total								1E-03					5E+01
			Exposure Medium Total								1E-03					5E+01
			Medium Total								1E-03					5E+01
			Receptor Total								1E-03					5E+01
										Receptor HI Total				5E+01		

Total Risk Across All Media = 1E-03

Total Hazard Across All Media = 5E+01

Total Liver HI Across All Media = 5E-01
Total Kidney HI Across All Media = 4E-01
Total Nervous System Effects HI Across All Media = 6E+00
Total Lymphocyte Effects HI Across All Media = 2E+01
Total Nasal/Respiratory Effects HI Across All Media = 3E+00
Total Ocular Effects HI Across All Media = 1E-01
Total Other Effects HI Across All Media = 1E+01

TABLE 9.9a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-04	--	2E-04
			ARSENIC	--	6E-10	--	6E-10	Development, cardiovascular, nervous system	--	2E-05	--	2E-05
			CADMIUM	--	7E-10	--	7E-10	--	--	--	--	--
			CHROMIUM	--	3E-08	--	3E-08	--	--	2E-04	--	2E-04
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-03	--	1E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	1E-06	--	1E-06
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	1E-11	--	1E-11	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	2E-10	--	2E-10	Decreased lymphocyte count	--	7E-06	--	7E-06
	Chemical Total			--	3E-08	--	3E-08	--	2E-03	--	2E-03	
Exposure Point Total			3E-08				2E-03					
Exposure Medium Total			3E-08				2E-03					
Medium Total			3E-08				2E-03					
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	3E-07	--	5E-08	3E-07	Developmental effects	1E-02	--	3E-03	2E-02
			ALUMINUM	--	--	--	--	Neurotoxicology	2E-03	--	--	2E-03
			ARSENIC	5E-07	--	1E-07	6E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	9E-03	--	2E-03	1E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	7E-03	--	2E-03	9E-03
			CHROMIUM	--	--	--	--	--	2E-02	--	--	2E-02
			COPPER	--	--	--	--	Gastrointestinal effects	1E-03	--	--	1E-03
			IRON	--	--	--	--	Gastrointestinal effects	8E-03	--	--	8E-03
			MANGANESE	--	--	--	--	CNS (N)	1E-03	--	--	1E-03
			MERCURY	--	--	--	--	autoimmune effects	3E-03	--	--	3E-03

TABLE 9.9a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
			VANADIUM	--	--	--		Decreased hair cystine	7E-04	--	--	7E-04
			HIGHLY CHLORINATED PCBs	1E-07	--	1E-07	2E-07	--	2E-02	--	2E-02	4E-02
			ACENAPHTHYLENE	--	--	--		--	3E-05	--	2E-05	5E-05
			BENZ(A)ANTHRACENE	4E-07	--	3E-07	7E-07	--	--	--	--	
			BENZO(A)PYRENE	3E-06	--	2E-06	5E-06	--	--	--	--	
			BENZO(B)FLUORANTHENE	4E-07	--	3E-07	7E-07	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--		--	3E-05	--	3E-05	6E-05
			BENZO(K)FLUORANTHENE	1E-08	--	1E-08	2E-08	--	--	--	--	
			CHRYSENE	4E-09	--	3E-09	7E-09	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	2E-07	--	2E-07	4E-07	--	--	--	--	
			DIBENZOFURAN	--	--	--		Reduced length and organ weight. Excess abdominal fat (O).	7E-04	--	5E-04	1E-03
			INDENO(1,2,3-CD)PYRENE	7E-08	--	6E-08	1E-07	--	--	--	--	
			PHENANTHRENE	--	--	--		--	2E-04	--	2E-04	4E-04
			BENZENE	3E-12	--	--	3E-12	Reduced lymphocyte count	1E-07	--	--	1E-07
	Chemical Total	5E-06	--	3E-06	8E-06		8E-02	--	3E-02	1E-01		
	Exposure Point Total			8E-06					1E-01			
	Exposure Medium Total			8E-06					1E-01			
Medium Total						8E-06				1E-01		
Receptor Total						8E-06		Receptor HI Total			1E-01	

Total Risk Across All Media = 8E-06

Total Hazard Across All Media = 1E-01

TABLE 9.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient			
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	3E-05	--	--	3E-05	Developmental effects	2E+00	--	--
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	4E-01	--	--
			ARSENIC	2E-06	--	--	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-02	--	--
			CHROMIUM	--	--	--	--	--	3E-02	--	--
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	5E-02	--	--
			MANGANESE	--	--	--	--	CNS (N)	4E-03	--	--
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	2E+00	--	--
			SELENIUM	--	--	--	--	Clinical selenosis	5E-02	--	--
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-02	--	--
			ZINC	--	--	--	--	Decreased ESOD (B)	3E-02	--	--
			HIGHLY CHLORINATED PCBs	1E-05	--	--	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E+00	--	--
			LESS CHLORINATED PCBs	1E-05	--	--	1E-05	Reduced birth weights (W)	8E-01	--	--
			4,4-DDD	5E-08	--	--	5E-08	--	--	--	--
			4,4'-DDT	5E-08	--	--	5E-08	Liver lesions (H)	3E-03	--	--
			ALDRIN	7E-07	--	--	7E-07	Liver toxicity (H)	2E-02	--	--
			DELTA-BHC	--	--	--	--	--	--	--	--
			DIELDRIN	9E-07	--	--	9E-07	Hepatic (H)	1E-02	--	--
			HEPTACHLOR EPOXIDE	6E-07	--	--	6E-07	Increased liver-to-body weight ratio in males and females (H)	6E-02	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-07	--	--	5E-07	Increased relative liver weight (H)	2E-02	--	--
			HEXACHLOROBENZENE	3E-07	--	--	3E-07	Hepatic (H)	3E-03	--	--
	Chemical Total	6E-05	--	--	6E-05		1E+01	--	--		
	Exposure Point Total						6E-05				
	Exposure Medium Total						6E-05				
Medium Total							6E-05				
Sediment	Surface Sediment	Exposure Unit 6	2,3,7,8-TCDD Equivalent	4E-07	--	1E-07	6E-07	Developmental effects	3E-02	--	1E-02
			ARSENIC	4E-07	--	1E-07	5E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	3E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-02	--	6E-04
			CHROMIUM	--	--	--	--	None Reported (O)	3E-01	--	--
			IRON	--	--	--	--	Gastrointestinal effects	5E-03	--	--
			LEAD	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	1E-02	--	--
			MERCURY	--	--	--	--	Autoimmune effects	1E-01	--	--

TABLE 9.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient			
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal
Sediment	Surface Sediment	Exposure Unit 6	THALLIUM	--	--	--	--	Hematological effects	3E-03	--	--
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-02	--	--
			HIGHLY CHLORINATED PCBs	4E-08	--	6E-08	1E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-02	--	2E-02
			DIELDRIN	8E-09	--	--	8E-09	Hepatic (H)	1E-04	--	--
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	5E-05	--	--
			HEPTACHLOR EPOXIDE	2E-09	--	--	2E-09	Increased liver-to-body weight ratio in males and females (H)	2E-04	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E-03	--	4E-03
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	5E-05	--	8E-05
			BENZO(A)ANTHRACENE	1E-04	--	3E-05	1E-04	--	--	--	--
			BENZO(A)PYRENE	7E-04	--	2E-04	9E-04	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-04	--	4E-05	2E-04	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	6E-04	--	9E-04
			BENZO(K)FLUORANTHENE	4E-06	--	1E-06	5E-06	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	2E-08	--	2E-08	5E-08	Increased relative liver weight (H)	9E-04	--	1E-03
			CARBAZOLE	--	--	--	--	--	--	--	--
			CHRYSENE	1E-06	--	4E-07	2E-06	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	8E-05	--	2E-05	1E-04	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	8E-03	--	9E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	1E-03	--	1E-03
			HEXACHLOROBENZENE	5E-09	--	6E-09	1E-08	Hepatic (H)	5E-05	--	5E-05
			INDENO(1,2,3-CD)PYRENE	4E-05	--	9E-06	5E-05	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E-03	--	1E-03
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-03	--	4E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	4E-03	--	6E-03
			1,2,4-TRICHLOROBENZENE	7E-11	--	--	7E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	2E-05	--	--
			1,4-DICHLOROBENZENE	7E-09	--	--	7E-09	Liver	2E-04	--	--
			BENZENE	1E-08	--	--	1E-08	Reduced lymphocyte count	6E-04	--	--
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	7E-04	--	--
			METHYLENE CHLORIDE	1E-10	--	--	1E-10	Liver toxicity (H)	4E-06	--	--

TABLE 9.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient			
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal
Sediment	Surface Sediment	Exposure Unit 6	TOLUENE	--	--	--	--	Increased kidney weight (R)	8E-05	--	--
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-04	--	--
			Chemical Total	1E-03	--	3E-04	1E-03		5E-01	--	6E-02
		Exposure Point Total					1E-03				
	Exposure Medium Total						1E-03				
Medium Total							1E-03				
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	PNS (N); CNS (N)	--	--	--
			ALUMINUM	--	--	--	--	--	--	1E-05	--
			ARSENIC	--	3E-11	--	3E-11	--	--	2E-06	--
			BARIUM	--	--	--	--	--	--	8E-06	--
			CADMIUM	--	6E-11	--	6E-11	--	--	--	--
			CHROMIUM	--	1E-09	--	1E-09	--	--	1E-05	--
			COPPER	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	--	--	6E-05	--
			MERCURY	--	--	--	--	--	--	4E-07	--
			SILVER	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	8E-13	--	8E-13	--	--	--	--
			LESS CHLORINATED PCBs	--	4E-13	--	4E-13	--	--	--	--
			DIELDRIN	--	5E-13	--	5E-13	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	Nasal/respiratory (P)	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	Liver	--	--	--
			DIBENZOFURAN	--	--	--	--	Decreased lymphocyte count	--	--	--
			HEXACHLOROBENZENE	--	4E-13	--	4E-13	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	8E-13	--	8E-13	--	--	--	--
			NAPHTHALENE	--	--	--	--	--	--	8E-08	--
			PHENANTHRENE	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	Psychomotor and cognitive impairments	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	Development, cardiovascular, nervous system	--	3E-04	--
			1,4-DICHLOROBENZENE	--	1E-07	--	1E-07	Renal toxicity	--	2E-04	--

TABLE 9.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient			
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal
Surface Soil	Outdoor Air	Exposure Unit 6	BENZENE	--	8E-09	--	8E-09	--	--	4E-04	--
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--
			Chemical Total	--	2E-07	--	2E-07	--	--	9E-04	--
		Exposure Point Total					2E-07				
	Exposure Medium Total						2E-07				
Medium Total							2E-07				
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	2E-06	--	7E-07	3E-06	Developmental effects	2E-01	--	5E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	2E-03	--	--
			ARSENIC	3E-07	--	1E-07	4E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	8E-03	--	3E-03
			BARIIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	5E-04	--	--
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-02	--	5E-03
			CHROMIUM	--	--	--	--	--	1E-02	--	--
			COPPER	--	--	--	--	Gastrointestinal effects	2E-03	--	--
			IRON	--	--	--	--	Gastrointestinal effects	5E-03	--	--
			LEAD	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	6E-04	--	--
			MERCURY	--	--	--	--	Autoimmune effects	1E-02	--	--
			SILVER	--	--	--	--	Argyria (In)	9E-04	--	--
			THALLIUM	--	--	--	--	Hematological effects	3E-03	--	--
			VANADIUM	--	--	--	--	Decreased hair cystine	7E-04	--	--
			HIGHLY CHLORINATED PCBs	8E-08	--	1E-07	2E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-02	--	4E-02
			LESS CHLORINATED PCBs	4E-08	--	6E-08	9E-08	Reduced birth weights (W)	3E-03	--	5E-03
			DIELDRIN	5E-08	--	--	5E-08	Hepatic (H)	7E-04	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	9E-04	--	1E-03
			ACENAPHTHYLENE	--	--	--	--	--	4E-05	--	6E-05
			BENZ(A)ANTHRACENE	9E-07	--	1E-06	2E-06	--	--	--	--
			BENZO(A)PYRENE	1E-05	--	2E-05	3E-05	--	--	--	--
			BENZO(B)FLUORANTHENE	8E-07	--	1E-06	2E-06	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-05	--	7E-05
			BENZO(K)FLUORANTHENE	7E-08	--	1E-07	2E-07	--	--	--	--
			CHRYSENE	1E-08	--	1E-08	2E-08	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-06	--	3E-06	5E-06	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-03	--	1E-03
			HEXACHLOROBENZENE	4E-08	--	4E-08	8E-08	Hepatic (H)	3E-04	--	4E-04
			INDENO(1,2,3-CD)PYRENE	6E-07	--	8E-07	1E-06	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	3E-04	--	5E-04

TABLE 9.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal			
Soil	Surface Soil	Exposure Unit 6	PHENANTHRENE	--	--	--	--	--	2E-04	--	3E-04			
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--				
			1,2,4-TRICHLOROBENZENE	4E-10	--	--	4E-10	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1E-04	--	--			
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	3E-05	--	--			
			1,4-DICHLOROBENZENE	4E-09	--	--	4E-09	Liver	1E-04	--	--			
			BENZENE	7E-10	--	--	7E-10	Reduced lymphocyte count	4E-05	--	--			
Soil	Surface Soil	Exposure Unit 6	P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--			
			DODECANE	--	--	--	--	--	--	--	--			
			Chemical Total	2E-05	--	2E-05	4E-05	--	2E-01	--	1E-01			
			Exposure Point Total	4E-05										
			Exposure Medium Total				4E-05							
			Medium Total				4E-05							
Water	Surface Water	Exposure Unit 6	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	1E-03			
			ARSENIC	--	--	8E-09	8E-09	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	2E-04			
			CHROMIUM	--	--	--	--	--	--	--	5E-03			
			IRON	--	--	--	--	Gastrointestinal effects	--	--	2E-04			
			LEAD	--	--	--	--	--	--	--	--			
			MERCURY	--	--	--	--	Autoimmune effects	--	--	2E-04			
			THALLIUM	--	--	--	--	Hematological effects	--	--	2E-03			
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	3E-03			
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--			
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	1E-03			
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--			
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--			
			BENZ(A)ANTHRACENE	--	--	7E-05	7E-05	--	--	--	--			
			BENZO(A)PYRENE	--	--	6E-04	6E-04	--	--	--	--			
			BENZO(B)FLUORANTHENE	--	--	9E-05	9E-05	--	--	--	--			
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	5E-08	5E-08	Increased relative liver weight (H)	--	--	2E-03			
			CARBAZOLE	--	--	--	--	--	--	--	--			
			CHRYSENE	--	--	5E-07	5E-07	--	--	--	--			
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--			
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--			
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	2E-01			
			PHENANTHRENE	--	--	--	--	--	--	--	9E-03			
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--			
			Water	Surface Water	Exposure Unit 6	1,4-DICHLOROBENZENE	--	--	9E-09	9E-09	Liver	--	--	3E-04
						BENZENE	--	--	2E-07	2E-07	Reduced lymphocyte count	--	--	1E-02

TABLE 9.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient			
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal
Water	Surface Water	Exposure Unit 6	DICHLOROBENZENES	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	6E-03
			XYLENES, TOTAL	--	--	--	--		--	--	
			Chemical Total	--	--	8E-04	8E-04	--	--	2E-01	
	Exposure Point Total					8E-04					
	Exposure Medium Total					8E-04					
Medium Total							8E-04				
Receptor Total							2E-03			Receptor HI Total	

Total Risk Across All Media = 2E-03

Total Hazard Across All Media =

Total Liver HI Across All Media =
 Total Kidney HI Across All Media =
 Total Nervous System Effects HI Across All Media =
 Total Lymphocyte Effects HI Across All Media =
 Total Nasal/Respiratory Effects HI Across All Media =
 Total Ocular Effects HI Across All Media =
 Total Other Effects HI Across All Media =

Exposure Routes Total
2E+00
4E-01
5E-02
3E-02
5E-02
4E-03
2E+00
5E-02
1E-02
3E-02
3E+00
8E-01
--
3E-03
2E-02
--
1E-02
6E-02
2E-02
3E-03
1E+01
1E+01
1E+01
1E+01
4E-02
1E-02
6E-02
3E-01
5E-03
--
1E-02
1E-01

Exposure Routes Total
3E-03
2E-02
3E-02
1E-04
5E-05
2E-04
6E-03
1E-04
--
--
--
2E-03
--
2E-03
--
--
--
2E-02
2E-03
1E-04
--
2E-03
6E-03
1E-02
2E-05
2E-04
6E-04
7E-04
4E-06

Exposure Routes Total
8E-05
1E-04
6E-01
6E-01
6E-01
6E-01
--
1E-05
2E-06
8E-06
--
1E-05
--
--
--
6E-05
4E-07
--
--
--
--
--
--
--
--
--
--
--
--
--
8E-08
--
--
--
3E-04
2E-04

Exposure Routes Total
4E-04
--
--
9E-04
9E-04
9E-04
9E-04
2E-01
2E-03
1E-02
5E-04
2E-02
1E-02
2E-03
5E-03
--
6E-04
1E-02
9E-04
3E-03
7E-04
6E-02
8E-03
7E-04
2E-03
9E-05
--
--
--
1E-04
--
--
--
3E-03
7E-04
--
8E-04

Exposure Routes Total
5E-04
--
1E-04
3E-05
1E-04
4E-05
--
--
3E-01
3E-01
3E-01
3E-01
1E-03
2E-04
5E-03
2E-04
--
2E-04
2E-03
3E-03
--
1E-03
--
--
--
--
2E-03
--
--
--
2E-01
9E-03
--
3E-04
1E-02

Exposure Routes Total
--
6E-03
--
2E-01
2E-01
2E-01
2E-01
1E+01
1E+01
1E-01
1E-01
2E+00
1E-02
9E-03
4E+00
5E+00

TABLE 9.10a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	1E-07	--	4E-08	2E-07	--	9E-03	--	3E-03	1E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	2E-03	--	--	2E-03
			ARSENIC	2E-07	--	8E-08	3E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	6E-03	--	2E-03	8E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-03	--	2E-03	7E-03
			CHROMIUM	--	--	--	--	--	1E-02	--	--	1E-02
			COPPER	--	--	--	--	Gastrointestinal effects	9E-04	--	--	9E-04
			IRON	--	--	--	--	Gastrointestinal effects	6E-03	--	--	6E-03
			MANGANESE	--	--	--	--	CNS (N)	7E-04	--	--	7E-04
			MERCURY	--	--	--	--	Autoimmune effects	2E-03	--	--	2E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	5E-04	--	--	5E-04
			HIGHLY CHLORINATED PCBs	5E-08	--	7E-08	1E-07	--	1E-02	--	2E-02	4E-02
			ACENAPHTHYLENE	--	--	--	--	--	2E-05	--	3E-05	5E-05
			BENZ(A)ANTHRACENE	1E-06	--	2E-06	3E-06	--	--	--	--	--
			BENZO(A)PYRENE	8E-06	--	1E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	2E-06	3E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-05	--	3E-05	6E-05
			BENZO(K)FLUORANTHENE	4E-08	--	6E-08	1E-07	--	--	--	--	--
			CHRYSENE	1E-08	--	2E-08	3E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	7E-07	--	1E-06	2E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E-04	--	6E-04	1E-03
			INDENO(1,2,3-CD)PYRENE	2E-07	--	3E-07	5E-07	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	1E-04	--	2E-04	4E-04
			BENZENE	1E-12	--	--	1E-12	Reduced lymphocyte count	7E-08	--	--	7E-08
			Chemical Total	1E-05	--	2E-05	3E-05	--	6E-02	--	3E-02	9E-02
		Exposure Point Total					3E-05					9E-02
	Exposure Medium Total						3E-05					9E-02
Medium Total							3E-05					9E-02
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-05	--	2E-05
			ARSENIC	--	4E-11	--	4E-11	Development, cardiovascular, nervous system	--	2E-06	--	2E-06
			CADMIUM	--	5E-11	--	5E-11	--	--	--	--	--
			CHROMIUM	--	2E-09	--	2E-09	--	--	2E-05	--	2E-05
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-04	--	1E-04
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	1E-07	--	1E-07
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	8E-13	--	8E-13	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--

TABLE 9.10a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	
			PHENANTHRENE	--	--	--	--	--	--	--	--	
			BENZENE	--	1E-11	--	1E-11	Decreased lymphocyte count	--	7E-07	--	7E-07
			Chemical Total	--	2E-09	--	2E-09	--	--	2E-04	--	2E-04
		Exposure Point Total	--	--	--	2E-09	--	--	--	--	2E-04	
	Exposure Medium Total	--	--	--	2E-09	--	--	--	--	2E-04		
Medium Total	--	--	--	2E-09	--	--	--	--	2E-04			
Receptor Total					3E-05	Receptor HI Total				9E-02		

Total Risk Across All Media = 3E-05

Total Hazard Across All Media = 9E-02

TABLE 9.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	3E-05	--	--	3E-05	Developmental effects	2E+00	--	--	2E+00		
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	3E-01	--	--	3E-01		
			ARSENIC	2E-06	--	--	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	--	3E-02		
			CHROMIUM	--	--	--	--	--	2E-02	--	--	2E-02		
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	3E-02	--	--	3E-02		
			MANGANESE	--	--	--	--	CNS (N)	3E-03	--	--	3E-03		
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	1E+00	--	--	1E+00		
			SELENIUM	--	--	--	--	Clinical selenosis	3E-02	--	--	3E-02		
			VANADIUM	--	--	--	--	Decreased hair cystine	8E-03	--	--	8E-03		
			ZINC	--	--	--	--	Decreased ESOD (B)	2E-02	--	--	2E-02		
			HIGHLY CHLORINATED PCBs	1E-05	--	--	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+00	--	--	2E+00		
			LESS CHLORINATED PCBs	9E-06	--	--	9E-06	Reduced birth weights (W)	5E-01	--	--	5E-01		
			4,4-DDD	5E-08	--	--	5E-08	--	--	--	--	--		
			4,4'-DDT	5E-08	--	--	5E-08	Liver lesions (H)	2E-03	--	--	2E-03		
			ALDRIN	6E-07	--	--	6E-07	Liver toxicity (H)	1E-02	--	--	1E-02		
			DELTA-BHC	--	--	--	--	--	--	--	--	--		
			DIELDRIN	9E-07	--	--	9E-07	Hepatic (H)	9E-03	--	--	9E-03		
			HEPTACHLOR EPOXIDE	5E-07	--	--	5E-07	Increased liver-to-body weight ratio in males and females (H)	4E-02	--	--	4E-02		
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-07	--	--	5E-07	Increased relative liver weight (H)	1E-02	--	--	1E-02		
			HEXACHLOROBENZENE	3E-07	--	--	3E-07	Hepatic (H)	2E-03	--	--	2E-03		
			Chemical Total	6E-05	--	--	6E-05		6E+00	--	--	6E+00		
			Exposure Point Total											6E+00
			Exposure Medium Total											6E+00
			Medium Total											6E+00
			Sediment	Surface Sediment	Exposure Unit 6	2,3,7,8-TCDD Equivalent	7E-08	--	7E-08	1E-07	Developmental effects	3E-03	--	4E-03
		ARSENIC				6E-08	--	6E-08	1E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-03	--	1E-03	2E-03
CADMIUM	--	--				--	--	Renal (R); Significant Proteinuria	6E-03	--	2E-04	6E-03		
CHROMIUM	--	--				--	--	None Reported (O)	3E-02	--	--	3E-02		
IRON	--	--				--	--	Gastrointestinal effects	5E-04	--	--	5E-04		
LEAD	--	--				--	--	--	--	--	--	--		

TABLE 9.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 6	MANGANESE	--	--	--	--	CNS (N)	1E-03	--	--	1E-03
			MERCURY	--	--	--	--	Autoimmune effects	1E-02	--	--	1E-02
			THALLIUM	--	--	--	--	Hematological effects	3E-04	--	--	3E-04
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	6E-09	--	3E-08	4E-08	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-03	--	6E-03	7E-03
			DIELDRIN	1E-09	--	--	1E-09	Hepatic (H)	1E-05	--	--	1E-05
			ENDRIN KETONE	--	--	--	--	Mild histological lesions in liver (H), occasional convulsions	6E-06	--	--	6E-06
			HEPTACHLOR EPOXIDE	4E-10	--	--	4E-10	Increased liver-to-body weight ratio in males and females (H)	2E-05	--	--	2E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E-04	--	1E-03	2E-03
			ACENAPHTHYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	6E-06	--	3E-05	3E-05
			BENZ(A)ANTHRACENE	4E-07	--	2E-06	2E-06	--	--	--	--	--
			BENZO(A)PYRENE	3E-06	--	1E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	7E-07	--	3E-06	4E-06	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	7E-05	--	3E-04	4E-04
			BENZO(K)FLUORANTHENE	2E-08	--	8E-08	9E-08	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	3E-09	--	1E-08	2E-08	Increased relative liver weight (H)	1E-04	--	3E-04	4E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	6E-09	--	3E-08	3E-08	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-07	--	2E-06	2E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	9E-04	--	3E-03	4E-03
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	1E-04	--	5E-04	6E-04
			HEXACHLOROBENZENE	8E-10	--	3E-09	4E-09	Hepatic (H)	5E-06	--	2E-05	2E-05
			INDENO(1,2,3-CD)PYRENE	2E-07	--	7E-07	9E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E-04	--	5E-04	6E-04
			PHENANTHRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	3E-04	--	1E-03	1E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	5E-04	--	2E-03	3E-03

TABLE 9.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Sediment	Surface Sediment	Exposure Unit 6	1,2,4-TRICHLOROBENZENE	1E-11	--	--	1E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-06	--	--	3E-06		
			1,4-DICHLOROBENZENE	1E-09	--	--	1E-09	Liver	2E-05	--	--	2E-05		
			BENZENE	2E-09	--	--	2E-09	Reduced lymphocyte count	6E-05	--	--	6E-05		
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	8E-05	--	--	8E-05		
			METHYLENE CHLORIDE	2E-11	--	--	2E-11	Liver toxicity (H)	4E-07	--	--	4E-07		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	8E-06	--	--	8E-06		
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-05	--	--	1E-05		
		Chemical Total	5E-06	--	2E-05	3E-05		6E-02	--	2E-02	8E-02			
		Exposure Point Total					3E-05					8E-02		
	Exposure Medium Total					3E-05					8E-02			
Medium Total							3E-05					8E-02		
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--		
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	3E-06	--	3E-06		
			ARSENIC	--	1E-11	--	1E-11	Development, cardiovascular, nervous system	--	4E-07	--	4E-07		
			BARIUM	--	--	--	--	Renal toxicity	--	2E-06	--	2E-06		
			CADMIUM	--	2E-11	--	2E-11	--	--	--	--	--		
			CHROMIUM	--	4E-10	--	4E-10	--	--	3E-06	--	3E-06		
			COPPER	--	--	--	--	--	--	--	--	--		
			IRON	--	--	--	--	--	--	--	--	--		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	1E-05	--	1E-05		
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	9E-08	--	9E-08		
			SILVER	--	--	--	--	--	--	--	--	--		
			THALLIUM	--	--	--	--	--	--	--	--	--		
			VANADIUM	--	--	--	--	--	--	--	--	--		
					HIGHLY CHLORINATED PCBs	--	2E-13	--	2E-13	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	--	--	--	--
					LESS CHLORINATED PCBs	--	1E-13	--	1E-13	Reduced birth weights (W)	--	--	--	--
					DIELDRIN	--	1E-13	--	1E-13	--	--	--	--	--
					2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
					ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
					BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
					BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
					BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
					BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
					BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--

TABLE 9.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Surface Soil	Outdoor Air	Exposure Unit 6	CHRYSENE	--	--	--	--	--	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--		
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--		
			HEXACHLOROBENZENE	--	1E-13	--	1E-13	--	--	--	--	--		
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--		
			NAPHTHALENE	--	2E-13	--	2E-13	Nasal/respiratory (P)	--	2E-08	--	2E-08		
			PHENANTHRENE	--	--	--	--	--	--	--	--	--		
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	5E-05	--	5E-05		
			1,4-DICHLOROBENZENE	--	5E-08	--	5E-08	Liver	--	4E-05	--	4E-05		
			BENZENE	--	3E-09	--	3E-09	Decreased lymphocyte count	--	9E-05	--	9E-05		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
	DODECANE	--	--	--	--	--	--	--	--	--				
		Chemical Total	--	5E-08	--	5E-08		--	2E-04	--	2E-04			
		Exposure Point Total						--	2E-04	--	2E-04			
	Exposure Medium Total						5E-08				2E-04			
Medium Total								5E-08				2E-04		
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	3E-07	--	3E-07	6E-07	Developmental effects	2E-02	--	2E-02	3E-02		
			ALUMINUM	--	--	--	--	Neurotoxicity	2E-04	--	--	2E-04		
			ARSENIC	5E-08	--	5E-08	1E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	9E-04	--	9E-04	2E-03		
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	6E-05	--	--	6E-05		
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-03	--	2E-03	3E-03		
			CHROMIUM	--	--	--	--	--	1E-03	--	--	1E-03		
			COPPER	--	--	--	--	Gastrointestinal effects	2E-04	--	--	2E-04		
			IRON	--	--	--	--	Gastrointestinal effects	6E-04	--	--	6E-04		
			LEAD	--	--	--	--	--	--	--	--	--		
			MANGANESE	--	--	--	--	CNS (N)	7E-05	--	--	7E-05		
			MERCURY	--	--	--	--	Autoimmune effects	1E-03	--	--	1E-03		
			SILVER	--	--	--	--	Argyria (In)	9E-05	--	--	9E-05		
			THALLIUM	--	--	--	--	Hematological effects	3E-04	--	--	3E-04		
			VANADIUM	--	--	--	--	Decreased hair cystine	8E-05	--	--	8E-05		
					HIGHLY CHLORINATED PCBs	1E-08	--	6E-08	7E-08	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-03	--	1E-02	1E-02

TABLE 9.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 6	LESS CHLORINATED PCBs	6E-09	--	3E-08	3E-08	Reduced birth weights (W)	3E-04	--	2E-03	2E-03
			DIELDRIN	7E-09	--	--	7E-09	Hepatic (H)	7E-05	--	--	7E-05
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E-04	--	4E-04	5E-04
			ACENAPHTHYLENE	--	--	--	--	--	4E-06	--	2E-05	2E-05
			BENZ(A)ANTHRACENE	2E-08	--	1E-07	1E-07	--	--	--	--	--
			BENZO(A)PYRENE	3E-07	--	1E-06	1E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-08	--	9E-08	1E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-06	--	2E-05	3E-05
			BENZO(K)FLUORANTHENE	2E-09	--	7E-09	9E-09	--	--	--	--	--
			CHRYSENE	2E-10	--	1E-09	1E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	5E-08	--	2E-07	3E-07	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-04	--	4E-04	6E-04
			HEXACHLOROBENZENE	6E-09	--	2E-08	3E-08	Hepatic (H)	4E-05	--	1E-04	2E-04
			INDENO(1,2,3-CD)PYRENE	1E-08	--	6E-08	7E-08	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	4E-05	--	2E-04	2E-04
			PHENANTHRENE	--	--	--	--	--	2E-05	--	9E-05	1E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	6E-11	--	--	6E-11	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	1E-05	--	--	1E-05
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	3E-06	--	--	3E-06
			1,4-DICHLOROBENZENE	7E-10	--	--	7E-10	Liver	1E-05	--	--	1E-05
			BENZENE	1E-10	--	--	1E-10	Reduced lymphocyte count	4E-06	--	--	4E-06
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	8E-07	--	2E-06	3E-06		3E-02	--	3E-02	6E-02
		Exposure Point Total					3E-06					6E-02
	Exposure Medium Total						3E-06					6E-02
Medium Total							3E-06					6E-02
Surface Water	Surface Water	Exposure Unit 6	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	5E-04	5E-04
			ARSENIC	--	--	5E-09	5E-09	Hyperpigmentation (In); Vascular (V); PNS (N)	--	--	9E-05	9E-05
			CHROMIUM	--	--	--	--	--	--	--	2E-03	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	--	--	1E-04	1E-04
			LEAD	--	--	--	--	--	--	--	--	--
			MERCURY	--	--	--	--	Autoimmune effects	--	--	8E-05	8E-05
			THALLIUM	--	--	--	--	Hematological effects	--	--	7E-04	7E-04
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	--	--	1E-03	1E-03

TABLE 9.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 6	2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	--	--	6E-04	6E-04
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	7E-06	7E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	6E-05	6E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	9E-06	9E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	--	--	3E-08	3E-08	Increased relative liver weight (H)	--	--	9E-04	9E-04
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	7E-08	7E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	--	--	--	--
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	--	--	9E-02	9E-02
			PHENANTHRENE	--	--	--	--	--	--	--	4E-03	4E-03
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	--	--	--	--
			1,4-DICHLOROBENZENE	--	--	6E-09	6E-09	Liver	--	--	1E-04	1E-04
			BENZENE	--	--	1E-07	1E-07	Reduced lymphocyte count	--	--	5E-03	5E-03
			DICHLOROBENZENES	--	--	--	--	--	--	--	--	--
			TOLUENE	--	--	--	--	Increased kidney weight (R)	--	--	3E-03	3E-03
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	--	--	--	--
		Chemical Total		--	--	8E-05	8E-05		--	--	1E-01	1E-01
		Exposure Point Total					8E-05					1E-01
	Exposure Medium Total						8E-05					1E-01
Medium Total							8E-05					1E-01
Receptor Total							2E-04					Receptor HI Total 6E+00

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 6E+00

Total Liver HI Across All Media = 7E-02
Total Kidney HI Across All Media = 2E-02
Total Nervous System Effects HI Across All Media = 1E+00
Total Lymphocyte Effects HI Across All Media = 5E-03
Total Nasal/Respiratory Effects HI Across All Media = 2E-03
Total Ocular Effects HI Across All Media = 2E+00
Total Other Effects HI Across All Media = 3E+00

TABLE 9.11a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--	
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	4E-06	--	4E-06	
			ARSENIC	--	1E-11	--	1E-11	Development, cardiovascular, nervous system	--	5E-07	--	5E-07	
			CADMIUM	--	2E-11	--	2E-11	--	--	--	--	--	
			CHROMIUM	--	7E-10	--	7E-10	--	--	5E-06	--	5E-06	
			COPPER	--	--	--	--	--	--	--	--	--	
			IRON	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	3E-05	--	3E-05	
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-08	--	2E-08	
			VANADIUM	--	--	--	--	--	--	--	--	--	
			HIGHLY CHLORINATED PCBs	--	3E-13	--	3E-13	--	--	--	--	--	
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--	
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--	
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--	
			CHRYSENE	--	--	--	--	--	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--	
			PHENANTHRENE	--	--	--	--	--	--	--	--	--	
			BENZENE	--	5E-12	--	5E-12	Decreased lymphocyte count	--	2E-07	--	2E-07	
	Chemical Total			--	8E-10	--	8E-10		--	3E-05	--	3E-05	
Exposure Point Total							8E-10					3E-05	
Exposure Medium Total							8E-10					3E-05	
Medium Total								8E-10					3E-05
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	2E-08	--	2E-08	4E-08	--	1E-03	--	1E-03	2E-03	
			ALUMINUM	--	--	--	--	Neurotoxicity	2E-04	--	--	2E-04	
			ARSENIC	4E-08	--	4E-08	8E-08	Hyperpigmentation (In); Vascular (V); PNS (N)	7E-04	--	7E-04	1E-03	
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-04	--	7E-04	1E-03	
			CHROMIUM	--	--	--	--	--	1E-03	--	--	1E-03	
			COPPER	--	--	--	--	Gastrointestinal effects	9E-05	--	--	9E-05	
			IRON	--	--	--	--	Gastrointestinal effects	6E-04	--	--	6E-04	
			MANGANESE	--	--	--	--	CNS (N)	7E-05	--	--	7E-05	
			MERCURY	--	--	--	--	Autoimmune effects	2E-04	--	--	2E-04	

TABLE 9.11a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	VANADIUM	--	--	--	--	Decreased hair cystine	5E-05	--	--	5E-05
			HIGHLY CHLORINATED PCBs	8E-09	--	4E-08	4E-08	--	1E-03	--	7E-03	8E-03
			ACENAPHTHYLENE	--	--	--	--	--	2E-06	--	9E-06	1E-05
			BENZ(A)ANTHRACENE	3E-08	--	1E-07	1E-07	--	--	--	--	--
			BENZO(A)PYRENE	2E-07	--	9E-07	1E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-08	--	1E-07	2E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	2E-06	--	1E-05	1E-05
			BENZO(K)FLUORANTHENE	1E-09	--	4E-09	5E-09	--	--	--	--	--
			CHRYSENE	3E-10	--	1E-09	2E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-08	--	8E-08	9E-08	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E-05	--	2E-04	2E-04
			INDENO(1,2,3-CD)PYRENE	5E-09	--	2E-08	3E-08	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	2E-05	--	7E-05	8E-05
			BENZENE	2E-13	--	--	2E-13	Reduced lymphocyte count	8E-09	--	--	8E-09
	Chemical Total	3E-07		1E-06	2E-06		6E-03		1E-02	2E-02		
	Exposure Point Total				2E-06					2E-02		
	Exposure Medium Total				2E-06					2E-02		
Medium Total				2E-06					2E-02			
Receptor Total				2E-06	Receptor HI Total				2E-02			

Total Risk Across All Media = 2E-06

Total Hazard Across All Media = 2E-02

TABLE 9.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	8E-04	--	8E-04
			ARSENIC	--	2E-09	--	2E-09	Development, cardiovascular, nervous system	--	1E-04	--	1E-04
			BARIIUM	--	--	--	--	Renal toxicity	--	4E-04	--	4E-04
			CADMIUM	--	3E-09	--	3E-09	--	--	--	--	--
			CHROMIUM	--	7E-08	--	7E-08	Respiratory (P)	--	7E-04	--	7E-04
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	3E-03	--	3E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-05	--	2E-05
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	4E-11	--	4E-11	--	--	--	--	--
			LESS CHLORINATED PCBs	--	2E-11	--	2E-11	--	--	--	--	--
			DIELDRIN	--	3E-11	--	3E-11	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	2E-11	--	2E-11	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	4E-11	--	4E-11	Nasal/respiratory (P)	--	4E-06	--	4E-06
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	1E-02	--	1E-02
			1,4-DICHLOROBENZENE	--	8E-06	--	8E-06	Liver	--	1E-02	--	1E-02
			BENZENE	--	4E-07	--	4E-07	Decreased lymphocyte count	--	2E-02	--	2E-02
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	--	8E-06	--	8E-06		--	5E-02	--	5E-02
		Exposure Point Total					8E-06					5E-02
	Exposure Medium Total						8E-06					5E-02
Medium Total							8E-06					5E-02

TABLE 9.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	4E-05	--	1E-06	4E-05	Developmental effects	3E+00	--	1E-01	3E+00
			ALUMINUM	--	--	--	--	Neurotoxicity	5E-02	--	--	5E-02
			ARSENIC	7E-06	--	2E-07	7E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-01	--	6E-03	2E-01
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	1E-02	--	--	1E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-01	--	1E-02	2E-01
			CHROMIUM	--	--	--	--	--	2E-01	--	--	2E-01
			COPPER	--	--	--	--	Gastrointestinal effects	4E-02	--	--	4E-02
			IRON	--	--	--	--	Gastrointestinal effects	1E-01	--	--	1E-01
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	1E-02	--	--	1E-02
			MERCURY	--	--	--	--	Autoimmune effects	2E-01	--	--	2E-01
			SILVER	--	--	--	--	Argyria (In)	2E-02	--	--	2E-02
			THALLIUM	--	--	--	--	Hematological effects	6E-02	--	--	6E-02
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-02	--	--	2E-02
			HIGHLY CHLORINATED PCBs	2E-06	--	3E-07	2E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	5E-01	--	8E-02	6E-01
			LESS CHLORINATED PCBs	8E-07	--	1E-07	9E-07	Reduced birth weights (W)	7E-02	--	1E-02	8E-02
			DIELDRIN	1E-06	--	--	1E-06	Hepatic (H)	1E-02	--	--	1E-02
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E-02	--	3E-03	2E-02
			ACENAPHTHYLENE	--	--	--	--	--	8E-04	--	1E-04	1E-03
			BENZ(A)ANTHRACENE	2E-05	--	3E-06	2E-05	--	--	--	--	--
			BENZO(A)PYRENE	2E-04	--	3E-05	3E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-05	--	3E-06	2E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-03	--	2E-04	1E-03
			BENZO(K)FLUORANTHENE	2E-06	--	2E-07	2E-06	--	--	--	--	--
			CHRYSENE	2E-07	--	3E-08	2E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-05	--	6E-06	5E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-02	--	3E-03	3E-02
			HEXACHLOROBENZENE	8E-07	--	9E-08	9E-07	Hepatic (H)	8E-03	--	9E-04	8E-03
			INDENO(1,2,3-CD)PYRENE	1E-05	--	2E-06	1E-05	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	8E-03	--	1E-03	9E-03
			PHENANTHRENE	--	--	--	--	--	4E-03	--	6E-04	5E-03
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	8E-09	--	--	8E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-03	--	--	3E-03
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	6E-04	--	--	6E-04
			1,4-DICHLOROBENZENE	9E-08	--	--	9E-08	Liver	3E-03	--	--	3E-03

TABLE 9.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Soil	Surface Soil	Exposure Unit 6	BENZENE	2E-08	--	--	2E-08	Reduced lymphocyte count	9E-04	--	--	9E-04			
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--				
			DODECANE	--	--	--	--	--	--	--	--				
			Chemical Total	4E-04	--	5E-05	4E-04		5E+00	--	2E-01	5E+00			
		Exposure Point Total				4E-04				5E+00					
	Exposure Medium Total				4E-04				5E+00						
Medium Total							4E-04				5E+00				
Ground Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicity	2E+00	--	3E-03	2E+00			
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	4E-01	--	5E-03	4E-01			
			ARSENIC	8E-05	--	2E-07	8E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	2E+00	--	4E-03	2E+00			
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney (R)	5E-01	--	1E-02	5E-01			
			BERYLLIUM	--	--	--	--	Small intestinal lesions	3E-02	--	8E-03	3E-02			
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-01	--	1E-02	1E-01			
			CHROMIUM	--	--	--	--	--	1E+00	--	3E-01	2E+00			
			COBALT	--	--	--	--	--	--	--	--	--			
			COPPER	--	--	--	--	Gastrointestinal effects	1E-01	--	3E-04	1E-01			
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	1E-01	--	2E-04	1E-01			
			IRON	--	--	--	--	Gastrointestinal effects	4E+00	--	8E-03	4E+00			
			LEAD	--	--	--	--	--	--	--	--	--			
			MANGANESE	--	--	--	--	CNS (N)	8E-01	--	5E-02	9E-01			
			MERCURY	--	--	--	--	Autoimmune effects	5E-01	--	6E-03	5E-01			
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	2E-01	--	2E-03	2E-01			
			SELENIUM	--	--	--	--	Clinical selenosis	5E-02	--	1E-04	5E-02			
			SILVER	--	--	--	--	Argyria (In)	3E-02	--	9E-04	3E-02			
			THALLIUM	--	--	--	--	Hematological effects	6E+00	--	1E-02	6E+00			
			VANADIUM	--	--	--	--	Decreased hair cystine	3E-01	--	3E-02	3E-01			
			ZINC	--	--	--	--	Decreased ESOD (B)	2E-02	--	3E-05	2E-02			
						HIGHLY CHLORINATED PCBs	8E-07	--	--	8E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-01	--	--	2E-01
						4,4'-DDD	1E-07	--	5E-07	6E-07	--	--	--	--	
						4,4'-DDT	2E-06	--	1E-05	2E-05	Liver lesions (H)	1E-01	--	9E-01	1E+00
						ALDRIN	3E-06	--	2E-07	3E-06	Liver toxicity (H)	7E-02	--	4E-03	8E-02
			ALPHA-BHC	7E-06	--	--	7E-06	--	--	--	--	--			

TABLE 9.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	ENDOSULFAN II	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	7E-04	--	--	7E-04
			ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	2E-04	--	--	2E-04
			HEPTACHLOR EPOXIDE	5E-07	--	--	5E-07	Increased liver-to-body weight ratio in males and females (H)	5E-02	--	--	5E-02
			1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	2E-02	--	--	2E-02
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	2E-01	--	4E-02	3E-01
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	1E+01	--	1E+00	1E+01
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E+01	--	--	1E+01
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	1E+00	--	7E-02	1E+00
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	6E+00	--	3E-01	6E+00
			4-CHLORO-3-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			4-METHYLPHENOL	--	--	--	--	--	1E+01	--	6E-01	1E+01
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	1E-01	--	--	1E-01
			ACENAPHTHYLENE	--	--	--	--	--	4E-01	--	--	4E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	2E-02	--	--	2E-02
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	1E-01	--	--	1E-01
			BENZ(A)ANTHRACENE	2E-04	--	1E-02	1E-02	--	--	--	--	--
			BENZO(A)PYRENE	6E-04	--	6E-02	6E-02	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-05	--	7E-03	7E-03	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-02	--	--	1E-02
			BENZO(K)FLUORANTHENE	5E-06	--	--	5E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	8E-07	--	7E-07	1E-06	Increased relative liver weight (H)	3E-02	--	3E-02	6E-02
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	1E-06	--	6E-05	6E-05	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	9E-05	--	1E-02	1E-02	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E+01	--	--	1E+01
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-01	--	7E-01	1E+00
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	3E-01	--	--	3E-01
			HEXACHLOROBUTADIENE	4E-07	--	6E-07	1E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	3E-05	--	3E-03	3E-03	--	--	--	--	--

TABLE 9.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+01	--	5E+00	2E+01
			NITROBENZENE	--	--	--	--	Hematologic (B), adrenal, renal (R) and hepatic (H) lesions	3E-01	--	--	3E-01
			PHENANTHRENE	--	--	--	--	--	9E-01	--	1E+00	2E+00
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	4E-01	--	1E-02	4E-01
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	2E-01	--	--	2E-01
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	3E-07	--	2E-07	5E-07	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	9E-02	--	6E-02	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	4E-01	--	1E-01	5E-01
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	1E-05	--	5E-06	2E-05	Liver	4E-01	--	2E-01	6E-01
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	6E-04	--	--	6E-04
			ACETONE	--	--	--	--	Nephropathy	6E-03	--	--	6E-03
			BENZENE	2E-03	--	1E-04	2E-03	Reduced lymphocyte count	9E+01	--	8E+00	1E+02
			BROMODICHLOROMETHANE	1E-06	--	5E-08	1E-06	Renal cytomegaly (R)	1E-02	--	4E-04	1E-02
			CARBON DISULFIDE	--	--	--	--	Fetal toxicity/malformations	8E-03	--	8E-04	9E-03
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	6E-01	--	1E-01	7E-01
			CHLOROETHANE	--	--	--	--	--	--	--	--	--
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	9E-02	--	3E-02	1E-01
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	3E-03	--	--	3E-03
			METHYLENE CHLORIDE	3E-08	--	6E-10	3E-08	Liver toxicity (H)	8E-04	--	2E-05	8E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	3E-01	--	7E-02	3E-01
			TETRACHLOROETHENE	9E-07	--	3E-07	1E-06	Hepatotoxicity in mice (H), weight gain in rats	2E-03	--	6E-04	3E-03
			TOLUENE	--	--	--	--	Increased kidney weight (R)	1E+00	--	2E-01	1E+00
			VINYL CHLORIDE	5E-06	--	1E-07	5E-06	Liver cell polymorphism (H)	2E-02	--	7E-04	2E-02
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	3E-01	--	--	3E-01
			Chemical Total	3E-03	--	9E-02	1E-01		2E+02	--	2E+01	2E+02
		Exposure Point Total					1E-01					2E+02
	Exposure Medium Total						1E-01					2E+02
	Shower Vapor	Exposure Unit 8	1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRIMETHYLBENZENE	--	--	--	--	Hematological and Pulmonary	--	3E+01	--	3E+01
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	2E+00	--	2E+00
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,4-DICHLOROBENZENE	--	3E-04	--	3E-04	Liver	--	4E-01	--	4E-01

TABLE 9.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shower Vapor	Exposure Unit 8	2-HEXANONE	--	--	--	--	Peripheral neuropathy	--	6E-03	--	6E-03
			ACETONE	--	--	--	--	Neurological effects	--	2E-03	--	2E-03
			BENZENE	--	3E-03	--	3E-03	Decreased lymphocyte count	--	1E+02	--	1E+02
			BROMODICHLOROMETHANE	--	6E-06	--	6E-06	--	--	--	--	--
			CARBON DISULFIDE	--	--	--	--	Peripheral nervous system dysfunction	--	1E-02	--	1E-02
			CHLOROBENZENE	--	--	--	--	--	--	--	--	--
			CHLOROETHANE	--	--	--	--	Delayed fetal ossification	--	3E-04	--	3E-04
			CHLOROFORM	--	2E-05	--	2E-05	Hepatic effects	--	8E-02	--	8E-02
			ETHYLBENZENE	--	--	--	--	Developmental toxicity	--	9E-02	--	9E-02
			ISOPROPYLBENZENE	--	--	--	--	Increased kidney and adrenal weights	--	7E-03	--	7E-03
			METHYLENE CHLORIDE	--	2E-08	--	2E-08	Hepatic effects	--	5E-04	--	5E-04
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Central nervous system effects	--	5E-02	--	5E-02
			TETRACHLOROETHENE	--	1E-10	--	1E-10	Neurological effects	--	7E-04	--	7E-04
			TOLUENE	--	--	--	--	Neurological effects	--	2E-01	--	2E-01
			VINYL CHLORIDE	--	5E-07	--	5E-07	Liver cell polymorphism	--	6E-03	--	6E-03
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	5E+00	--	5E+00
			Chemical Total	--	3E-03	--	3E-03		--	2E+02	--	2E+02
		Exposure Point Total					3E-03					2E+02
	Exposure Medium Total						3E-03					2E+02
Medium Total							1E-01					4E+02
Receptor Total							1E-01					4E+02
								Receptor HI Total				

Total Risk Across All Media = 1E-01

Total Hazard Across All Media = 4E+02

Total Liver HI Across All Media =	4E+00
Total Kidney HI Across All Media =	1E+00
Total Nervous System Effects HI Across All Media =	3E+01
Total Lymphocyte Effects HI Across All Media =	2E+02
Total Nasal/Respiratory Effects HI Across All Media =	4E+01
Total Ocular Effects HI Across All Media =	8E-01
Total Other Effects HI Across All Media =	7E+01

TABLE 9.12a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E-03	--	1E-03
			ARSENIC	--	2E-09	--	2E-09	Development, cardiovascular, nervous system	--	1E-04	--	1E-04
			CADMIUM	--	3E-09	--	3E-09	--	--	--	--	--
			CHROMIUM	--	1E-07	--	1E-07	--	1E-03	--	1E-03	--
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	7E-03	--	7E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	6E-06	--	6E-06
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	4E-11	--	4E-11	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	8E-10	--	8E-10	Decreased lymphocyte count	--	4E-05	--	4E-05
	Chemical Total			--	1E-07	--	1E-07					9E-03
Exposure Point Total											9E-03	
Exposure Medium Total											9E-03	
Medium Total											9E-03	
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	3E-06	--	9E-08	3E-06	--	2E-01	--	7E-03	2E-01
			ALUMINUM	--	--	--	--	Neurotoxicity	3E-02	--	--	3E-02
			ARSENIC	5E-06	--	2E-07	5E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	4E-03	1E-01
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-01	--	5E-03	1E-01
			CHROMIUM	--	--	--	--	--	3E-01	--	--	3E-01
			COPPER	--	--	--	--	Gastrointestinal effects	2E-02	--	--	2E-02
			IRON	--	--	--	--	Gastrointestinal effects	1E-01	--	--	1E-01
			MANGANESE	--	--	--	--	CNS (N)	2E-02	--	--	2E-02
			MERCURY	--	--	--	--	Autoimmune effects	4E-02	--	--	4E-02

TABLE 9.12a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	VANADIUM	--	--	--	--	Decreased hair cystine	1E-02	--	--	1E-02
			HIGHLY CHLORINATED PCBs	1E-06	--	2E-07	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-01	--	5E-02	3E-01
			ACENAPHTHYLENE	--	--	--	--	--	4E-04	--	6E-05	5E-04
			BENZ(A)ANTHRACENE	3E-05	--	4E-06	3E-05	--	--	--	--	--
			BENZO(A)PYRENE	2E-04	--	3E-05	2E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-05	--	4E-06	3E-05	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-04	--	7E-05	6E-04
			BENZO(K)FLUORANTHENE	9E-07	--	1E-07	1E-06	--	--	--	--	--
			CHRYSENE	3E-07	--	4E-08	3E-07	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-05	--	2E-06	2E-05	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-02	--	1E-03	1E-02
			INDENO(1,2,3-CD)PYRENE	5E-06	--	7E-07	6E-06	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	3E-03	--	5E-04	4E-03
			BENZENE	3E-11	--	--	3E-11	Reduced lymphocyte count	2E-06	--	--	2E-06
	Chemical Total	3E-04	--	4E-05	3E-04		1E+00	--	6E-02	1E+00		
Exposure Point Total				3E-04					1E+00			
Exposure Medium Total				3E-04					1E+00			
Medium Total						3E-04				1E+00		
Receptor Total						3E-04	Receptor HI Total				1E+00	

Total Risk Across All Media = 3E-04

Total Hazard Across All Media = 1E+00

TABLE 9.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 6	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	2E-04	--	2E-04
			ARSENIC	--	7E-10	--	7E-10	Development, cardiovascular, nervous system	--	3E-05	--	3E-05
			BARIUM	--	--	--	--	Renal toxicity	--	1E-04	--	1E-04
			CADMIUM	--	1E-09	--	1E-09	--	--	--	--	--
			CHROMIUM	--	3E-08	--	3E-08	--	--	2E-04	--	2E-04
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	9E-04	--	9E-04
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	6E-06	--	6E-06
			SILVER	--	--	--	--	--	--	--	--	--
			THALLIUM	--	--	--	--	--	--	--	--	--
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-11	--	2E-11	--	--	--	--	--
			LESS CHLORINATED PCBs	--	8E-12	--	8E-12	--	--	--	--	--
			DIELDRIN	--	1E-11	--	1E-11	--	--	--	--	--
			2-METHYLNAPHTHALENE	--	--	--	--	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			HEXACHLOROBENZENE	--	9E-12	--	9E-12	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			NAPHTHALENE	--	2E-11	--	2E-11	Nasal/respiratory (P)	--	1E-06	--	1E-06
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	4E-03	--	4E-03
			1,4-DICHLOROBENZENE	--	3E-06	--	3E-06	Liver	--	3E-03	--	3E-03
			BENZENE	--	2E-07	--	2E-07	Decreased lymphocyte count	--	6E-03	--	6E-03
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			DODECANE	--	--	--	--	--	--	--	--	--
			Chemical Total	--	3E-06	--	3E-06	--	--	1E-02	--	1E-02
		Exposure Point Total					3E-06					1E-02
	Exposure Medium Total						3E-06					1E-02
Medium Total							3E-06					1E-02

TABLE 9.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	7E-06	--	2E-07	7E-06	Developmental effects	4E-01	--	1E-02	4E-01
			ALUMINUM	--	--	--	--	Neurotoxicity	5E-03	--	--	5E-03
			ARSENIC	1E-06	--	4E-08	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	7E-04	2E-02
			BARIIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney	1E-03	--	--	1E-03
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	2E-02	--	1E-03	3E-02
			CHROMIUM	--	--	--	--	--	3E-02	--	--	3E-02
			COPPER	--	--	--	--	Gastrointestinal effects	4E-03	--	--	4E-03
			IRON	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02
			LEAD	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	CNS (N)	1E-03	--	--	1E-03
			MERCURY	--	--	--	--	Autoimmune effects	3E-02	--	--	3E-02
			SILVER	--	--	--	--	Argyria (In)	2E-03	--	--	2E-03
			THALLIUM	--	--	--	--	Hematological effects	7E-03	--	--	7E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	2E-03	--	--	2E-03
			HIGHLY CHLORINATED PCBs	3E-07	--	4E-08	3E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	5E-02	--	8E-03	6E-02
			LESS CHLORINATED PCBs	1E-07	--	2E-08	1E-07	Reduced birth weights (W)	7E-03	--	1E-03	8E-03
			DIELDRIN	2E-07	--	--	2E-07	Hepatic (H)	2E-03	--	--	2E-03
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E-03	--	3E-04	3E-03
			ACENAPHTHYLENE	--	--	--	--	--	9E-05	--	1E-05	1E-04
			BENZ(A)ANTHRACENE	5E-07	--	7E-08	6E-07	--	--	--	--	--
			BENZO(A)PYRENE	6E-06	--	9E-07	7E-06	--	--	--	--	--
			BENZO(B)FLUORANTHENE	4E-07	--	6E-08	5E-07	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-04	--	2E-05	1E-04
			BENZO(K)FLUORANTHENE	4E-08	--	5E-09	4E-08	--	--	--	--	--
			CHRYSENE	5E-09	--	8E-10	6E-09	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-07	1E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E-03	--	3E-04	3E-03
			HEXACHLOROBENZENE	1E-07	--	2E-08	1E-07	Hepatic (H)	8E-04	--	9E-05	9E-04
			INDENO(1,2,3-CD)PYRENE	3E-07	--	4E-08	3E-07	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	8E-04	--	1E-04	9E-04
			PHENANTHRENE	--	--	--	--	--	4E-04	--	6E-05	5E-04
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	1E-09	--	--	1E-09	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	3E-04	--	--	3E-04
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	6E-05	--	--	6E-05
			1,4-DICHLOROBENZENE	1E-08	--	--	1E-08	Liver	3E-04	--	--	3E-04
			BENZENE	3E-09	--	--	3E-09	Reduced lymphocyte count	9E-05	--	--	9E-05

TABLE 9.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Soil	Surface Soil	Exposure Unit 6	P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--	
			DODECANE	--	--	--	--	--	--	--	--		
			Chemical Total	2E-05	--	2E-06	2E-05		6E-01	--	2E-02	6E-01	
		Exposure Point Total				2E-05					6E-01		
	Exposure Medium Total				2E-05					6E-01			
Medium Total							2E-05					6E-01	
Ground Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicity	7E-01	--	2E-03	7E-01	
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	2E-01	--	2E-03	2E-01	
			ARSENIC	5E-05	--	1E-07	5E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	9E-01	--	2E-03	9E-01	
			BARIUM	--	--	--	--	Humans - none observed (O); Rats - Kidney	2E-01	--	7E-03	2E-01	
			BERYLLIUM	--	--	--	--	Small intestinal lesions	1E-02	--	4E-03	1E-02	
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-02	--	5E-03	6E-02	
			CHROMIUM	--	--	--	--	--	6E-01	--	1E-01	8E-01	
			COBALT	--	--	--	--	--	--	--	--	--	
			COPPER	--	--	--	--	Gastrointestinal effects	6E-02	--	1E-04	6E-02	
			CYANIDE	--	--	--	--	Weight loss, thyroid effects, myelin degeneration	4E-02	--	9E-05	4E-02	
			IRON	--	--	--	--	Gastrointestinal effects	2E+00	--	3E-03	2E+00	
			LEAD	--	--	--	--	--	--	--	--	--	
			MANGANESE	--	--	--	--	CNS (N)	4E-01	--	2E-02	4E-01	
			MERCURY	--	--	--	--	Autoimmune effects	2E-01	--	6E-03	2E-01	
			NICKEL	--	--	--	--	Decreased body and organ weight (W)	7E-02	--	8E-04	7E-02	
			SELENIUM	--	--	--	--	Clinical selenosis	2E-02	--	4E-05	2E-02	
			SILVER	--	--	--	--	Argyria (In)	1E-02	--	4E-04	1E-02	
			THALLIUM	--	--	--	--	Hematological effects	2E+00	--	5E-03	2E+00	
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-01	--	1E-02	1E-01	
			ZINC	--	--	--	--	Decreased ESOD (B)	9E-03	--	1E-05	9E-03	
				HIGHLY CHLORINATED PCBs	5E-07	--	--	5E-07	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	--	1E-01
				4,4'-DDD	7E-08	--	3E-07	4E-07	--	--	--	--	--
				4,4'-DDT	1E-06	--	1E-05	1E-05	Liver lesions (H)	6E-02	--	5E-01	5E-01
				ALDRIN	2E-06	--	1E-07	2E-06	Liver toxicity (H)	3E-02	--	2E-03	3E-02
				ALPHA-BHC	4E-06	--	--	4E-06	--	--	--	--	--
				ENDOSULFAN II	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	3E-04	--	--	3E-04
				ENDOSULFAN SULFATE	--	--	--	--	Reduced body weight gain in males and females (W); increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males (B)	1E-04	--	--	1E-04
				HEPTACHLOR EPOXIDE	3E-07	--	--	3E-07	Increased liver-to-body weight ratio in males and females (H)	2E-02	--	--	2E-02

TABLE 9.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	1,1'-BIPHENYL	--	--	--	--	Kidney Damage (R)	7E-03	--	--	7E-03
			2,4-DICHLOROPHENOL	--	--	--	--	Decreased delayed hypersensitivity response (O)	9E-02	--	2E-02	1E-01
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	6E+00	--	5E-01	6E+00
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	4E+00	--	--	4E+00
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	5E-01	--	3E-02	6E-01
			2-NITROPHENOL	--	--	--	--	--	--	--	--	--
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	2E+00	--	1E-01	3E+00
			4-CHLORO-3-METHYLPHENOL	--	--	--	--	--	--	--	--	--
			4-METHYLPHENOL	--	--	--	--	--	5E+00	--	3E-01	5E+00
			4-NITROPHENOL	--	--	--	--	--	--	--	--	--
			ACENAPHTHENE	--	--	--	--	Hepatotoxicity (H)	5E-02	--	--	5E-02
			ACENAPHTHYLENE	--	--	--	--	--	2E-01	--	--	2E-01
			ANTHRACENE	--	--	--	--	No observed effects (O)	1E-02	--	--	1E-02
			ATRAZINE	--	--	--	--	Decreased body weight gain (W)	4E-02	--	--	4E-02
			BENZ(A)ANTHRACENE	1E-04	--	1E-03	1E-03	--	--	--	--	--
			BENZO(A)PYRENE	5E-04	--	7E-03	8E-03	--	--	--	--	--
			BENZO(B)FLUORANTHENE	5E-05	--	8E-04	9E-04	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	5E-03	--	--	5E-03
			BENZO(K)FLUORANTHENE	5E-06	--	--	5E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-07	--	5E-07	1E-06	Increased relative liver weight (H)	1E-02	--	1E-02	3E-02
			CARBAZOLE	--	--	--	--	--	--	--	--	--
			CHRYSENE	9E-07	--	8E-06	8E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	7E-05	--	2E-03	2E-03	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E+00	--	--	5E+00
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	1E-01	--	4E-01	5E-01
			FLUORENE	--	--	--	--	Decreased RBC (B), packed cell volumen and hemoglobin (B)	1E-01	--	--	1E-01
			HEXACHLOROBUTADIENE	3E-07	--	4E-07	7E-07	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	2E-05	--	3E-04	3E-04	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	5E+00	--	2E+00	8E+00
			NITROBENZENE	--	--	--	--	Hematologic (B), adrenal, renal (R) and hepatic (H) lesions	1E-01	--	--	1E-01
			PHENANTHRENE	--	--	--	--	--	4E-01	--	7E-01	1E+00
			PHENOL	--	--	--	--	Decreased maternal weight gain (W)	2E-01	--	6E-03	2E-01
			PYRENE	--	--	--	--	Kidney effects (renal tubular pathology, decreased kidney weights) (R)	9E-02	--	--	9E-02
			1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--
			1,2,4-TRICHLOROBENZENE	2E-07	--	1E-07	3E-07	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	4E-02	--	3E-02	7E-02

TABLE 9.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Ground Water	Potable Water	Exposure Unit 8	1,2,4-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,2-DICHLOROBENZENE	--	--	--	--	No adverse effects observed (O)	2E-01	--	7E-02	2E-01		
			1,3,5-TRIMETHYLBENZENE	--	--	--	--	--	--	--	--	--		
			1,3-DICHLOROBENZENE	--	--	--	--	--	--	--	--	--		
			1,4-DICHLOROBENZENE	9E-06	--	4E-06	1E-05	Liver	2E-01	--	8E-02	3E-01		
			2-HEXANONE	--	--	--	--	Myofibrillar atrophy of the quadriceps.	3E-04	--	--	3E-04		
			ACETONE	--	--	--	--	Nephropathy	2E-03	--	--	2E-03		
			BENZENE	1E-03	--	1E-04	1E-03	Reduced lymphocyte count	4E+01	--	4E+00	4E+01		
			BROMODICHLOROMETHANE	7E-07	--	4E-08	7E-07	Renal cytomegaly (R)	4E-03	--	2E-04	4E-03		
			CARBON DISULFIDE	--	--	--	--	Fetal toxicity/malformations	3E-03	--	4E-04	4E-03		
			CHLOROBENZENE	--	--	--	--	Histopathologic changes in liver	2E-01	--	6E-02	3E-01		
			CHLOROETHANE	--	--	--	--	--	--	--	--	--		
			ETHYLBENZENE	--	--	--	--	Liver (H) and kidney (R) toxicity	4E-02	--	2E-02	6E-02		
			ISOPROPYLBENZENE	--	--	--	--	Increased average kidney weight in female rats (R)	1E-03	--	--	1E-03		
			METHYLENE CHLORIDE	2E-08	--	5E-10	2E-08	Liver toxicity (H)	3E-04	--	8E-06	3E-04		
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--		
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--		
			STYRENE	--	--	--	--	Red blood cell (B) and liver effects (H)	1E-01	--	3E-02	1E-01		
			TETRACHLOROETHENE	6E-07	--	2E-07	8E-07	Hepatotoxicity in mice (H), weight gain in rats	8E-04	--	3E-04	1E-03		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	4E-01	--	1E-01	5E-01		
			VINYL CHLORIDE	3E-06	--	1E-07	3E-06	Liver cell polymorphism (H)	1E-02	--	3E-04	1E-02		
			XYLENES, TOTAL	--	--	--	--	Decreased body weight (W), increased mortality (M)	1E-01	--	--	1E-01		
			Chemical Total			2E-03	--	1E-02	1E-02		8E+01	--	1E+01	9E+01
		Exposure Point Total							1E-02					9E+01
		Exposure Medium Total							1E-02					9E+01
		Shower Vapor	Exposure Unit 8	1,2,3-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--	
				1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	--	--	--	
1,2,4-TRIMETHYLBENZENE				--	--	--	--	Hematological and Pulmonary	--	5E+00	--	5E+00		
1,2-DICHLOROBENZENE				--	--	--	--	--	--	4E-01	--	4E-01		
1,3,5-TRIMETHYLBENZENE				--	--	--	--	--	--	--	--	--		
1,3-DICHLOROBENZENE				--	--	--	--	--	--	--	--	--		
1,4-DICHLOROBENZENE				--	3E-04	--	3E-04	Liver	--	7E-02	--	7E-02		
2-HEXANONE				--	--	--	--	Peripheral neuropathy	--	1E-03	--	1E-03		
ACETONE				--	--	--	--	Neurological effects	--	3E-04	--	3E-04		
BENZENE				--	2E-03	--	2E-03	Decreased lymphocyte count	--	2E+01	--	2E+01		
BROMODICHLOROMETHANE				--	5E-06	--	5E-06	--	--	--	--	--		
CARBON DISULFIDE				--	--	--	--	Peripheral nervous system dysfunction	--	2E-03	--	2E-03		
CHLOROBENZENE				--	--	--	--	--	--	--	--	--		
CHLOROETHANE				--	--	--	--	Delayed fetal ossification	--	5E-05	--	5E-05		
CHLOROFORM				--	1E-05	--	1E-05	Hepatic effects	--	1E-02	--	1E-02		
ETHYLBENZENE				--	--	--	--	Developmental toxicity	--	2E-02	--	2E-02		

TABLE 9.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shower Vapor	Exposure Unit 8	ISOPROPYLBENZENE	--	--	--	--	Increased kidney and adrenal weights	--	1E-03	--	1E-03
			METHYLENE CHLORIDE	--	2E-08	--	2E-08	Hepatic effects	--	8E-05	--	8E-05
			P-ISOPROPYLTOLUENE	--	--	--	--	--	--	--	--	--
			SEC-BUTYLBENZENE	--	--	--	--	--	--	--	--	--
			STYRENE	--	--	--	--	Central nervous system effects	--	9E-03	--	9E-03
			TETRACHLOROETHENE	--	8E-11	--	8E-11	Neurological effects	--	1E-04	--	1E-04
			TOLUENE	--	--	--	--	Neurological effects	--	3E-02	--	3E-02
			VINYL CHLORIDE	--	2E-07	--	2E-07	Liver cell polymorphism	--	1E-03	--	1E-03
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	8E-01	--	8E-01
		Chemical Total	--	2E-03	--	2E-03		--	3E+01	--	3E+01	
	Exposure Point Total			2E-03					3E+01			
	Exposure Medium Total			2E-03					3E+01			
Medium Total						2E-02				1E+02		
Receptor Total						2E-02		Receptor HI Total			1E+02	

Total Risk Across All Media = 2E-02

Total Hazard Across All Media = 1E+02

Total Liver HI Across All Media = 2E+00
Total Kidney HI Across All Media = 6E-01
Total Nervous System Effects HI Across All Media = 1E+01
Total Lymphocyte Effects HI Across All Media = 7E+01
Total Nasal/Respiratory Effects HI Across All Media = 9E+00
Total Ocular Effects HI Across All Media = 2E-01
Total Other Effects HI Across All Media = 3E+01

TABLE 9.13a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	2,3,7,8-TCDD Equivalent	--	--	--	--	--	--	--	--	--
			ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	3E-04	--	3E-04
			ARSENIC	--	9E-10	--	9E-10	Development, cardiovascular, nervous system	--	3E-05	--	3E-05
			CADMIUM	--	1E-09	--	1E-09	--	--	--	--	--
			CHROMIUM	--	5E-08	--	5E-08	--	--	3E-04	--	3E-04
			COPPER	--	--	--	--	--	--	--	--	--
			IRON	--	--	--	--	--	--	--	--	--
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E-03	--	2E-03
			MERCURY	--	--	--	--	PNS (N); CNS (N)	--	2E-06	--	2E-06
			VANADIUM	--	--	--	--	--	--	--	--	--
			HIGHLY CHLORINATED PCBs	--	2E-11	--	2E-11	--	--	--	--	--
			ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--
			BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--
			BENZO(A)PYRENE	--	--	--	--	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--
			CHRYSENE	--	--	--	--	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	--	--	--	--	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	--	--	--	--	--	--	--
			PHENANTHRENE	--	--	--	--	--	--	--	--	--
			BENZENE	--	3E-10	--	3E-10	Decreased lymphocyte count	--	1E-05	--	1E-05
	Chemical Total			--	5E-08	--	5E-08			--	2E-03	--
Exposure Point Total							5E-08					2E-03
Exposure Medium Total							5E-08					2E-03
Medium Total							5E-08					2E-03
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	4E-07	--	1E-08	4E-07	--	2E-02	--	7E-04	2E-02
			ALUMINUM	--	--	--	--	Neurotoxicity	4E-03	--	--	4E-03
			ARSENIC	8E-07	--	3E-08	9E-07	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	5E-04	1E-02
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	1E-02	--	5E-04	1E-02
			CHROMIUM	--	--	--	--	--	3E-02	--	--	3E-02
			COPPER	--	--	--	--	Gastrointestinal effects	2E-03	--	--	2E-03
			IRON	--	--	--	--	Gastrointestinal effects	1E-02	--	--	1E-02
			MANGANESE	--	--	--	--	CNS (N)	2E-03	--	--	2E-03
			MERCURY	--	--	--	--	Autoimmune effects	4E-03	--	--	4E-03
			VANADIUM	--	--	--	--	Decreased hair cystine	1E-03	--	--	1E-03
			HIGHLY CHLORINATED PCBs	2E-07	--	3E-08	2E-07	--	3E-02	--	5E-03	4E-02
			ACENAPHTHYLENE	--	--	--	--	--	5E-05	--	7E-06	5E-05
			BENZ(A)ANTHRACENE	6E-07	--	9E-08	7E-07	--	--	--	--	--

TABLE 9.13a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	BENZO(A)PYRENE	4E-06	--	6E-07	5E-06	--	--	--	--	
			BENZO(B)FLUORANTHENE	6E-07	--	9E-08	7E-07	--	--	--	--	
			BENZO(G,H,I)PERYLENE	--	--	--	--	5E-05	--	8E-06	6E-05	
			BENZO(K)FLUORANTHENE	2E-08	--	3E-09	2E-08	--	--	--	--	
			CHRYSENE	6E-09	--	9E-10	7E-09	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	4E-07	--	6E-08	4E-07	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E-03	--	1E-04	1E-03
			INDENO(1,2,3-CD)PYRENE	1E-07	--	2E-08	1E-07	--	--	--	--	
			PHENANTHRENE	--	--	--	--	3E-04	--	5E-05	4E-04	
			BENZENE	5E-12	--	--	5E-12	Reduced lymphocyte count	2E-07	--	--	2E-07
			Chemical Total	7E-06	--	1E-06	8E-06		1E-01	--	7E-03	1E-01
		Exposure Point Total				8E-06					1E-01	
	Exposure Medium Total				8E-06					1E-01		
Medium Total					8E-06					1E-01		
Receptor Total					8E-06	Receptor HI Total				1E-01		

Total Risk Across All Media = 8E-06

Total Hazard Across All Media = 1E-01

RAGS Table 10 RME Series

TABLE 10.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	8E-05	--	--	8E-05	Developmental effects	6E+00	--	--	6E+00
			ARSENIC	3E-06	--	--	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	8E-02	--	--	8E-02
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	3E+00	--	--	3E+00
			HIGHLY CHLORINATED PCBs	3E-05	--	--	3E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	9E+00	--	--	9E+00
			LESS CHLORINATED PCBs	2E-05	--	--	2E-05	Reduced birth weights (W)	2E+00	--	--	2E+00
			ALDRIN	1E-06	--	--	1E-06	Liver toxicity (H)	2E-02	--	--	2E-02
			DIELDRIN	2E-06	--	--	2E-06	Hepatic (H)	2E-02	--	--	2E-02
			Chemical Total	1E-04	--	--	1E-04		2E+01	--	--	2E+01
		Exposure Point Total					1E-04					2E+01
	Exposure Medium Total						1E-04					2E+01
Medium Total							1E-04					2E+01
Sediment	Surface Sediment	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-07	--	8E-07	1E-06	Developmental effects	1E-02	--	7E-02	8E-02
			ARSENIC	2E-07	--	8E-07	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-03	--	2E-02	3E-02
			BENZO(A)ANTHRACENE	9E-06	--	2E-04	2E-04	--	--	--	--	--
			BENZO(A)PYRENE	2E-05	--	4E-04	4E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	3E-06	--	6E-05	6E-05	--	--	--	--	--
			BENZO(K)FLUORANTHENE	--	--	2E-06	2E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-06	--	7E-05	7E-05	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	1E-06	--	2E-05	2E-05	--	--	--	--	--
			Chemical Total	4E-05	--	7E-04	7E-04		2E-02	--	9E-02	1E-01
		Exposure Point Total					7E-04					1E-01
	Exposure Medium Total						7E-04					1E-01
Medium Total							7E-04					1E-01
Surface Soil	Outdoor Air	Exposure Unit 1	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total					0E+00					0E+00
	Exposure Medium Total						0E+00					0E+00
Medium Total							0E+00					0E+00

TABLE 10.1 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	1E-06	--	7E-06	8E-06	Developmental effects	1E-01	--	5E-01	6E-01
			ARSENIC	2E-07	--	1E-06	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	6E-03	--	3E-02	4E-02
			HIGHLY CHLORINATED PCBs	5E-08	--	1E-06	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-02	--	3E-01	3E-01
			BENZ(A)ANTHRACENE	5E-07	--	9E-06	1E-05	--	--	--	--	
			BENZO(A)PYRENE	5E-06	--	9E-05	1E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	4E-07	--	8E-06	8E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-05	2E-05	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	3E-07	--	6E-06	6E-06	--	--	--	--	
			Chemical Total	9E-06	--	1E-04	2E-04		1E-01	--	9E-01	1E+00
		Exposure Point Total				2E-04					1E+00	
Exposure Medium Total				2E-04					1E+00			
Medium Total				2E-04					1E+00			
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	2E-05	2E-05	--	--	--	--	
			BENZO(A)PYRENE	--	--	2E-04	2E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	3E-05	3E-05	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	1E-05	1E-05	--	--	--	--	
			Chemical Total	--	--	3E-04	3E-04		--	--	--	0E+00
		Exposure Point Total				3E-04					0E+00	
Exposure Medium Total				3E-04					0E+00			
Medium Total				3E-04					0E+00			
Receptor Total							1E-03	Receptor HI Total			2E+01	

Total Risk Across All Media = 1E-03

Total Hazard Across All Media = 2E+01

Total Liver HI Across All Media = 5E-02
Total Nervous System Effects HI Across All Media = 3E+00
Total Ocular Effects HI Across All Media = 9E+00
Total Other Effects HI Across All Media = 9E+00

TABLE 10.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	5E-04	--	--	5E-04	Developmental effects	7E+00	--	--	7E+00
			ARSENIC	2E-05	--	--	2E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	--	1E-01
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	4E+00	--	--	4E+00
			HIGHLY CHLORINATED PCBs	2E-04	--	--	2E-04	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E+01	--	--	1E+01
			LESS CHLORINATED PCBs	1E-04	--	--	1E-04	Reduced birth weights (W)	2E+00	--	--	2E+00
			4,4-DDD	5E-07	--	--	5E-07	--	--	--	--	--
			4,4'-DDT	5E-07	--	--	5E-07	Liver lesions (H)	7E-03	--	--	7E-03
			ALDRIN	7E-06	--	--	7E-06	Liver toxicity (H)	3E-02	--	--	3E-02
			DELTA-BHC	--	--	--	--	--	--	--	--	--
			DIELDRIN	9E-06	--	--	9E-06	--	3E-02	--	--	3E-02
			HEPTACHLOR EPOXIDE	6E-06	--	--	6E-06	Increased liver-to-body weight ratio in males and females (H)	1E-01	--	--	1E-01
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-06	--	--	5E-06	Increased relative liver weight (H)	4E-02	--	--	4E-02
			HEXACHLOROBENZENE	3E-06	--	--	3E-06	Hepatic (H)	6E-03	--	--	6E-03
			Chemical Total	8E-04	--	--	8E-04		2E+01	--	--	2E+01
	Exposure Point Total			8E-04				2E+01				
Exposure Medium Total			8E-04				2E+01					
Medium Total			8E-04				2E+01					
Sediment	Surface Sediment	Exposure Unit 1	BENZ(A)ANTHRACENE	7E-06	--	3E-05	4E-05	--	--	--	--	
			BENZO(A)PYRENE	2E-05	--	7E-05	9E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	2E-06	--	1E-05	1E-05	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	3E-06	--	1E-05	2E-05	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	8E-07	--	4E-06	5E-06	--	--	--	--	
			Chemical Total	3E-05	--	1E-04	2E-04	--	--	--	0E+00	
	Exposure Point Total			2E-04				0E+00				
Exposure Medium Total			2E-04				0E+00					
Medium Total			2E-04				0E+00					
Surface Soil	Outdoor Air	Exposure Unit 1	None	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	--	--	--	--	--	
		Exposure Point Total			--				--			
	Exposure Medium Total			--				--				
Medium Total			--				--					

TABLE 10.2 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	3E-06	--	3E-06	6E-06	Developmental effects	4E-02	--	4E-02	9E-02
			BENZO(A)ANTHRACENE	4E-07	--	2E-06	2E-06	--	--	--	--	
			BENZO(A)PYRENE	4E-06	--	2E-05	2E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	3E-07	--	1E-06	2E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	8E-07	--	4E-06	5E-06	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	2E-07	--	1E-06	1E-06	--	--	--	--	
		Chemical Total	8E-06	--	3E-05	4E-05		4E-02	--	4E-02	9E-02	
		Exposure Point Total				4E-05					9E-02	
	Exposure Medium Total				4E-05					9E-02		
Medium Total							4E-05				9E-02	
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	4E-05	4E-05	--	--	--	--	
			BENZO(A)PYRENE	--	--	4E-04	4E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	5E-05	5E-05	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	--	--	3E-05	3E-05	--	--	--	--	
			Chemical Total	--	--	5E-04	5E-04		--	--	--	0E+00
		Exposure Point Total				5E-04					0E+00	
	Exposure Medium Total				5E-04					0E+00		
Medium Total							5E-04				0E+00	
Receptor Total							2E-03				Receptor HI Total	2E+01

Total Risk Across All Media = 2E-03

Total Hazard Across All Media = 2E+01

Total Liver HI Across All Media = 2E-01
Total Nervous System Effects HI Across All Media = 4E+00
Total Ocular Effects HI Across All Media = 1E+01
Total Other Effects HI Across All Media = 1E+01

TABLE 10.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Utility Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E+00	--	2E-01	2E+00
			BENZ(A)ANTHRACENE	1E-05	--	1E-05	3E-05	--	--	--	--	
			BENZO(A)PYRENE	3E-05	--	3E-05	6E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	4E-06	--	5E-06	9E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	5E-06	--	6E-06	1E-05	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E+00	--	2E-01	3E+00
			INDENO(1,2,3-CD)PYRENE	1E-06	--	2E-06	3E-06	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+00	--	9E-02	1E+00
			Chemical Total	5E-05	--	6E-05	1E-04		--	--	--	4E+00
	Exposure Point Total					1E-04					4E+00	
Exposure Medium Total					1E-04					4E+00		
Medium Total					1E-04					4E+00		
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	7E-06	--	6E-07	8E-06	Developmental effects	1E-01	--	1E-02	1E-01
			ARSENIC	2E-06	--	1E-07	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	9E-04	1E-02
			BENZ(A)ANTHRACENE	6E-06	--	2E-06	9E-06	--	--	--	--	
			BENZO(A)PYRENE	4E-05	--	2E-05	6E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	6E-06	--	2E-06	8E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	5E-06	--	2E-06	7E-06	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	1E-06	--	6E-07	2E-06	--	--	--	--	
			Chemical Total	7E-05	--	3E-05	1E-04		1E-01	--	1E-02	2E-01
			Exposure Point Total					1E-04				2E-01
	Exposure Medium Total					1E-04				2E-01		
Medium Total					1E-04				2E-01			
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	CHROMIUM	--	2E-05	--	2E-05	--	--	4E-02	--	4E-02
			1,4-DICHLOROBENZENE	--	2E-06	--	2E-06	Liver	--	6E-04	--	6E-04
			Chemical Total	--	2E-05	--	2E-05		--	4E-02	--	4E-02
		Exposure Point Total					2E-05					4E-02
	Exposure Medium Total					2E-05					4E-02	
Medium Total					2E-05					4E-02		
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	BENZO(A)PYRENE	--	--	2E-06	2E-06	--	--	--	--	--
			Chemical Total	--	--	2E-06	2E-06		--	--	--	--
		Exposure Point Total					2E-06					--
Exposure Medium Total					2E-06					--		
Medium Total					2E-06					--		

TABLE 10.3 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Utility Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	1E-05	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-04	1E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-05	2E-05	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	9E-06	9E-06	--	--	--	--	--
		Chemical Total		--	--	2E-04	2E-04		--	--	--	0E+00
		Exposure Point Total					2E-04					0E+00
	Exposure Medium Total						2E-04					0E+00
Medium Total							2E-04					0E+00
Receptor Total							4E-04				Receptor HI Total	4E+00

Total Risk Across All Media = 4E-04

Receptor HI Total 4E+00

Total Liver HI Across All Media = 6E-04
Total Nervous System Effects HI Across All Media = 1E-02
Total Other Effects HI Across All Media = 4E+00

TABLE 10.3a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Utility Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	BENZO(A)PYRENE	5E-06	--	2E-06	6E-06	--	--	--	--	--
			Chemical Total	5E-06	--	2E-06	6E-06		--	--	--	0E+00
		Exposure Point Total				6E-06					0E+00	
	Exposure Medium Total						6E-06					0E+00
Medium Total							6E-06					0E+00
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	--		--	--	--	--
		Exposure Point Total				--					--	
	Exposure Medium Total						--					--
Medium Total							--					--
Shallow Ground Water	Shallow Ground Water	Exposure Unit 9	BENZ(A)ANTHRACENE	--	--	2E-05	2E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	3E-04	3E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	4E-05	4E-05	--	--	--	--	--
		Chemical Total	--	--	4E-04	4E-04		--	--	--	0E+00	
	Exposure Point Total						4E-04					0E+00
Exposure Medium Total							4E-04					0E+00
Medium Total							4E-04					0E+00
Receptor Total							4E-04				Receptor HI Total	0E+00

Total Risk Across All Media = 4E-04

Total Hazard Across All Media = 0E+00

TABLE 10.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	2E+00	--	2E+00	4E+00
			BENZ(A)ANTHRACENE	6E-06	--	7E-06	1E-05	--	--	--	--	
			BENZO(A)PYRENE	1E-05	--	2E-05	3E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	2E-06	--	2E-06	5E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	3E-06	--	3E-06	5E-06	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	2E+00	--	2E+00	5E+00
			INDENO(1,2,3-CD)PYRENE	7E-07	--	9E-07	2E-06	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+00	--	1E+00	2E+00
			Chemical Total	3E-05	--	3E-05	6E-05		5E+00	--	5E+00	1E+01
		Exposure Point Total				6E-05				1E+01		
	Exposure Medium Total				6E-05				1E+01			
Medium Total					6E-05				1E+01			
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	4E-06	--	3E-07	4E-06	Developmental effects	2E+00	--	2E-01	2E+00
			BENZ(A)ANTHRACENE	3E-06	--	1E-06	4E-06	--	--	--	--	
			BENZO(A)PYRENE	2E-05	--	9E-06	3E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	3E-06	--	1E-06	4E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	2E-06	--	9E-07	3E-06	--	--	--	--	
			Chemical Total	3E-05	--	1E-05	5E-05		2E+00	--	2E-01	2E+00
				Exposure Point Total				5E-05				2E+00
		Exposure Medium Total				5E-05				2E+00		
Medium Total					5E-05				2E+00			
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	ALUMINUM	--	--	--	--	Psychomotor and cognitive impairments	--	1E+00	--	1E+00
			CHROMIUM	--	2E-05	--	2E-05	--	--	1E+00	--	1E+00
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	6E+00	--	6E+00
			1,4-DICHLOROBENZENE	--	2E-06	--	2E-06	Liver	--	2E-02	--	2E-02
			Chemical Total	--	2E-05	--	2E-05		--	9E+00	--	9E+00
				Exposure Point Total				2E-05				9E+00
		Exposure Medium Total				2E-05				9E+00		
Medium Total					2E-05				9E+00			
Shallow Ground Water	Shallow Ground Water	Exposure Unit 1	None	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	--		--	--	--	--
				Exposure Point Total				--				--
		Exposure Medium Total				--				--		
Medium Total					--				--			

TABLE 10.4 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	7E-06	7E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	6E-05	6E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	9E-06	9E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	4E-06	4E-06	--	--	--	--	--
		Chemical Total		--	--	8E-05	8E-05		--	--	--	--
		Exposure Point Total					8E-05					--
	Exposure Medium Total						8E-05					--
Medium Total							8E-05					--
Receptor Total							2E-04					Receptor HI Total 2E+01

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 2E+01

Total Liver HI Across All Media = 2E-02
Total Nervous System Effects HI Across All Media = 8E+00
Total Nasal/Respiratory Effects HI Across All Media = 4E+00
Total Other Effects HI Across All Media = 1E+01

TABLE 10.4a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	BENZO(A)PYRENE	2E-06	--	9E-07	3E-06	--	--	--	--	--
			Chemical Total	2E-06	--	9E-07	3E-06	--	--	--	--	
		Exposure Point Total						3E-06				--
	Exposure Medium Total						3E-06				--	
Medium Total							3E-06				--	
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	--	--	--	--	--	
		Exposure Point Total						--				--
	Exposure Medium Total						--				--	
Medium Total							--				--	
Shallow Ground Water	Shallow Ground Water	Exposure Unit 9	CHROMIUM	--	--	--	--	--	--	--	1E+00	1E+00
			BENZ(A)ANTHRACENE	--	--	9E-06	9E-06	--	--	--	--	
			BENZO(A)PYRENE	--	--	2E-04	2E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	2E-05	2E-05	--	--	--	--	
			Chemical Total	--	--	2E-04	2E-04	--	--	1E+00	1E+00	
	Exposure Point Total						2E-04				1E+00	
Exposure Medium Total						2E-04				1E+00		
Medium Total							2E-04				1E+00	
Receptor Total							2E-04	Receptor HI Total			1E+00	

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 1E+00

Total Other Effects HI Across All Media = 1E+00

TABLE 10.5 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Surveillance Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 2	2,3,7,8-TCDD Equivalent	3E-06	--	2E-07	4E-06	Developmental effects	6E-02	--	3E-03	7E-02
			Chemical Total	3E-06	--	2E-07	4E-06		6E-02	--	3E-03	7E-02
			Exposure Point Total				4E-06					7E-02
	Exposure Medium Total						4E-06					7E-02
Medium Total							4E-06					7E-02
Surface Soil	Outdoor Air	Exposure Unit 2	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	--		--	--	--	--
			Exposure Point Total				--					--
	Exposure Medium Total						--					--
Medium Total							--					--
Receptor Total							4E-06	Receptor HI Total				7E-02

Total Risk Across All Media = 4E-06

Total Hazard Across All Media = 7E-02

TABLE 10.6 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Ditch Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 3	None	--	--	--		--	--	--	--	--
			Chemical Total	--	--	--	--		--	--	--	--
		Exposure Point Total					--				--	
	Exposure Medium Total						--				--	
Medium Total							--				--	
Surface Soil	Outdoor Air	Exposure Unit 3	None	--	--	--		--	--	--	--	--
			Chemical Total	--	--	--	--		--	--	--	--
		Exposure Point Total					--				--	
	Exposure Medium Total						--				--	
Medium Total							--				--	
Surface Water	Surface Water	Exposure Unit 3	None	--	--	--		--	--	--	--	--
			Chemical Total	--	--	--	--		--	--	--	--
		Exposure Point Total					--				--	
	Exposure Medium Total						--				--	
Medium Total							--				--	
Receptor Total							0E+00	Receptor HI Total			0E+00	

Total Risk Across All Media = 0E+00

Total Hazard Across All Media = 0E+00

TABLE 10.7 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Railroad Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 4	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	--	--	--	--	--	
		Exposure Point Total						--				--
	Exposure Medium Total						--				--	
Medium Total							--				--	
Soil	Surface Soil	Exposure Unit 4	ARSENIC	5E-06	--	1E-06	7E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	7E-03	4E-02
			BENZO(A)PYRENE	8E-07	--	7E-07	1E-06	--	--	--	--	
			Chemical Total	6E-06	--	2E-06	8E-06	3E-02	--	7E-03	4E-02	
		Exposure Point Total						8E-06				4E-02
	Exposure Medium Total						8E-06				4E-02	
Medium Total							8E-06				4E-02	
Receptor Total							8E-06	Receptor HI Total			4E-02	

Total Risk Across All Media = 8E-06

Total Hazard Across All Media = 4E-02

TABLE 10.7a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Railroad Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	--		--	--	--	--
		Exposure Point Total						--				--
	Exposure Medium Total						--				--	
Medium Total							--				--	
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	1E-06	--	2E-07	1E-06	--	2E-02	--	5E-03	3E-02
			ARSENIC	2E-06	--	5E-07	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	3E-03	2E-02
			BENZ(A)ANTHRACENE	2E-06	--	2E-06	3E-06	--	--	--	--	
			BENZO(A)PYRENE	1E-05	--	1E-05	2E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	2E-06	--	2E-06	3E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	1E-06	--	1E-06	2E-06	--	--	--	--	
			Chemical Total	2E-05	--	2E-05	4E-05		4E-02	--	8E-03	5E-02
		Exposure Point Total						4E-05				5E-02
	Exposure Medium Total						4E-05				5E-02	
Medium Total							4E-05				5E-02	
Receptor Total							4E-05	Receptor HI Total			5E-02	

Total Risk Across All Media = 4E-05

Total Hazard Across All Media = 5E-02

TABLE 10.8 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 5	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	--	--	--	--	--	
		Exposure Point Total				--				--		
	Exposure Medium Total				--				--			
Medium Total							--				--	
Soil	Surface Soil	Exposure Unit 5	ARSENIC	8E-06	--	2E-06	1E-05	Hyperpigmentation (In); Vascular (V); PNS (N) Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	5E-02	--	1E-02	6E-02
			HIGHLY CHLORINATED PCBs	4E-06	--	6E-06	1E-05		3E-01	--	4E-01	7E-01
			BENZ(A)ANTHRACENE	9E-06	--	1E-05	2E-05		--	--	--	--
			BENZO(A)PYRENE	9E-05	--	1E-04	2E-04		--	--	--	--
			BENZO(B)FLUORANTHENE	8E-06	--	1E-05	2E-05		--	--	--	--
			BENZO(K)FLUORANTHENE	1E-06	--	1E-06	2E-06		--	--	--	--
			DIBENZ(A,H)ANTHRACENE	3E-05	--	3E-05	6E-05		--	--	--	--
			INDENO(1,2,3-CD)PYRENE	7E-06	--	9E-06	2E-05		--	--	--	--
			Chemical Total	2E-04	--	2E-04	3E-04			3E-01	--	4E-01
		Exposure Point Total				3E-04					8E-01	
	Exposure Medium Total				3E-04					8E-01		
Medium Total							3E-04				8E-01	
Receptor Total							3E-04				8E-01	
								Receptor HI Total			8E-01	

Total Risk Across All Media = 3E-04

Total Hazard Across All Media = 8E-01

Total Nervous System Effects HI Across All Media = 6E-02

Total Ocular Effects HI Across All Media = 7E-01

TABLE 10.9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 7	1,4-DICHLOROBENZENE	--	5E-06	--	5E-06	Liver	--	2E-03	--	2E-03	
			Chemical Total	--	5E-06	--	5E-06		--	2E-03	--	2E-03	
		Exposure Point Total					5E-06					2E-03	
	Exposure Medium Total					5E-06					2E-03		
Medium Total								5E-06					2E-03
Soil	Surface Soil	Exposure Unit 7	2,3,7,8-TCDD Equivalent	3E-05	--	8E-06	4E-05	Developmental effects	5E-01	--	2E-01	7E-01	
			ARSENIC	5E-06	--	1E-06	6E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	9E-03	4E-02	
			HIGHLY CHLORINATED PCBs	1E-06	--	1E-06	2E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	7E-02	--	1E-01	2E-01	
			LESS CHLORINATED PCBs	5E-07	--	7E-07	1E-06	Reduced birth weights (W)	1E-02	--	1E-02	2E-02	
			BENZ(A)ANTHRACENE	5E-06	--	6E-06	1E-05	--	--	--	--		
			BENZO(A)PYRENE	4E-05	--	6E-05	1E-04	--	--	--	--		
			BENZO(B)FLUORANTHENE	3E-06	--	4E-06	7E-06	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	8E-06	--	1E-05	2E-05	--	--	--	--		
			INDENO(1,2,3-CD)PYRENE	3E-06	--	3E-06	6E-06	--	--	--	--		
			Chemical Total	1E-04	--	9E-05	2E-04		6E-01	--	3E-01	9E-01	
		Exposure Point Total					2E-04					9E-01	
		Exposure Medium Total					2E-04					9E-01	
	Medium Total								2E-04				
Ground Water	Potable Water	Exposure Unit 8	ARSENIC	1E-04	--	--	1E-04	Hyperpigmentation (In); Vascular (V); PNS (N)	6E-01	--	--	6E-01	
			IRON	--	--	--	--	Gastrointestinal effects	1E+00	--	--	1E+00	
			THALLIUM	--	--	--	--	Hematological effects	2E+00	--	--	2E+00	
			4,4'-DDT	3E-06	--	--	3E-06	Liver lesions (H)	4E-02	--	--	4E-02	
			ALDRIN	4E-06	--	--	4E-06	Liver toxicity (H)	2E-02	--	--	2E-02	
			ALPHA-BHC	8E-06	--	--	8E-06	--	--	--	--		
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	4E+00	--	--	4E+00	
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E+00	--	--	3E+00	
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	2E+00	--	--	2E+00	
			4-METHYLPHENOL	--	--	--	--	--	3E+00	--	--	3E+00	
			BENZ(A)ANTHRACENE	3E-04	--	--	3E-04	--	--	--	--		
			BENZO(A)PYRENE	1E-03	--	--	1E-03	--	--	--	--		
			BENZO(B)FLUORANTHENE	1E-04	--	--	1E-04	--	--	--	--		
			BENZO(K)FLUORANTHENE	9E-06	--	--	9E-06	--	--	--	--		

TABLE 10.9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
			BIS(2-ETHYLHEXYL)PHTHALATE	1E-06	--	--	1E-06	Increased relative liver weight (H)	1E-02	--	--	1E-02		
			CHRYSENE	2E-06	--	--	2E-06	--	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	1E-04	--	--	1E-04	--	--	--	--	--		
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	4E+00	--	--	4E+00		
			INDENO(1,2,3-CD)PYRENE	4E-05	--	--	4E-05	--	--	--	--	--		
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	4E+00	--	--	4E+00		
			1,4-DICHLOROBENZENE	2E-05	--	--	2E-05	Liver	1E-01	--	--	1E-01		
			BENZENE	2E-03	--	--	2E-03	Reduced lymphocyte count	3E+01	--	--	3E+01		
			BROMODICHLOROMETHANE	1E-06	--	--	1E-06	Renal cytomegaly (R)	3E-03	--	--	3E-03		
			TETRACHLOROETHENE	1E-06	--	--	1E-06	Hepatotoxicity in mice (H), weight gain in rats	6E-04	--	--	6E-04		
			VINYL CHLORIDE	6E-06	--	--	6E-06	Liver cell polymorphism (H)	7E-03	--	--	7E-03		
			Chemical Total	4E-03	--	--	4E-03		5E+01	--	--	5E+01		
			Exposure Point Total							4E-03				5E+01
			Exposure Medium Total							4E-03				5E+01
	Medium Total							4E-03				5E+01		
Receptor Total							4E-03	Receptor HI Total			5E+01			

Total Risk Across All Media = 4E-03

Total Hazard Across All Media = 5E+01

Total Liver HI Across All Media = 2E-01
Total Kidney HI Across All Media = 3E-03
Total Nervous System Effects HI Across All Media = 6E+00
Total Lymphocyte Effects HI Across All Media = 3E+01
Total Nasal/Respiratory Effects HI Across All Media = 3E+00
Total Ocular Effects HI Across All Media = 2E-01
Total Other Effects HI Across All Media = 1E+01

TABLE 10.9a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	--	--	--	--	--		
		Exposure Point Total							--				
	Exposure Medium Total							--					
Medium Total								--					
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	2E-06	--	5E-07	2E-06	Developmental effects Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	9E-03	4E-02	
			ARSENIC	3E-06	--	1E-06	4E-06		2E-02	--	6E-03	3E-02	
			HIGHLY CHLORINATED PCBs	7E-07	--	9E-07	2E-06		--	5E-02	--	6E-02	1E-01
			BENZ(A)ANTHRACENE	2E-06	--	3E-06	5E-06		--	--	--	--	
			BENZO(A)PYRENE	2E-05	--	2E-05	4E-05		--	--	--	--	
			BENZO(B)FLUORANTHENE	2E-06	--	3E-06	6E-06		--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-06	3E-06		--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	5E-07	--	6E-07	1E-06		--	--	--	--	
			Chemical Total	3E-05	--	3E-05	6E-05		--	1E-01	--	8E-02	2E-01
		Exposure Point Total							6E-05				
	Exposure Medium Total							6E-05					
Medium Total								6E-05					
Receptor Total								6E-05	Receptor HI Total				
									2E-01				

Total Risk Across All Media = 6E-05

Total Hazard Across All Media = 2E-01

TABLE 10.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	1E-04	--	--	1E-04	Developmental effects	1E+01	--	--	1E+01
			ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	1E+00	--	--	1E+00
			ARSENIC	6E-06	--	--	6E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	--	1E-01
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	6E+00	--	--	6E+00
			HIGHLY CHLORINATED PCBs	6E-05	--	--	6E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+01	--	--	2E+01
			LESS CHLORINATED PCBs	5E-05	--	--	5E-05	Reduced birth weights (W)	4E+00	--	--	4E+00
			ALDRIN	2E-06	--	--	2E-06	Liver toxicity (H)	5E-02	--	--	5E-02
			DELTA-BHC	--	--	--	--	--	--	--	--	--
			DIELDRIN	3E-06	--	--	3E-06	Hepatic (H)	4E-02	--	--	4E-02
			HEPTACHLOR EPOXIDE	2E-06	--	--	2E-06	Increased liver-to-body weight ratio in males and females (H)	2E-01	--	--	2E-01
			BIS(2-ETHYLHEXYL)PHTHALATE	2E-06	--	--	2E-06	Increased relative liver weight (H)	6E-02	--	--	6E-02
			HEXACHLOROBENZENE	1E-06	--	--	1E-06	Hepatic (H)	9E-03	--	--	9E-03
			Chemical Total	3E-04			3E-04		4E+01			4E+01
	Exposure Point Total				3E-04					4E+01		
Exposure Medium Total				3E-04					4E+01			
Medium Total				3E-04					4E+01			
Sediment	Surface Sediment	Exposure Unit 6	2,3,7,8-TCDD Equivalent	2E-06	--	3E-06	5E-06	Developmental effects	2E-01	--	2E-01	4E-01
			ARSENIC	2E-06	--	3E-06	5E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-02	--	7E-02	1E-01
			CHROMIUM	--	--	--	--	None Reported (O)	1E+00	--	--	1E+00
			HIGHLY CHLORINATED PCBs	2E-07	--	1E-06	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-02	--	3E-01	4E-01
			BENZ(A)ANTHRACENE	1E-04	--	5E-04	6E-04	--	--	--	--	--
			BENZO(A)PYRENE	7E-04	--	4E-03	4E-03	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-04	--	8E-04	1E-03	--	--	--	--	--
			BENZO(K)FLUORANTHENE	4E-06	--	2E-05	2E-05	--	--	--	--	--

TABLE 10.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 6	CHRYSENE	1E-06	--	7E-06	9E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	8E-05	--	4E-04	5E-04	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	4E-05	--	2E-04	2E-04	--	--	--	--	
			Chemical Total	1E-03	--	6E-03	7E-03	2E+00	--	6E-01	2E+00	
		Exposure Point Total				7E-03				2E+00		
	Exposure Medium Total				7E-03				2E+00			
Medium Total							7E-03				2E+00	
Surface Soil	Outdoor Air	Exposure Unit 6	None	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	--	--	--	--	--	
			Exposure Point Total				--				--	
		Exposure Medium Total				--				--		
Medium Total							--				--	
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	1E-05	--	1E-05	2E-05	Developmental effects	8E-01	--	1E+00	2E+00
			ARSENIC	2E-06	--	2E-06	4E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	4E-02	--	5E-02	1E-01
			HIGHLY CHLORINATED PCBs	4E-07	--	2E-06	3E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	7E-01	8E-01
			LESS CHLORINATED PCBs	2E-07	--	1E-06	1E-06	Reduced birth weights (W)	2E-02	--	9E-02	1E-01
			BENZ(A)ANTHRACENE	5E-06	--	2E-06	7E-06	--	--	--	--	
			BENZO(A)PYRENE	6E-05	--	2E-05	8E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	4E-06	--	2E-06	6E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	1E-05	--	4E-06	1E-05	--	--	--	--	
			HEXACHLOROBENZENE	2E-07	--	8E-07	1E-06	Hepatic (H)	2E-03	--	8E-03	1E-02
			INDENO(1,2,3-CD)PYRENE	3E-06	--	1E-06	4E-06	--	--	--	--	
			Chemical Total	9E-05	--	5E-05	1E-04	1E+00	--	2E+00	3E+00	
			Exposure Point Total				1E-04				3E+00	
			Exposure Medium Total				1E-04				3E+00	
	Medium Total							1E-04			3E+00	

TABLE 10.10 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 6	BENZ(A)ANTHRACENE	--	--	1E-04	1E-04	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-03	1E-03	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-04	2E-04	--	--	--	--	--
			Chemical Total	--	--	1E-03	1E-03	--	--	--	--	--
		Exposure Point Total					1E-03					--
	Exposure Medium Total						1E-03					--
Medium Total							1E-03					--
Receptor Total							9E-03					Receptor HI Total 4E+01

Total Risk Across All Media = 9E-03

Total Hazard Across All Media = 4E+01

Total Liver HI Across All Media = 3E-01
Total Nervous System Effects HI Across All Media = 6E+00
Total Ocular Effects HI Across All Media = 2E+01
Total Other Effects HI Across All Media = 2E+01

TABLE 10.10a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	6E-07	--	8E-07	1E-06	--	5E-02	--	6E-02	1E-01
			ARSENIC	1E-06	--	2E-06	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	4E-02	7E-02
			HIGHLY CHLORINATED PCBs	2E-07	--	1E-06	2E-06	--	7E-02	--	4E-01	5E-01
			BENZ(A)ANTHRACENE	6E-06	--	3E-05	4E-05	--	--	--	--	--
			BENZO(A)PYRENE	4E-05	--	2E-04	3E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-06	--	3E-05	4E-05	--	--	--	--	--
			BENZO(K)FLUORANTHENE	2E-07	--	1E-06	1E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-06	--	2E-05	2E-05	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	1E-06	--	6E-06	7E-06	--	--	--	--	--
			Chemical Total	6E-05	--	3E-04	4E-04		2E-01	--	5E-01	7E-01
	Exposure Point Total						4E-04				7E-01	
	Exposure Medium Total						4E-04				7E-01	
	Medium Total						4E-04				7E-01	
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total						0E+00				0E+00
	Exposure Medium Total						0E+00				0E+00	
Medium Total						0E+00				0E+00		
Receptor Total						4E-04	Receptor HI Total			7E-01		

Total Risk Across All Media = 4E-04

Total Hazard Across All Media = 7E-01

TABLE 10.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	5E-04	--	--	5E-04	Developmental effects	7E+00	--	--	7E+00			
			ARSENIC	2E-05	--	--	2E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	--	1E-01			
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	4E+00	--	--	4E+00			
			HIGHLY CHLORINATED PCBs	2E-04	--	--	2E-04	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E+01	--	--	1E+01			
			LESS CHLORINATED PCBs	1E-04	--	--	1E-04	Reduced birth weights (W)	2E+00	--	--	2E+00			
			ALDRIN	7E-06	--	--	7E-06	Liver toxicity (H)	3E-02	--	--	3E-02			
			DIELDRIN	9E-06	--	--	9E-06	Hepatic (H)	3E-02	--	--	3E-02			
			HEPTACHLOR EPOXIDE	6E-06	--	--	6E-06	Increased liver-to-body weight ratio in males and females (H)	1E-01	--	--	1E-01			
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-06	--	--	5E-06	Increased relative liver weight (H)	4E-02	--	--	4E-02			
			HEXACHLOROBENZENE	3E-06	--	--	3E-06	Hepatic (H)	6E-03	--	--	6E-03			
			Chemical Total	8E-04	--	--	8E-04		2E+01	--	--	2E+01			
			Exposure Point Total							8E-04					2E+01
	Exposure Medium Total							8E-04					2E+01		
Medium Total							8E-04					2E+01			
Sediment	Surface Sediment	Exposure Unit 6	2,3,7,8-TCDD Equivalent	6E-07	--	6E-07	1E-06	Developmental effects	9E-03	--	9E-03	2E-02			
			ARSENIC	5E-07	--	6E-07	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-03	--	3E-03	6E-03			
			BENZ(A)ANTHRACENE	4E-06	--	2E-05	2E-05	--	--	--	--	--			
			BENZO(A)PYRENE	3E-05	--	1E-04	1E-04	--	--	--	--	--			
			BENZO(B)FLUORANTHENE	6E-06	--	3E-05	3E-05	--	--	--	--	--			
			DIBENZ(A,H)ANTHRACENE	3E-06	--	1E-05	2E-05	--	--	--	--	--			
			INDENO(1,2,3-CD)PYRENE	1E-06	--	6E-06	8E-06	--	--	--	--	--			
			Chemical Total	4E-05	--	2E-04	2E-04		1E-02	--	1E-02	2E-02			
			Exposure Point Total							2E-04					2E-02
			Exposure Medium Total							2E-04					2E-02
			Medium Total							2E-04					2E-02
			Surface Soil	Outdoor Air	Exposure Unit 6	None	--	--	--	--	--	--	--	--	--
	Chemical Total	--				--	--	--	--	--	--	--	--		
Exposure Point Total									--					--	
Exposure Medium Total								--					--		
Medium Total							--					--			

TABLE 10.11 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient								
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total				
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	3E-06	--	3E-06	6E-06	Developmental effects	4E-02	--	4E-02	9E-02				
			BENZ(A)ANTHRACENE	2E-07	--	9E-07	1E-06						--	--	--	--
			BENZO(A)PYRENE	2E-06	--	1E-05	1E-05						--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-07	--	2E-06	2E-06						--	--	--	--
			Chemical Total	6E-06	--	2E-05	2E-05							4E-02	--	4E-02
	Exposure Point Total				2E-05				9E-02							
	Exposure Medium Total				2E-05				9E-02							
Medium Total							2E-05				9E-02					
Surface Water	Surface Water	Exposure Unit 6	ANTIMONY	--	--	--	--	Longevity (M); Blood glucose (E); Cholesterol (E)	--	--	1E-03	1E-03				
			BENZ(A)ANTHRACENE	--	--	4E-05	4E-05						--	--	--	--
			BENZO(A)PYRENE	--	--	4E-04	4E-04						--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	6E-05	6E-05						--	--	--	--
			BENZENE	--	--	1E-06	1E-06						Reduced lymphocyte count	--	--	1E-02
	Chemical Total	--	--	5E-04	5E-04	--	--	1E-02	1E-02							
	Exposure Point Total				5E-04				1E-02							
Exposure Medium Total				5E-04				1E-02								
Medium Total							5E-04				1E-02					
Receptor Total							2E-03	Receptor HI Total			2E+01					

Total Risk Across All Media = 2E-03

Total Hazard Across All Media = 2E+01

Total Nervous System Effects HI Across All Media = 6E-03

Total Lymphocyte Effects HI Across All Media = 1E-02

Total Other Effects HI Across All Media = 2E+01

TABLE 10.11a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--		--	--	--	--
			Chemical Total		--		--		--		--	
		Exposure Point Total										
	Exposure Medium Total											
Medium Total												
Soil	Surface Soil	Exposure Unit 9	BENZ(A)ANTHRACENE	2E-07	--	1E-06	1E-06		--	--	--	--
			BENZO(A)PYRENE	2E-06	--	8E-06	9E-06		--	--	--	
			BENZO(B)FLUORANTHENE	2E-07	--	1E-06	1E-06		--	--	--	
			Chemical Total	2E-06		1E-05	1E-05		--		--	
		Exposure Point Total										
	Exposure Medium Total											
Medium Total												
Receptor Total								Receptor HI Total				

Total Risk Across All Media = 1E-05

Total Hazard Across All Media = 0E+00

TABLE 10.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 6	1,4-DICHLOROBENZENE	--	8E-06	--	8E-06	Liver	--	1E-02	--	1E-02
			Chemical Total	--	8E-06	--	8E-06	--	1E-02	--	1E-02	
		Exposure Point Total			8E-06			1E-02				
	Exposure Medium Total			8E-06			1E-02					
Medium Total				8E-06			1E-02					
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	8E-05	--	1E-04	2E-04	Developmental effects	7E+00	--	8E+00	1E+01
			ARSENIC	1E-05	--	2E-05	3E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	4E-01	--	4E-01	8E-01
			CADMIUM	--	--	--	--	Renal (R); Significant Proteinuria	5E-01	--	8E-01	1E+00
			HIGHLY CHLORINATED PCBs	3E-06	--	2E-05	2E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E+00	--	6E+00	7E+00
			LESS CHLORINATED PCBs	2E-06	--	9E-06	1E-05	Reduced birth weights (W)	1E-01	--	8E-01	9E-01
			DIELDRIN	2E-06	--	--	2E-06	Hepatic (H)	3E-02	--	--	3E-02
			BENZ(A)ANTHRACENE	4E-05	--	1E-05	6E-05	--	--	--	--	
			BENZO(A)PYRENE	5E-04	--	2E-04	7E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	4E-05	--	1E-05	5E-05	--	--	--	--	
			BENZO(K)FLUORANTHENE	3E-06	--	1E-06	4E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	9E-05	--	3E-05	1E-04	--	--	--	--	
			HEXACHLOROBENZENE	2E-06	--	7E-06	9E-06	Hepatic (H)	2E-02	--	6E-02	8E-02
			INDENO(1,2,3-CD)PYRENE	3E-05	--	9E-06	3E-05	--	--	--	--	
			Chemical Total	8E-04	--	4E-04	1E-03	--	9E+00	--	2E+01	2E+01
			Exposure Point Total			1E-03			2E+01			
		Exposure Medium Total			1E-03			2E+01				
	Medium Total				1E-03			2E+01				
Ground Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicity	2E+00	--	1E-02	2E+00
			ARSENIC	8E-05	--	5E-07	8E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	2E+00	--	1E-02	2E+00
			CHROMIUM	--	--	--	--	--	1E+00	--	8E-01	2E+00
			IRON	--	--	--	--	Gastrointestinal effects	4E+00	--	2E-02	4E+00
			THALLIUM	--	--	--	--	Hematological effects	6E+00	--	4E-02	6E+00
			4,4'-DDT	2E-06	--	2E-05	2E-05	Liver lesions (H)	1E-01	--	2E+00	2E+00
			ALDRIN	3E-06	--	3E-07	3E-06	Liver toxicity (H)	7E-02	--	6E-03	8E-02
			ALPHA-BHC	7E-06	--	--	7E-06	--	--	--	--	
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	1E+01	--	2E+00	1E+01
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E+01	--	--	1E+01
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	1E+00	--	1E-01	1E+00

TABLE 10.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	6E+00	--	5E-01	6E+00
			4-METHYLPHENOL	--	--	--	--	--	1E+01	--	1E+00	1E+01
			BENZ(A)ANTHRACENE	2E-04	--	2E-02	2E-02	--	--	--	--	--
			BENZO(A)PYRENE	8E-04	--	5E-01	5E-01	--	--	--	--	--
			BENZO(B)FLUORANTHENE	8E-05	--	6E-02	6E-02	--	--	--	--	--
			BENZO(K)FLUORANTHENE	7E-06	--	--	7E-06	--	--	--	--	--
			BIS(2-ETHYLHEXYL)PHTHALATE	8E-07	--	1E-06	2E-06	Increased relative liver weight (H)	3E-02	--	5E-02	8E-02
			CHRYSENE	1E-06	--	5E-04	5E-04	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-04	--	1E-01	1E-01	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E+01	--	--	1E+01
			FLUORANTHENE	--	--	--	--	Nephropathy, increased liver weights (H), hematological alterations (B), and clinical effects	3E-01	--	1E+00	2E+00
			HEXACHLOROBUTADIENE	4E-07	--	1E-06	1E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	3E-05	--	2E-02	2E-02	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+01	--	8E+00	2E+01
			PHENANTHRENE	--	--	--	--	--	9E-01	--	2E+00	3E+00
			1,4-DICHLOROBENZENE	1E-05	--	9E-06	2E-05	Liver	4E-01	--	3E-01	7E-01
			BENZENE	2E-03	--	3E-04	2E-03	Reduced lymphocyte count	9E+01	--	1E+01	1E+02
			BROMODICHLOROMETHANE	1E-06	--	8E-08	1E-06	Renal cytomegaly (R)	1E-02	--	8E-04	1E-02
			TETRACHLOROETHENE	9E-07	--	5E-07	1E-06	Hepatotoxicity in mice (H), weight gain in rats	2E-03	--	1E-03	3E-03
			TOLUENE	--	--	--	--	Increased kidney weight (R)	1E+00	--	3E-01	1E+00
			VINYL CHLORIDE	5E-06	--	2E-07	5E-06	Liver cell polymorphism (H)	2E-02	--	1E-03	2E-02
		Chemical Total			3E-03	--	7E-01	7E-01		2E+02	--	3E+01
Exposure Point Total							7E-01					2E+02
Exposure Medium Total							7E-01					2E+02

TABLE 10.12 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Shower Vapor	Exposure Unit 8	1,2,4-TRIMETHYLBENZENE	--	--	--	--	Hematological and Pulmonary	--	1E+02	--	1E+02
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	8E+00	--	8E+00
			1,4-DICHLOROBENZENE	--	9E-04	--	9E-04	Liver	--	1E+00	--	1E+00
			BENZENE	--	8E-03	--	8E-03	Decreased lymphocyte count	--	4E+02	--	4E+02
			BROMODICHLOROMETHANE	--	2E-05	--	2E-05	--	--	--	--	--
			CHLOROFORM	--	5E-05	--	5E-05	Hepatic effects	--	2E-01	--	2E-01
			VINYL CHLORIDE	--	2E-06	--	2E-06	Liver cell polymorphism	--	2E-02	--	2E-02
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	1E+01	--	1E+01
			Chemical Total	--	9E-03	--	9E-03		--	5E+02	--	5E+02
	Exposure Point Total			9E-03			5E+02					
Exposure Medium Total			9E-03			5E+02						
Medium Total			7E-01			7E+02						
Receptor Total			7E-01			Receptor HI Total	8E+02					

Total Risk Across All Media = 7E-01

Total Hazard Across All Media = 8E+02

Total Liver HI Across All Media =	6E+00
Total Kidney HI Across All Media =	1E+00
Total Nervous System Effects HI Across All Media =	3E+01
Total Lymphocyte Effects HI Across All Media =	5E+02
Total Nasal/Respiratory Effects HI Across All Media =	1E+02
Total Ocular Effects HI Across All Media =	7E+00
Total Other Effects HI Across All Media =	1E+02

TABLE 10.12a, RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--		
			Chemical Total	--	--	--	--	--	--	--	--			
		Exposure Point Total					--					--		
		Exposure Medium Total					--					--		
Medium Total								--					--	
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	5E-06	--	6E-06	1E-05	--	4E-01	--	5E-01	9E-01		
			ARSENIC	1E-05	--	1E-05	2E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-01	--	3E-01	6E-01		
			HIGHLY CHLORINATED PCBs	2E-06	--	1E-05	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	6E-01	--	4E+00	4E+00		
			BENZ(A)ANTHRACENE	5E-05	--	2E-05	7E-05	--	--	--	--			
			BENZO(A)PYRENE	4E-04	--	1E-04	5E-04	--	--	--	--			
			BENZO(B)FLUORANTHENE	5E-05	--	2E-05	7E-05	--	--	--	--			
			BENZO(K)FLUORANTHENE	2E-06	--	6E-07	2E-06	--	--	--	--			
			DIBENZ(A,H)ANTHRACENE	3E-05	--	1E-05	4E-05	--	--	--	--			
			INDENO(1,2,3-CD)PYRENE	1E-05	--	3E-06	1E-05	--	--	--	--			
			Chemical Total	5E-04	--	2E-04	7E-04		1E+00	--	4E+00	6E+00		
			Exposure Point Total					7E-04					6E+00	
			Exposure Medium Total					7E-04					6E+00	
		Medium Total								7E-04				
	Receptor Total								7E-04	Receptor HI Total				6E+00

Total Risk Across All Media = 7E-04

Total Hazard Across All Media = 6E+00

Total Nervous System Effects HI Across All Media = 6E-01

Total Ocular Effects HI Across All Media = 4E+00

Total Other Effects HI Across All Media = 9E-01

TABLE 10.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 6	1,4-DICHLOROBENZENE	--	1E-05	--	1E-05	Liver	--	3E-03	--	3E-03
			Chemical Total	--	1E-05	--	1E-05		--	3E-03	--	3E-03
		Exposure Point Total					1E-05					3E-03
	Exposure Medium Total					1E-05					3E-03	
Medium Total						1E-05					3E-03	
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	2E-05	--	5E-06	3E-05	Developmental effects	4E-01	--	8E-02	4E-01
			ARSENIC	4E-06	--	9E-07	5E-06	Hyperpigmentation (In); Vascular (V); PNS (N) Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E-02	--	5E-03	2E-02
			HIGHLY CHLORINATED PCBs	9E-07	--	1E-06	2E-06		5E-02	--	6E-02	1E-01
			BENZ(A)ANTHRACENE	2E-06	--	2E-06	3E-06	--	--	--	--	--
			BENZO(A)PYRENE	2E-05	--	2E-05	4E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	1E-06	3E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	4E-06	--	4E-06	7E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			Chemical Total	5E-05	--	4E-05	9E-05		4E-01	--	1E-01	6E-01
		Exposure Point Total					9E-05					6E-01
	Exposure Medium Total					9E-05					6E-01	
Medium Total						9E-05					6E-01	
Ground Water	Potable Water	Exposure Unit 8	ARSENIC	2E-04	--	9E-07	2E-04	Hyperpigmentation (In); Vascular (V); PNS (N)	9E-01	--	4E-03	9E-01
			IRON	--	--	--	--	Gastrointestinal effects	2E+00	--	8E-03	2E+00
			THALLIUM	--	--	--	--	Hematological effects	2E+00	--	1E-02	2E+00
			HIGHLY CHLORINATED PCBs	2E-06	--	--	2E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	--	1E-01
			4,4'-DDD	2E-07	--	2E-06	2E-06	--	--	--	--	--
			4,4'-DDT	4E-06	--	5E-05	6E-05	Liver lesions (H)	6E-02	--	7E-01	8E-01
			ALDRIN	7E-06	--	6E-07	7E-06	Liver toxicity (H)	3E-02	--	3E-03	3E-02
			ALPHA-BHC	1E-05	--	--	1E-05	--	--	--	--	--
			HEPTACHLOR EPOXIDE	1E-06	--	--	1E-06	Increased liver-to-body weight ratio in males and females (H)	2E-02	--	--	2E-02
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	6E+00	--	8E-01	6E+00
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	4E+00	--	--	4E+00
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	2E+00	--	2E-01	3E+00
			4-METHYLPHENOL	--	--	--	--		5E+00	--	4E-01	5E+00
			BENZ(A)ANTHRACENE	5E-04	--	6E-03	6E-03	--	--	--	--	--
			BENZO(A)PYRENE	2E-03	--	4E-02	4E-02	--	--	--	--	--
			BENZO(B)FLUORANTHENE	2E-04	--	4E-03	4E-03	--	--	--	--	--
			BENZO(K)FLUORANTHENE	2E-05	--	--	2E-05	--	--	--	--	--

TABLE 10.13 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
			BIS(2-ETHYLHEXYL)PHTHALATE	2E-06	--	3E-06	4E-06	Increased relative liver weight (H)	1E-02	--	2E-02	4E-02
			CHRYSENE	3E-06	--	4E-05	4E-05	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-04	--	8E-03	8E-03	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E+00	--	--	5E+00
			HEXACHLOROBUTADIENE	9E-07	--	2E-06	3E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	7E-05	--	2E-03	2E-03	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	5E+00	--	4E+00	9E+00
			PHENANTHRENE	--	--	--	--	--	4E-01	--	1E+00	1E+00
			1,2,4-TRICHLOROBENZENE	6E-07	--	8E-07	1E-06	Increased adrenal weights; vacuolization of zona fasciculata in the cortex	4E-02	--	5E-02	9E-02
			1,4-DICHLOROBENZENE	3E-05	--	2E-05	5E-05	Liver	2E-01	--	1E-01	3E-01
			BENZENE	4E-03	--	6E-04	4E-03	Reduced lymphocyte count	4E+01	--	6E+00	5E+01
			BROMODICHLOROMETHANE	2E-06	--	2E-07	2E-06	Renal cytomegaly (R)	4E-03	--	3E-04	4E-03
			TETRACHLOROETHENE	2E-06	--	1E-06	3E-06	Hepatotoxicity in mice (H), weight gain in rats	8E-04	--	5E-04	1E-03
			VINYL CHLORIDE	1E-05	--	5E-07	1E-05	Liver cell polymorphism (H)	1E-02	--	5E-04	1E-02
			Chemical Total	7E-03	--	6E-02	6E-02		7E+01	--	1E+01	9E+01
		Exposure Point Total							6E-02			
	Exposure Medium Total							6E-02				9E+01
	Shower Vapor	Exposure Unit 8	1,2,4-TRIMETHYLBENZENE	--	--	--	--	Hematological and Pulmonary	--	1E+01	--	1E+01
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	1E+00	--	1E+00
			1,4-DICHLOROBENZENE	--	6E-04	--	6E-04	Liver	--	2E-01	--	2E-01
			BENZENE	--	5E-03	--	5E-03	Decreased lymphocyte count	--	5E+01	--	5E+01
			BROMODICHLOROMETHANE	--	1E-05	--	1E-05	--	--	--	--	--
			CHLOROFORM	--	3E-05	--	3E-05	Hepatic effects	--	3E-02	--	3E-02
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	2E+00	--	2E+00
			Chemical Total	--	6E-03	--	6E-03		--	7E+01	--	7E+01
		Exposure Point Total							6E-03			
	Exposure Medium Total							6E-03				7E+01
Medium Total							7E-02				2E+02	
Receptor Total							7E-02	Receptor HI Total			2E+02	

Total Risk Across All Media = 7E-02

Total Hazard Across All Media = 2E+02

Total Liver HI Across All Media = 1E+00
Total Kidney HI Across All Media = 9E-02
Total Nervous System Effects HI Across All Media = 1E+01
Total Lymphocyte Effects HI Across All Media = 1E+02
Total Nasal/Respiratory Effects HI Across All Media = 2E+01
Total Ocular Effects HI Across All Media = 2E-01
Total Other Effects HI Across All Media = 3E+01

RAGS Table 10 CT Series

TABLE 10.1 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-05	--	--	2E-05	Developmental effects	1E+00	--	--	1E+00
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	1E+00	--	--	1E+00
			HIGHLY CHLORINATED PCBs	6E-06	--	--	6E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+00	--	--	2E+00
			LESS CHLORINATED PCBs	5E-06	--	--	5E-06	Reduced birth weights (W)	4E-01	--	--	4E-01
			Chemical Total	3E-05	--	--	3E-05		5E+00	--	--	5E+00
		Exposure Point Total				3E-05					5E+00	
	Exposure Medium Total				3E-05					5E+00		
Medium Total							3E-05				5E+00	
Sediment	Surface Sediment	Exposure Unit 1	BENZ(A)ANTHRACENE	2E-06	--	9E-06	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	4E-06	--	2E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	6E-07	--	3E-06	4E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	6E-07	--	3E-06	4E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	2E-07	--	1E-06	1E-06	--	--	--	--	--
			Chemical Total	7E-06	--	4E-05	4E-05	--	--	--	--	0E+00
	Exposure Point Total				4E-05					0E+00		
Exposure Medium Total				4E-05					0E+00			
Medium Total							4E-05				0E+00	
Soil	Surface Soil	Exposure Unit 1	BENZO(A)PYRENE	9E-07	--	5E-06	6E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	2E-07	--	1E-06	1E-06	--	--	--	--	--
			Chemical Total	1E-06	--	6E-06	7E-06		--	--	--	0E+00
		Exposure Point Total				7E-06					0E+00	
	Exposure Medium Total				7E-06					0E+00		
Medium Total							7E-06				0E+00	
Surface Soil	Outdoor Air	Exposure Unit 1	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total				0E+00					0E+00	
	Exposure Medium Total				0E+00					0E+00		
Medium Total							0E+00				0E+00	

TABLE 10.1 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Older Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	1E-05	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-04	1E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-05	2E-05	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	8E-06	8E-06	--	--	--	--	--
			Chemical Total	--	--	2E-04	2E-04		--	--	--	0E+00
		Exposure Point Total					2E-04					0E+00
	Exposure Medium Total						2E-04					0E+00
Medium Total							2E-04					0E+00
Receptor Total							2E-04				Receptor HI Total	5E+00

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 5E+00

Total Nervous System Effects HI Across All Media = 1E+00

Total Ocular Effects HI Across All Media = 2E+00

Total Other Effects HI Across All Media = 2E+00

TABLE 10.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 1	2,3,7,8-TCDD Equivalent	3E-05	--	--	3E-05	Developmental effects	2E+00	--	--	2E+00	
			ARSENIC	2E-06	--	--	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	--	3E-02	
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	1E+00	--	--	1E+00	
			HIGHLY CHLORINATED PCBs	1E-05	--	--	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+00	--	--	2E+00	
			LESS CHLORINATED PCBs	9E-06	--	--	9E-06	Reduced birth weights (W)	5E-01	--	--	5E-01	
			HEXACHLOROBENZENE	3E-07	--	--	3E-07	Hepatic (H)	2E-03	--	--	2E-03	
		Chemical Total	5E-05	--	--	5E-05		6E+00	--	--	6E+00		
		Exposure Point Total					5E-05					6E+00	
		Exposure Medium Total					5E-05					6E+00	
	Medium Total								5E-05				
Sediment	Surface Sediment	Exposure Unit 1	BENZ(A)ANTHRACENE	2E-06	--	4E-06	5E-06	--	--	--	--	--	
			BENZO(A)PYRENE	3E-07	--	8E-06	8E-06	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	1E-06	1E-06	--	4E-05	--	--	4E-05	
			DIBENZ(A,H)ANTHRACENE	--	--	1E-06	1E-06	--	7E-04	--	--	7E-04	
			Chemical Total	2E-06	--	1E-05	2E-05		--	--	--	7E-04	
		Exposure Point Total					2E-05					7E-04	
		Exposure Medium Total					2E-05					7E-04	
Medium Total								2E-05					7E-04
Soil	Surface Soil	Exposure Unit 1	BENZO(A)PYRENE	4E-07	--	2E-06	2E-06	--	--	--	--	--	
			Chemical Total	4E-07	--	2E-06	2E-06		--	--	--	0E+00	
		Exposure Point Total					2E-06					0E+00	
	Exposure Medium Total					2E-06					0E+00		
Medium Total								2E-06					0E+00
Surface Soil	Outdoor Air	Exposure Unit 1	None	--	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00	
		Exposure Point Total					0E+00					0E+00	
	Exposure Medium Total					0E+00					0E+00		
Medium Total								0E+00					0E+00

TABLE 10.2 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Trespasser
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	7E-06	7E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	6E-05	6E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	9E-06	9E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	4E-06	4E-06	--	--	--	--	--
			Chemical Total	--	--	8E-05	8E-05	--	--	--	--	0E+00
		Exposure Point Total				8E-05					0E+00	
	Exposure Medium Total				8E-05					0E+00		
Medium Total							8E-05					0E+00
Receptor Total							2E-04					6E+00
								Receptor HI Total				

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 6E+00

Total Liver HI Across All Media = 2E-03
Total Nervous System Effects HI Across All Media = 1E+00
Total Ocular Effects HI Across All Media = 2E+00
Total Other Effects HI Across All Media = 2E+00

TABLE 10.3 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Utility Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	BENZO(A)PYRENE	8E-07	--	7E-07	1E-06	--	--	--	--	--
			Chemical Total	8E-07		7E-07	1E-06		--			0E+00
		Exposure Point Total					1E-06					0E+00
	Exposure Medium Total						1E-06					0E+00
Medium Total							1E-06					0E+00
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	BENZO(A)PYRENE	1E-06	--	1E-06	2E-06	--	--	--	--	--
			Chemical Total	1E-06	--	1E-06	2E-06		--	--	--	0E+00
		Exposure Point Total					2E-06					0E+00
	Exposure Medium Total						2E-06					0E+00
Medium Total							2E-06					0E+00
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	CHROMIUM	--	1E-06	--	1E-06	--	--	9E-03	--	9E-03
			Chemical Total	--	1E-06	--	1E-06		--	9E-03	--	9E-03
		Exposure Point Total					1E-06					9E-03
	Exposure Medium Total						1E-06					9E-03
Medium Total							1E-06					9E-03
Ground Water	Shallow Ground Water	Exposure Unit 1	None	--	--	--	--		--	--	--	0E+00
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total					0E+00					0E+00
	Exposure Medium Total						0E+00					0E+00
Medium Total							0E+00					0E+00
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	1E-06	1E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	1E-05	1E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	2E-06	2E-06	--	--	--	--	--
			Chemical Total	--	--	1E-05	1E-05		--	--	--	0E+00
		Exposure Point Total					1E-05					0E+00
Medium Total							1E-05					0E+00
Receptor Total							2E-05					0E+00
											Receptor HI Total	9E-03

Total Risk Across All Media = 2E-05

Total Hazard Across All Media = 9E-03

TABLE 10.3a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Utility Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00	--	-	--	0E+00	
		Exposure Point Total					0E+00				0E+00	
	Exposure Medium Total					0E+00				0E+00		
Medium Total							0E+00				0E+00	
Soil	Surface Soil and	Exposure Unit 9	None	--	--	--	0E+00	--	--	--	--	
			Chemical Total	--	--	--	0E+00	--	-	--	0E+00	
		Exposure Point Total					0E+00				0E+00	
	Exposure Medium Total					0E+00				0E+00		
Medium Total							0E+00				0E+00	
Ground Water	Shallow Ground Water	Exposure Unit 9	BENZ(A)ANTHRACENE	--	--	2E-06	2E-06	--	--	--	--	
			BENZO(A)PYRENE	--	--	3E-05	3E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	4E-06	4E-06	--	--	--	--	
			Chemical Total	--	--	4E-05	4E-05	--	--	--	0E+00	
	Exposure Point Total					4E-05				0E+00		
Exposure Medium Total					4E-05				0E+00			
Medium Total							4E-05				0E+00	
Receptor Total							4E-05	Receptor HI Total			0E+00	

Total Risk Across All Media = 4E-05

Total Hazard Across All Media = 0E+00

TABLE 10.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment and Subsurface Sediment	Exposure Unit 1	2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	9E-01	--	2E+00	3E+00
			BENZ(A)ANTHRACENE	3E-06	--	7E-06	1E-05	--	--	--	--	--
			BENZO(A)PYRENE	7E-06	--	2E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	2E-06	4E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	3E-06	4E-06	--	--	--	--	--
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E+00	--	2E+00	3E+00
			INDENO(1,2,3-CD)PYRENE	4E-07	--	9E-07	1E-06	--	--	--	--	--
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	5E-01	--	1E+00	2E+00
			Chemical Total	1E-05	--	3E-05	4E-05		3E+00	--	5E+00	8E+00
		Exposure Point Total				4E-05					8E+00	
	Exposure Medium Total				4E-05					8E+00		
Medium Total						4E-05					8E+00	
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 1	CHROMIUM	--	4E-06	--	4E-06	--	--	3E-01	--	3E-01
			MANGANESE	--	--	--	--	Neurobehavioral changes (N, O)	--	2E+00	--	2E+00
			Chemical Total	--	4E-06	--	4E-06		--	2E+00	--	2E+00
			Exposure Point Total				4E-06					2E+00
			Exposure Medium Total				4E-06					2E+00
	Medium Total						4E-06					2E+00
Soil	Surface Soil and Subsurface Soil	Exposure Unit 1	2,3,7,8-TCDD Equivalent	2E-06	--	5E-08	2E-06	Developmental effects	8E-01	--	3E-02	9E-01
			BENZ(A)ANTHRACENE	2E-06	--	2E-07	2E-06	--	--	--	--	--
			BENZO(A)PYRENE	1E-05	--	1E-06	1E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	2E-07	2E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-07	1E-06	--	--	--	--	--
			Chemical Total	2E-05	--	2E-06	2E-05		8E-01	--	3E-02	9E-01
			Exposure Point Total				2E-05					9E-01
		Exposure Medium Total				2E-05					9E-01	
Medium Total						2E-05					9E-01	
Ground Water	Shallow Ground Water	Exposure Unit 1	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
			Exposure Point Total				0E+00					0E+00
		Exposure Medium Total				0E+00					0E+00	
Medium Total						0E+00					0E+00	

TABLE 10.4 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Construction Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 1	BENZ(A)ANTHRACENE	--	--	3E-06	3E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	3E-05	3E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	4E-06	4E-06	--	--	--	--	--
			INDENO(1,2,3-CD)PYRENE	--	--	2E-06	2E-06	--	--	--	--	--
			Chemical Total	--	--	4E-05	4E-05		--	--	--	0E+00
		Exposure Point Total					4E-05					0E+00
	Exposure Medium Total						4E-05					0E+00
Medium Total							4E-05					0E+00
Receptor Total							1E-04				Receptor HI Total	1E+01

Total Risk Across All Media = 1E-04

Total Hazard Across All Media = 1E+01

Total Nervous System Effects HI Across All Media = 2E+00
Total Nasal/Respiratory Effects HI Across All Media = 3E+00
Total Other Effects HI Across All Media = 6E+00

TABLE 10.4a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil and Subsurface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	0E+00	--	--	--	0E+00		
		Exposure Point Total							0E+00				
	Exposure Medium Total							0E+00					0E+00
Medium Total								0E+00					0E+00
Soil	Surface Soil and Subsurface Soil	Exposure Unit 9	BENZO(A)PYRENE	1E-06	--	1E-07	1E-06	--	--	--	--	--	
			Chemical Total	1E-06	--	1E-07	1E-06	--	0E+00	--	0E+00	0E+00	
		Exposure Point Total							1E-06				
	Exposure Medium Total							1E-06					0E+00
Medium Total								1E-06					0E+00
Ground Water	Shallow Ground Water	Exposure Unit 9	BENZ(A)ANTHRACENE	--	--	4E-06	4E-06	--	--	--	--	--	
			BENZO(A)PYRENE	--	--	8E-05	8E-05	--	--	--	--	--	
			BENZO(B)FLUORANTHENE	--	--	1E-05	1E-05	--	--	--	--	--	
			Chemical Total	--	--	1E-04	1E-04	--	--	--	0E+00	0E+00	
	Exposure Point Total							1E-04					0E+00
Exposure Medium Total							1E-04					0E+00	
Medium Total								1E-04					0E+00
Receptor Total								1E-04	Receptor HI Total				0E+00

Total Risk Across All Media = 1E-04

Total Hazard Across All Media = 0E+00

TABLE 10.5 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Surveillance Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Exposure Unit 2	2,3,7,8-TCDD Equivalent	1E-06	--	9E-09	1E-06	--	6E-02	--	5E-04	6E-02
			Chemical Total	1E-06	--	9E-09	1E-06		6E-02	--	5E-04	6E-02
			Exposure Point Total				1E-06					6E-02
	Exposure Medium Total						1E-06					6E-02
	Medium Total						1E-06					6E-02
Surface Soil	Outdoor Air	Exposure Unit 2	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
			Exposure Point Total				0E+00					0E+00
	Exposure Medium Total						0E+00					0E+00
	Medium Total						0E+00					0E+00
Receptor Total							1E-06	Receptor HI Total				6E-02

Total Risk Across All Media = 1E-06

Total Hazard Across All Media = 6E-02

TABLE 10.6 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Ditch Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Surface Sediment	Exposure Unit 3	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total					0E+00					0E+00
	Exposure Medium Total						0E+00					0E+00
Medium Total							0E+00					0E+00
Surface Sediment	Outdoor Air	Exposure Unit 3	None	--	--	--	--	--	--	--	--	0E+00
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total					0E+00					0E+00
	Exposure Medium Total						0E+00					0E+00
Medium Total							0E+00					0E+00
Surface Water	Surface Water	Exposure Unit 3	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total					0E+00					0E+00
	Exposure Medium Total						0E+00					0E+00
Medium Total							0E+00					0E+00
Receptor Total							0E+00	Receptor HI Total				0E+00

Total Risk Across All Media = 0E+00

Total Hazard Across All Media = 0E+00

TABLE 10.7 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Railroad Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 4	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total						0E+00				0E+00
	Exposure Medium Total						0E+00				0E+00	
Medium Total							0E+00				0E+00	
Soil	Surface Soil	Exposure Unit 4	ARSENIC	2E-06	--	1E-07	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	2E-03	3E-02
			Chemical Total	2E-06	--	1E-07	2E-06		3E-02	--	2E-03	3E-02
		Exposure Point Total						2E-06				3E-02
	Exposure Medium Total						2E-06				3E-02	
Medium Total							2E-06				3E-02	
Receptor Total							2E-06	Receptor HI Total			3E-02	

Total Risk Across All Media = 2E-06

Total Hazard Across All Media = 3E-02

TABLE 10.7a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Railroad Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00	
		Exposure Point Total						0E+00				0E+00	
	Exposure Medium Total						0E+00				0E+00		
Medium Total							0E+00				0E+00		
Soil	Surface Soil	Exposure Unit 9	BENZO(A)PYRENE	4E-06	--	1E-06	5E-06	--	--	--	--	--	
			Chemical Total	4E-06	--	1E-06	5E-06		--	--	--	0E+00	
		Exposure Point Total						5E-06				0E+00	
	Exposure Medium Total						5E-06				0E+00		
Medium Total							5E-06				0E+00		
Receptor Total							5E-06	Receptor HI Total					0E+00

Total Risk Across All Media = 5E-06

Total Hazard Across All Media = 0E+00

TABLE 10.8 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 5	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00	--	--	--	0E+00	
		Exposure Point Total						0E+00				0E+00
	Exposure Medium Total						0E+00				0E+00	
Medium Total							0E+00				0E+00	
Soil	Surface Soil	Exposure Unit 5	ARSENIC	1E-06	--	2E-07	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	4E-03	3E-02
			HIGHLY CHLORINATED PCBs	7E-07	--	6E-07	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	1E-01	--	1E-01	2E-01
			BENZ(A)ANTHRACENE	1E-06	--	1E-06	3E-06	--	--	--	--	
			BENZO(A)PYRENE	1E-05	--	1E-05	3E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	1E-06	--	1E-06	2E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	4E-06	--	3E-06	7E-06	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	1E-06	--	1E-06	2E-06	--	--	--	--	
			Chemical Total	2E-05	--	2E-05	4E-05		1E-01	--	1E-01	3E-01
		Exposure Point Total						4E-05				3E-01
	Exposure Medium Total						4E-05				3E-01	
Medium Total							4E-05				3E-01	
Receptor Total							4E-05	Receptor HI Total			3E-01	

Total Risk Across All Media = 4E-05

Total Hazard Across All Media = 3E-01

TABLE 10.9 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 7	1,4-DICHLOROBENZENE	--	2E-06	--	2E-06	Liver	--	1E-03	--	1E-03
			Chemical Total	--	2E-06	--	2E-06		--	1E-03	--	1E-03
			Exposure Point Total				2E-06					1E-03
	Exposure Medium Total						2E-06				1E-03	
Medium Total							2E-06				1E-03	
Soil	Surface Soil	Exposure Unit 7	2,3,7,8-TCDD Equivalent	4E-06	--	8E-07	5E-06	Developmental effects	2E-01	--	4E-02	3E-01
			BENZ(A)ANTHRACENE	7E-07	--	6E-07	1E-06	--	--	--	--	
			BENZO(A)PYRENE	7E-06	--	6E-06	1E-05	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	1E-06	--	1E-06	2E-06	--	--	--	--	
			DIBENZOFURAN	1E-06	--	--	1E-06	Reduced length and organ weight. Excess abdominal fat (O).	2E-03	--	1E-03	3E-03
			Chemical Total	1E-05	--	9E-06	2E-05		2E-01	--	4E-02	3E-01
		Exposure Point Total				2E-05				3E-01		
Exposure Medium Total						2E-05				3E-01		
Medium Total							2E-05				3E-01	
Water	Potable Water	Exposure Unit 8	ARSENIC	3E-05	--	--	3E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-01	--	--	5E-01
			THALLIUM	--	--	--	--	Hematological effects	1E+00	--	--	1E+00
			ALDRIN	1E-06	--	--	1E-06	Liver toxicity (H)	2E-02	--	--	2E-02
			ALPHA-BHC	3E-06	--	--	3E-06	--	--	--	--	
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	3E+00	--	--	3E+00
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	3E+00	--	--	3E+00
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	1E+00	--	--	1E+00
			4-METHYLPHENOL	--	--	--	--	--	3E+00	--	--	3E+00
			BENZ(A)ANTHRACENE	9E-05	--	--	9E-05	--	--	--	--	
			BENZO(A)PYRENE	3E-04	--	--	3E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	3E-05	--	--	3E-05	--	--	--	--	
			BENZO(K)FLUORANTHENE	3E-06	--	--	3E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	5E-05	--	--	5E-05	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	3E+00	--	--	3E+00
			INDENO(1,2,3-CD)PYRENE	1E-05	--	--	1E-05	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	3E+00	--	--	3E+00

TABLE 10.9 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Commercial/Industrial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Water	Potable Water	Exposure Unit 8	1,4-DICHLOROBENZENE	6E-06	--	--	6E-06	Liver	1E-01	--	--	1E-01
			BENZENE	7E-04	--	--	7E-04	Reduced lymphocyte count	2E+01	--	--	2E+01
			VINYL CHLORIDE	2E-06	--	--	--	Liver cell polymorphism (H)	6E-03	--	--	6E-03
			Chemical Total	1E-03	--	--	1E-03		4E+01	--	--	4E+01
		Exposure Point Total			1E-03					4E+01		
	Exposure Medium Total			1E-03					4E+01			
Medium Total							1E-03					4E+01
Receptor Total							1E-03				Receptor HI Total	4E+01

Total Risk Across All Media = 1E-03

Total Hazard Across All Media = 4E+01

Total Liver HI Across All Media = 1E-01
 Total Nervous System Effects HI Across All Media = 5E+00
 Total Lymphocyte Effects HI Across All Media = 2E+01
 Total Nasal/Respiratory Effects HI Across All Media = 3E+00
 Total Other Effects HI Across All Media = 1E+01

TABLE 10.9a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Commercial/Industrial Worker
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00	--	--	--	0E+00	
		Exposure Point Total				0E+00				0E+00		
	Exposure Medium Total				0E+00				0E+00			
Medium Total							0E+00				0E+00	
Soil	Surface Soil	Exposure Unit 9	BENZO(A)PYRENE	3E-06	--	2E-06	5E-06	--	--	--	--	
			Chemical Total	3E-06	--	2E-06	5E-06	--	--	--	0E+00	
		Exposure Point Total				5E-06				0E+00		
	Exposure Medium Total				5E-06				0E+00			
Medium Total							5E-06				0E+00	
Receptor Total							5E-06	Receptor HI Total				0E+00

Total Risk Across All Media = 5E-06

Total Hazard Across All Media = 0E+00

TABLE 10.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Recreational Visitor
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	3E-05	--	--	3E-05	Developmental effects	2E+00	--	--	2E+00
			ARSENIC	2E-06	--	--	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	5E-02	--	--	5E-02
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	2E+00	--	--	2E+00
			HIGHLY CHLORINATED PCBs	1E-05	--	--	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E+00	--	--	3E+00
			LESS CHLORINATED PCBs	1E-05	--	--	1E-05	Reduced birth weights (W)	8E-01	--	--	8E-01
			Chemical Total	5E-05	--	--	5E-05		9E+00	--	--	9E+00
	Exposure Point Total					5E-05				9E+00		
	Exposure Medium Total						5E-05				9E+00	
	Medium Total							5E-05				9E+00
Sediment	Surface Sediment	Exposure Unit 6	BENZ(A)ANTHRACENE	1E-04	--	3E-05	1E-04	--	--	--	--	
			BENZO(A)PYRENE	7E-04	--	2E-04	9E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	2E-04	--	4E-05	2E-04	--	--	--	--	
			BENZO(K)FLUORANTHENE	4E-06	--	1E-06	5E-06	--	--	--	--	
			CHRYSENE	1E-06	--	4E-07	2E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	8E-05	--	2E-05	1E-04	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	4E-05	--	9E-06	5E-05	--	--	--	--	
			Chemical Total	1E-03	--	3E-04	1E-03		--	--	--	0E+00
	Exposure Point Total					1E-03				0E+00		
Exposure Medium Total						1E-03				0E+00		
Medium Total							1E-03				0E+00	
Surface Soil	Outdoor Air	Exposure Unit 6	None	--	--	--	--	--	--	--	--	
		Chemical Total	--	--	--	0E+00		--	--	--	0E+00	
		Exposure Point Total					0E+00				0E+00	
	Exposure Medium Total						0E+00				0E+00	
Medium Total							0E+00				0E+00	
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	2E-06	--	7E-07	3E-06	Developmental effects	2E-01	--	5E-02	2E-01
			BENZ(A)ANTHRACENE	9E-07	--	1E-06	2E-06	--	--	--	--	
			BENZO(A)PYRENE	1E-05	--	2E-05	3E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	8E-07	--	1E-06	2E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	2E-06	--	3E-06	5E-06	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	6E-07	--	8E-07	1E-06	--	--	--	--	
			Chemical Total	2E-05	--	2E-05	4E-05		2E-01	--	5E-02	2E-01
			Exposure Point Total					4E-05				2E-01
	Exposure Medium Total						4E-05				2E-01	
Medium Total							4E-05				2E-01	

TABLE 10.10 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Water	Surface Water	Exposure Unit 6	BENZ(A)ANTHRACENE	--	--	7E-05	7E-05	--	--	--	--	--
			BENZO(A)PYRENE	--	--	6E-04	6E-04	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	9E-05	9E-05	--	--	--	--	--
			Chemical Total	--	--	8E-04	8E-04	--	--	--	--	0E+00
		Exposure Point Total					8E-04					0E+00
	Exposure Medium Total						8E-04					0E+00
Medium Total							8E-04					0E+00
Receptor Total							2E-03					9E+00

Total Risk Across All Media = 2E-03

Total Hazard Across All Media = 9E+00

Total Nervous System Effects HI Across All Media =	2E+00
Total Ocular Effects HI Across All Media =	3E+00
Total Other Effects HI Across All Media =	3E+00

TABLE 10.10a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00	--	--	--	0E+00	
		Exposure Point Total		--	--	--	0E+00					0E+00
	Exposure Medium Total		--	--	--	0E+00					0E+00	
Medium Total				--	--	--	0E+00					0E+00
Soil	Surface Soil	Exposure Unit 9	BENZ(A)ANTHRACENE	1E-06	--	2E-06	3E-06	--	--	--	--	--
			BENZO(A)PYRENE	8E-06	--	1E-05	2E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	1E-06	--	2E-06	3E-06	--	--	--	--	--
			DIBENZ(A,H)ANTHRACENE	7E-07	--	1E-06	2E-06	--	--	--	--	--
		Chemical Total	1E-05	--	2E-05	3E-05	--	--	--	--	0E+00	
	Exposure Point Total					3E-05					0E+00	
Exposure Medium Total					3E-05					0E+00		
Medium Total							3E-05					0E+00
Receptor Total							3E-05	Receptor HI Total				0E+00

Total Risk Across All Media = 3E-05

Total Hazard Across All Media = 0E+00

TABLE 10.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Onondaga Lake Fish Tissue	Fish Tissue	Exposure Unit 6	2,3,7,8-TCDD Equivalent	3E-05	--	--	3E-05	Developmental effects	2E+00	--	--	2E+00
			ARSENIC	2E-06	--	--	2E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	3E-02	--	--	3E-02
			MERCURY (AS METHYLMERCURY)	--	--	--	--	Developmental neuropsychological impairment (N)	1E+00	--	--	1E+00
			HIGHLY CHLORINATED PCBs	1E-05	--	--	1E-05	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	2E+00	--	--	2E+00
			LESS CHLORINATED PCBs	9E-06	--	--	9E-06	Reduced birth weights (W)	5E-01	--	--	5E-01
		Chemical Total	5E-05	--	--	5E-05		6E+00	--	--	6E+00	
		Exposure Point Total				5E-05					6E+00	
		Exposure Medium Total				5E-05					6E+00	
Medium Total							5E-05				6E+00	
Sediment	Surface Sediment	Exposure Unit 6	BENZ(A)ANTHRACENE	4E-07	--	2E-06	2E-06	--	--	--	--	
			BENZO(A)PYRENE	3E-06	--	1E-05	2E-05	--	--	--	--	
			BENZO(B)FLUORANTHENE	7E-07	--	3E-06	4E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	4E-07	--	2E-06	2E-06	--	--	--	--	
		Chemical Total	5E-06	--	2E-05	3E-05		--	--	--	0E+00	
		Exposure Point Total				3E-05					0E+00	
	Exposure Medium Total				3E-05					0E+00		
Medium Total							3E-05				0E+00	
Soil	Surface Soil	Exposure Unit 6	BENZO(A)PYRENE	3E-07	--	1E-06	1E-06	--	--	--	--	
			Chemical Total	3E-07	--	1E-06	1E-06		--	--	--	0E+00
			Exposure Point Total				1E-06					0E+00
		Exposure Medium Total				1E-06					0E+00	
Medium Total							1E-06				0E+00	
Surface Soil	Outdoor Air	Exposure Unit 6	None	--	--	--	--	--	--	--	--	
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
			Exposure Point Total				0E+00					0E+00
		Exposure Medium Total				0E+00					0E+00	
Medium Total							0E+00				0E+00	

TABLE 10.11 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Surface Water	Exposure Unit 6	BENZ(A)ANTHRACENE	--	--	7E-06	7E-06	--	--	--	--	--
			BENZO(A)PYRENE	--	--	6E-05	6E-05	--	--	--	--	--
			BENZO(B)FLUORANTHENE	--	--	9E-06	9E-06	--	--	--	--	--
			Chemical Total	--	--	8E-05	8E-05	--	--	--	--	0E+00
		Exposure Point Total					8E-05					0E+00
	Exposure Medium Total						8E-05					0E+00
Medium Total							8E-05					0E+00
Receptor Total							2E-04					6E+00
								Receptor HI Total				

Total Risk Across All Media = 2E-04

Total Hazard Across All Media = 6E+00

Total Nervous System Effects HI Across All Media = 1E+00

Total Ocular Effects HI Across All Media = 2E+00

Total Other Effects HI Across All Media = 2E+00

TABLE 10.11a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Current/Future
Receptor Population:	Recreational Visitor
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00	--	--	--	0E+00	
		Exposure Point Total				0E+00				0E+00		
	Exposure Medium Total				0E+00				0E+00			
	Medium Total					0E+00				0E+00		
Soil	Surface Soil	Exposure Unit 9	BENZO(A)PYRENE	2E-07	--	9E-07	1E-06	--	--	--	--	
			Chemical Total	2E-07	--	9E-07	1E-06	--	--	--	0E+00	
		Exposure Point Total				1E-06				0E+00		
	Exposure Medium Total				1E-06				0E+00			
	Medium Total					1E-06				0E+00		
Receptor Total							1E-06	Receptor HI Total			0E+00	

Total Risk Across All Media = 1E-06

Total Hazard Across All Media = 0E+00

TABLE 10.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient					
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total	
Surface Soil	Outdoor Air	Exposure Unit 6	1,4-DICHLOROBENZENE	--	8E-06	--	8E-06	Liver	--	1E-02	--	1E-02	
			Chemical Total	--	8E-06	--	8E-06		--	1E-02	--	1E-02	
		Exposure Point Total					8E-06					1E-02	
	Exposure Medium Total					8E-06					1E-02		
Medium Total								8E-06					1E-02
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	4E-05	--	1E-06	4E-05	Developmental effects	3E+00	--	1E-01	3E+00	
			ARSENIC	7E-06	--	2E-07	7E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-01	--	6E-03	2E-01	
			HIGHLY CHLORINATED PCBs	2E-06	--	3E-07	2E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	5E-01	--	8E-02	6E-01	
			BENZ(A)ANTHRACENE	2E-05	--	3E-06	2E-05	--	--	--	--		
			BENZO(A)PYRENE	2E-04	--	3E-05	3E-04	--	--	--	--		
			BENZO(B)FLUORANTHENE	2E-05	--	3E-06	2E-05	--	--	--	--		
			BENZO(G,H,I)PERYLENE	--	--	--	--	--	1E-03	--	2E-04	1E-03	
			BENZO(K)FLUORANTHENE	2E-06	--	2E-07	2E-06	--	--	--	--		
			DIBENZ(A,H)ANTHRACENE	4E-05	--	6E-06	5E-05	--	--	--	--		
			INDENO(1,2,3-CD)PYRENE	1E-05	--	2E-06	1E-05	--	--	--	--		
			Chemical Total	4E-04	--	5E-05	4E-04		7E-01	--	8E-02	8E-01	
			Exposure Point Total					4E-04					8E-01
	Exposure Medium Total					4E-04					8E-01		
Medium Total								4E-04					8E-01
Ground Water	Potable Water	Exposure Unit 8	ALUMINUM	--	--	--	--	Neurotoxicity	2E+00	--	3E-03	2E+00	
			ARSENIC	8E-05	--	2E-07	8E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	2E+00	--	4E-03	2E+00	
			CHROMIUM	--	--	--	--	--	1E+00	--	3E-01	2E+00	
			IRON	--	--	--	--	Gastrointestinal effects	4E+00	--	8E-03	4E+00	
			THALLIUM	--	--	--	--	Hematological effects	6E+00	--	1E-02	6E+00	
			4,4'-DDT	2E-06	--	1E-05	2E-05	Liver lesions (H)	1E-01	--	9E-01	1E+00	
			ALDRIN	3E-06	--	2E-07	3E-06	Liver toxicity (H)	7E-02	--	4E-03	8E-02	
			ALPHA-BHC	7E-06	--	--	7E-06	--	--	--	--	--	
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	1E+01	--	1E+00	1E+01	
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	1E+01	--	--	1E+01	
			2-METHYLPHENOL	--	--	--	--	Decreased body weights and neurotoxicity	1E+00	--	7E-02	1E+00	
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	6E+00	--	3E-01	6E+00	
			4-METHYLPHENOL	--	--	--	--	--	1E+01	--	6E-01	1E+01	
			BENZ(A)ANTHRACENE	2E-04	--	1E-02	1E-02	--	--	--	--		
			BENZO(A)PYRENE	6E-04	--	6E-02	6E-02	--	--	--	--		
			BENZO(B)FLUORANTHENE	6E-05	--	7E-03	7E-03	--	--	--	--		
			BENZO(K)FLUORANTHENE	5E-06	--	--	5E-06	--	--	--	--		

TABLE 10.12 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Ground Water	Potable Water	Exposure Unit 8	BIS(2-ETHYLHEXYL)PHTHALATE	8E-07	--	7E-07	1E-06	Increased relative liver weight (H)	3E-02	--	3E-02	6E-02		
			CHRYSENE	1E-06	--	6E-05	6E-05	--	--	--	--			
			DIBENZ(A,H)ANTHRACENE	9E-05	--	1E-02	1E-02	--	--	--	--			
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	1E+01	--	--	1E+01		
			HEXACHLOROBUTADIENE	4E-07	--	6E-07	1E-06	--	--	--	--			
			INDENO(1,2,3-CD)PYRENE	3E-05	--	3E-03	3E-03	--	--	--	--			
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	1E+01	--	5E+00	2E+01		
			PHENANTHRENE	--	--	--	--	--	9E-01	--	1E+00	2E+00		
			1,4-DICHLOROBENZENE	1E-05	--	5E-06	2E-05	Liver	4E-01	--	2E-01	6E-01		
			BENZENE	2E-03	--	1E-04	2E-03	Reduced lymphocyte count	9E+01	--	8E+00	1E+02		
			BROMODICHLOROMETHANE	1E-06	--	5E-08	1E-06	Renal cytomegaly (R)	1E-02	--	4E-04	1E-02		
			TETRACHLOROETHENE	9E-07	--	3E-07	1E-06	Hepatotoxicity in mice (H), weight gain in rats	2E-03	--	6E-04	3E-03		
			TOLUENE	--	--	--	--	Increased kidney weight (R)	1E+00	--	2E-01	1E+00		
			VINYL CHLORIDE	5E-06	--	1E-07	5E-06	Liver cell polymorphism (H)	2E-02	--	7E-04	2E-02		
			Chemical Total	3E-03	--	9E-02	1E-01		2E+02	--	2E+01	2E+02		
	Exposure Point Total											2E+02		
	Exposure Medium Total											2E+02		
	Shower Vapor	Exposure Unit 8	1,2,4-TRIMETHYLBENZENE	--	--	--	--	Hematological and Pulmonary	--	3E+01	--	3E+01		
			1,2-DICHLOROBENZENE	--	--	--	--	--	--	2E+00	--	2E+00		
			1,4-DICHLOROBENZENE	--	3E-04	--	3E-04	Liver	--	4E-01	--	4E-01		
			BENZENE	--	3E-03	--	3E-03	Decreased lymphocyte count	--	1E+02	--	1E+02		
			BROMODICHLOROMETHANE	--	6E-06	--	6E-06	--	--	--	--	--		
			CHLOROFORM	--	2E-05	--	2E-05	Hepatic effects	--	8E-02	--	8E-02		
			XYLENES, TOTAL	--	--	--	--	Impaired motor coordination (decreased rotarod performance)	--	5E+00	--	5E+00		
			Chemical Total	--	3E-03	--	3E-03		--	2E+02	--	2E+02		
			Exposure Point Total											2E+02
			Exposure Medium Total											2E+02
			Medium Total											4E+02
			Receptor Total											4E+02

Total Risk Across All Media = 1E-01

Total Hazard Across All Media = 4E+02

Total Liver HI Across All Media = 2E+00
Total Kidney HI Across All Media = 1E+00
Total Nervous System Effects HI Across All Media = 2E+01
Total Lymphocyte Effects HI Across All Media = 2E+02
Total Nasal/Respiratory Effects HI Across All Media = 4E+01
Total Ocular Effects HI Across All Media = 6E-01
Total Other Effects HI Across All Media = 6E+01

TABLE 10.12a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00		--	--	--	0E+00
		Exposure Point Total					0E+00					0E+00
	Exposure Medium Total					0E+00					0E+00	
Medium Total							0E+00					0E+00
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	3E-06	--	9E-08	3E-06	--	2E-01	--	7E-03	2E-01
			ARSENIC	5E-06	--	2E-07	5E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-01	--	4E-03	1E-01
			HIGHLY CHLORINATED PCBs	1E-06	--	2E-07	1E-06	Ocular exudate (OC), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	3E-01	--	5E-02	3E-01
			BENZ(A)ANTHRACENE	3E-05	--	4E-06	3E-05	--	--	--	--	
			BENZO(A)PYRENE	2E-04	--	3E-05	2E-04	--	--	--	--	
			BENZO(B)FLUORANTHENE	3E-05	--	4E-06	3E-05	--	--	--	--	
			BENZO(K)FLUORANTHENE	9E-07	--	1E-07	1E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	2E-05	--	2E-06	2E-05	--	--	--	--	
			INDENO(1,2,3-CD)PYRENE	5E-06	--	7E-07	6E-06	--	--	--	--	
			Chemical Total	3E-04	--	4E-05	3E-04		6E-01	--	6E-02	7E-01
		Exposure Point Total					3E-04					7E-01
		Exposure Medium Total					3E-04					7E-01
		Medium Total						3E-04				
	Receptor Total						3E-04	Receptor HI Total				7E-01

Total Risk Across All Media = 3E-04

Total Hazard Across All Media = 7E-01

TABLE 10.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 6	1,4-DICHLOROBENZENE	--	3E-06	--	3E-06	Liver	--	3E-03	--	3E-03
			Chemical Total	--	3E-06	--	3E-06		--	3E-03	--	3E-03
		Exposure Point Total				3E-06					3E-03	
	Exposure Medium Total				3E-06					3E-03		
Medium Total							3E-06					3E-03
Soil	Surface Soil	Exposure Unit 6	2,3,7,8-TCDD Equivalent	7E-06	--	2E-07	7E-06	Developmental effects	4E-01	--	1E-02	4E-01
			ARSENIC	1E-06	--	4E-08	1E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	2E-02	--	7E-04	2E-02
			BENZO(A)PYRENE	6E-06	--	9E-07	7E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	1E-06	--	2E-07	1E-06	--	--	--	--	
			Chemical Total	1E-05	--	1E-06	2E-05		4E-01	--	1E-02	4E-01
	Exposure Point Total						2E-05					4E-01
Exposure Medium Total							2E-05				4E-01	
Medium Total							2E-05				4E-01	
Ground Water	Potable Water	Exposure Unit 8	ARSENIC	5E-05	--	1E-07	5E-05	Hyperpigmentation (In); Vascular (V); PNS (N)	9E-01	--	2E-03	9E-01
			IRON	--	--	--	--	Gastrointestinal effects	2E+00	--	3E-03	2E+00
			THALLIUM	--	--	--	--	Hematological effects	2E+00	--	5E-03	2E+00
			4,4'-DDT	1E-06	--	1E-05	1E-05	Liver lesions (H)	6E-02	--	5E-01	5E-01
			ALDRIN	2E-06	--	1E-07	2E-06	Liver toxicity (H)	3E-02	--	2E-03	3E-02
			ALPHA-BHC	4E-06	--	--	4E-06	--	--	--	--	
			2,4-DIMETHYLPHENOL	--	--	--	--	Clinical signs (lethargy, prostration, and ataxia) and hematological changes (B)	6E+00	--	5E-01	6E+00
			2-METHYLNAPHTHALENE	--	--	--	--	Pulmonary alveolar proteinosis	4E+00	--	--	4E+00
			3&4-METHYLPHENOL	--	--	--	--	Decreased body weight and neurotoxicity	2E+00	--	1E-01	3E+00
			4-METHYLPHENOL	--	--	--	--	--	5E+00	--	3E-01	5E+00
			BENZ(A)ANTHRACENE	1E-04	--	1E-03	1E-03	--	--	--	--	
			BENZO(A)PYRENE	5E-04	--	7E-03	8E-03	--	--	--	--	
			BENZO(B)FLUORANTHENE	5E-05	--	8E-04	9E-04	--	--	--	--	
			BENZO(K)FLUORANTHENE	5E-06	--	--	5E-06	--	--	--	--	
			BIS(2-ETHYLHEXYL)PHTHALATE	5E-07	--	5E-07	1E-06	Increased relative liver weight (H)	1E-02	--	1E-02	3E-02
			CHRYSENE	9E-07	--	8E-06	8E-06	--	--	--	--	
			DIBENZ(A,H)ANTHRACENE	7E-05	--	2E-03	2E-03	--	--	--	--	
			DIBENZOFURAN	--	--	--	--	Reduced length and organ weight. Excess abdominal fat (O).	5E+00	--	--	5E+00
			INDENO(1,2,3-CD)PYRENE	2E-05	--	3E-04	3E-04	--	--	--	--	
			NAPHTHALENE	--	--	--	--	Decreased body weight (W)	5E+00	--	2E+00	8E+00

TABLE 10.13 CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe:	Future
Receptor Population:	Resident
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Ground Water	Potable Water	Exposure Unit 8	PHENANTHRENE	--	--	--	--	--	4E-01	--	7E-01	1E+00
			1,4-DICHLOROBENZENE	9E-06	--	4E-06	1E-05	Liver	2E-01	--	8E-02	3E-01
			BENZENE	1E-03	--	1E-04	1E-03	Reduced lymphocyte count	4E+01	--	4E+00	4E+01
			VINYL CHLORIDE	3E-06	--	1E-07	3E-06	Liver cell polymorphism (H)	1E-02	--	3E-04	1E-02
		Chemical Total	2E-03	--	1E-02	1E-02		7E+01	--	9E+00	8E+01	
		Exposure Point Total				1E-02					8E+01	
		Exposure Medium Total				1E-02					8E+01	
	Shower Vapor	Exposure Unit 8	1,2,4-TRIMETHYLBENZENE	--	--	--	--	Hematological and Pulmonary	--	5E+00	--	5E+00
			1,4-DICHLOROBENZENE	--	3E-04	--	3E-04	Liver	--	7E-02	--	7E-02
			BENZENE	--	2E-03	--	2E-03	Decreased lymphocyte count	--	2E+01	--	2E+01
			BROMODICHLOROMETHANE	--	5E-06	--	5E-06	--	--	--	--	--
			CHLOROFORM	--	1E-05	--	1E-05	Hepatic effects	--	1E-02	--	1E-02
		Chemical Total	--	2E-03	--	2E-03		--	3E+01	--	3E+01	
			Exposure Point Total				2E-03					3E+01
	Exposure Medium Total				2E-03					3E+01		
Medium Total							2E-02				1E+02	
Receptor Total							2E-02	Receptor HI Total			1E+02	

Total Risk Across All Media = 2E-02

Total Hazard Across All Media = 1E+02

Total Liver HI Across All Media =	9E-01
Total Nervous System Effects HI Across All Media =	9E+00
Total Lymphocyte Effects HI Across All Media =	7E+01
Total Nasal/Respiratory Effects HI Across All Media =	9E+00
Total Other Effects HI Across All Media =	2E+01

TABLE 10.13a CT
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - SYW-12
CENTRAL TENDENCY
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	0E+00	--	--	--	0E+00	
		Exposure Point Total						0E+00				0E+00
		Exposure Medium Total						0E+00				0E+00
	Medium Total						0E+00				0E+00	
Soil	Surface Soil	Exposure Unit 9	BENZO(A)PYRENE	4E-06	--	6E-07	5E-06	--	--	--	--	--
			Chemical Total	4E-06	--	6E-07	5E-06	0E+00	--	0E+00	0E+00	
		Exposure Point Total						5E-06				0E+00
		Exposure Medium Total						5E-06				0E+00
Medium Total						5E-06				0E+00		
Receptor Total							5E-06	Receptor HI Total			0E+00	

Total Risk Across All Media = 5E-06

Total Hazard Across All Media = 0E+00

TABLE 10.13a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HONEYWELL WASTEBED B/HARBOR BROOK SITE - GEDDES AND SYRACUSE, NEW YORK

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Soil	Outdoor Air	Exposure Unit 9	None	--	--	--	--	--	--	--	--	--
			Chemical Total	--	--	--	--	--	--	--	--	
		Exposure Point Total				--				--		
	Exposure Medium Total						--				--	
Medium Total							--				--	
Soil	Surface Soil	Exposure Unit 9	2,3,7,8-TCDD Equivalent	1E-06	--	3E-07	2E-06	--	2E-02	--	5E-03	3E-02
			ARSENIC	3E-06	--	7E-07	3E-06	Hyperpigmentation (In); Vascular (V); PNS (N)	1E-02	--	3E-03	2E-02
			HIGHLY CHLORINATED PCBs	5E-07	--	6E-07	1E-06		3E-02	--	4E-02	7E-02
			BENZ(A)ANTHRACENE	2E-06	--	2E-06	4E-06		--	--	--	--
			BENZO(A)PYRENE	1E-05	--	1E-05	3E-05		--	--	--	--
			BENZO(B)FLUORANTHENE	2E-06	--	2E-06	4E-06		--	--	--	--
			DIBENZ(A,H)ANTHRACENE	1E-06	--	1E-06	3E-06		--	--	--	--
			Chemical Total	2E-05	--	2E-05	5E-05		--	7E-02	--	4E-02
		Exposure Point Total				5E-05				1E-01		
	Exposure Medium Total						5E-05				1E-01	
Medium Total							5E-05				1E-01	
Receptor Total							5E-05	Receptor HI Total			1E-01	

Total Risk Across All Media = 5E-05

Total Hazard Across All Media = 1E-01