HUMAN HEALTH RISK ASSESSMENT
DEARCOP FARM SITE
SITE NUMBER 8-28-016

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Prepared for:

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EXECUTIVE SUMMARY

Ecology and Environment, P.C., (E & E), under contract to the New York State Department of Environmental Conservation (NYSDEC) (Work Assignment No. D002625-10), performed Phases I and II of the Remedial Investigation (RI) at the Dearcop Farm site (NYSDEC site No. 8-28-016) in the Town of Gates, Monroe County, New York. The 16-acre site is situated in an urban area west of the New York State Barge Canal and the City of Rochester. The northern 10 acres of the site are overlain by the interchange of Interstate Routes 390 and 490. The southern 6 acres of the site consist of an undeveloped parcel adjacent to a residential area to the south composed of Dearcop Drive and Varian Lane.

During its operation from 1919 until 1970, wastes from various companies were disposed of on the Dearcop Farm site. These wastes include rubbish, office paper, wood, debris, scrap iron, foundry dirt, sandblasting debris, sand castings, and many materials that could pose a threat to human health, such as waste acids, heavy metals, waste oil, oily sludges, and halogenated organics. Also, 1,000 gallons of unknown substances from DuPont reportedly were burned in a burn pit on site.

The purpose of the RI was to provide a comprehensive characterization of the nature and extent of contamination through sampling of various media (e.g., groundwater, soil gas, soils, surface water/sediment, and homegrown vegetables) both on and off site. The results of the RI have been submitted in separate reports. This report includes only the Human Health Risk Assessment that was conducted as part of Phase II of the RI.

This baseline Human Health Risk Assessment was conducted in a manner consistent with U.S. Environmental Protection Agency (EPA) and NYSDEC guidance. For each potential exposure pathway, two cases were evaluated. The first was the "reasonable maximum exposure" (RME) case. This case was used to estimate the upper end of potential

exposures. An alternative exposure case was used to estimate a more "typical" exposure reflected by central tendency (i.e., average) exposure values. Where site-specific data were not available, exposure values based on EPA guidance were used.

Contaminants of potential concern (COPCs) evaluated in the quantitative risk assessment were selected by comparing detected concentrations to background concentrations and health-based screening levels. Chemicals that exceeded these levels were retained as COPCs. The potential exposure pathways evaluated were based on the presence of COPCs in various environmental media and on the potential for human exposure to these media. The following exposure scenarios were considered under current site conditions:

- Direct contact (incidental ingestion through hand-to-mouth contact and dermal contact) with contaminated surface soils by site trespassers;
- Inhalation of resuspended soil particles by site trespassers;
- Direct contact (incidental ingestion through hand-to-mouth contact and dermal contact) with contaminated surface soils in residential yards by nearby residents; and
- Consumption of vegetables grown in yards with fill material by nearby residents.

The possibility of future residential use of the landfill area itself or areas between the existing residences and the landfill was considered, but because Dearcop Drive and Varian Lane are surrounded on three sides by Interstate Route 390, Interstate Route 490, and the Barge Canal, this scenario was deemed unlikely. Consequently, future residential exposure in these areas was not evaluated.

Estimated Risks Under Current Site Conditions

Trespasser exposure to contaminants at the Dearcop Farm site under the RME case appears to pose a potential increased risk of developing cancer. Under existing site conditions, the estimated excess potential cancer risks under the RME case for adolescent trespassers is 3.4×10^{-6}. Total estimated current cancer risks for the average case are lower by approximately an order of magnitude (2.1×10^{-7}).

Site contaminants, under the RME case, do not appear to pose an increased risk of adverse noncarcinogenic health effects to site trespassers. The total hazard index for all potential pathways through which trespassers could be exposed to site-related contamination is 0.6 under the RME case, which is approximately half of the threshold hazard index of 1.

As discussed in sections of the RI and in this risk assessment, site-derived waste material reportedly was placed in residential yards and used as fill. The excess cancer risk and the potential for significant adverse health effects associated with this fill material were assessed for nearby residents. Data from composite soil samples collected from high-use areas (e.g., under swing sets, picnic tables) of residential yards were used to estimate these potential risks and adverse health effects. The maximum estimated potential cancer risk to residents from soil ingestion using RME assumptions was 1.2×10^{-4}. These excess estimated cancer risks are entirely due to polycyclic aromatic hydrocarbons (PAHs). The PAH concentrations were generally higher in residential soils than in on-site soils. Because PAHs are a common byproduct of combustion, there may be an off-site source of these PAHs. In addition, concentrations of PAHs in the residential soils were generally within the typical urban soil concentration range (600-3,000 μg/kg) reported by Menzie *et al.*, 1992.

Hazard indices for nearby residents were estimated for dermal exposure and soil ingestion. The maximum hazard indices due to dermal contact and soil ingestion under the RME case are 0.0072 and 0.10, respectively, which are below the threshold hazard index of 1. Hazard indices also were calculated for exposure to metals in tomatoes and broccoli grown in yards reportedly containing fill from the landfill. Because no formal screening criteria exist for vegetables, typical concentrations found in United States produce were used as benchmarks. Ingestion of broccoli yielded hazard indices greater than 1 under the RME case for cyanide (1.03) and manganese (2.68).

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1. INTRODUCTION

1.1 OVERVIEW

The Dearcop Farm site is an approximately 16-acre inactive landfill located in the Town of Gates, Monroe County, New York. The site is bordered on the east by a small man-made embankment about 70 feet west of the New York State Barge Canal, on the north by the westbound lanes of and an exit ramp from Interstate Route 490 (I-490), and on the west by Interstate Route 390 (I-390). The site is bordered on the south and southeast by the north ends of Dearcop Drive and Varian Lane, a medium-density residential area. Two Class 2 inactive hazardous-waste sites, Olin Chemical Corporation and the McKee Road Industrial Dump, are situated southeast of the site on the eastern side of the Barge Canal. One Class 2A site, Chevron USA Tank Farm, is located 0.5 mile south of the site on the southern side of Buffalo Road (State Route 33) (see Figure 1-1).

The site functioned as a disposal area from 1919 to 1970. The southern 6 acres of the site are currently owned by Mr. William L. Dearcop and Mr. Charles R. Dearcop, Jr. The northern 10 acres of the site were purchased by the New York State Department of Transportation (NYSDOT) about 1958 and are now overlain by the I-490/I-390 interchange.

Reportedly, the site received industrial waste between 1930 and 1970 from General Railway Signal Company; E.I. Dupont DeNemours and Company, Inc., (DuPont); the Pfaudler Company; and American Brakeshoe Company. When NYSDOT purchased the northern 10 acres, dumping in that area stopped.

The waste disposed of at the site included rubbish, office paper, wood, debris, scrap iron, foundry dirt, sand blasting debris, sand castings, and many materials that could potentially pose a threat to human health, such as waste acids, heavy metals, waste oil, oily

sludges, and halogenated organics. Also, 1,000 gallons of unknown substances from DuPont reportedly were burned in a burn pit on site.

The site investigation focused on characterizing the nature and extent of contamination associated with the site and identifying potential migration and exposure pathways that could pose a risk to human health. During the Phase I and II Remedial Investigation (RI) performed by Ecology and Environment Engineering, P.C. (E & E), surface soil, subsurface soil, soil gas, and groundwater samples were collected from locations on site and in the residential area to the south. Sediment and surface water samples also were collected from site drainage ditches and the Barge Canal. In addition, vegetable samples were collected from two residences during Phase II RI sampling.

Chlorinated and aromatic volatile and semivolatile organics and metals were detected in surface soil, subsurface soil, groundwater, drainage ditch sediments and water, and Barge Canal sediment and water. Polychlorinated biphenyls (PCBs) were detected in surface soil, subsurface soil, and Barge Canal sediment. Pesticides were detected in surface soil, subsurface soil, drainage ditch sediment and Barge Canal sediment. Chlorinated and aromatic volatile organics were detected in soil gas, and metals were detected in vegetables. Asbestos and other fibers (amosite, chrysotile, cellulose) were detected in surface and subsurface soils during the Phase I RI only.

There are two primary sources of site-related contamination to which site trespassers and nearby residents could potentially be exposed. The first is associated with the landfill area itself. Volatile organic compounds (VOCs), PCBs, polycyclic aromatic hydrocarbons (PAHs), and metals were detected in soils in the landfill area. Site trespassers potentially could be exposed to these contaminants through incidental ingestion of, or dermal contact with, contaminated soils. The VOCs could potentially migrate through the soil gas and be released at the soil surface to outdoor air; therefore, inhalation of soil vapors on site could be a potential exposure pathway for site trespassers.

The second source of contamination is fill material that reportedly was placed in the backyards of nearby residences. Several yards contained elevated levels of PAHs and metals; therefore, nearby residents may be exposed to these contaminants through incidental ingestion of, and dermal contact with, potentially contaminated soil in their yards.

1.2 SITE BACKGROUND

Detailed descriptions of the site, the site history, site activities, and the nature and extent of contamination are provided in the Phase I and II RI reports.

1.3 CONCEPTUAL SITE MODEL

A conceptual site model has been prepared and, as shown in Figure 1-2, there are five primary potential exposure pathways:

- Direct contact (incidental ingestion through hand-to-mouth contact and dermal contact) with contaminated site surface soils by site trespassers;
- Inhalation of airborne vapors by site trespassers and residents nearby the site;
- Inhalation of resuspended soil particles by site trespassers;
- Direct contact (incidental ingestion through hand-to-mouth contact and dermal contact) with contaminated soils in residential yards; and
- Consumption of vegetables grown in yards with fill material by residents nearby the site.

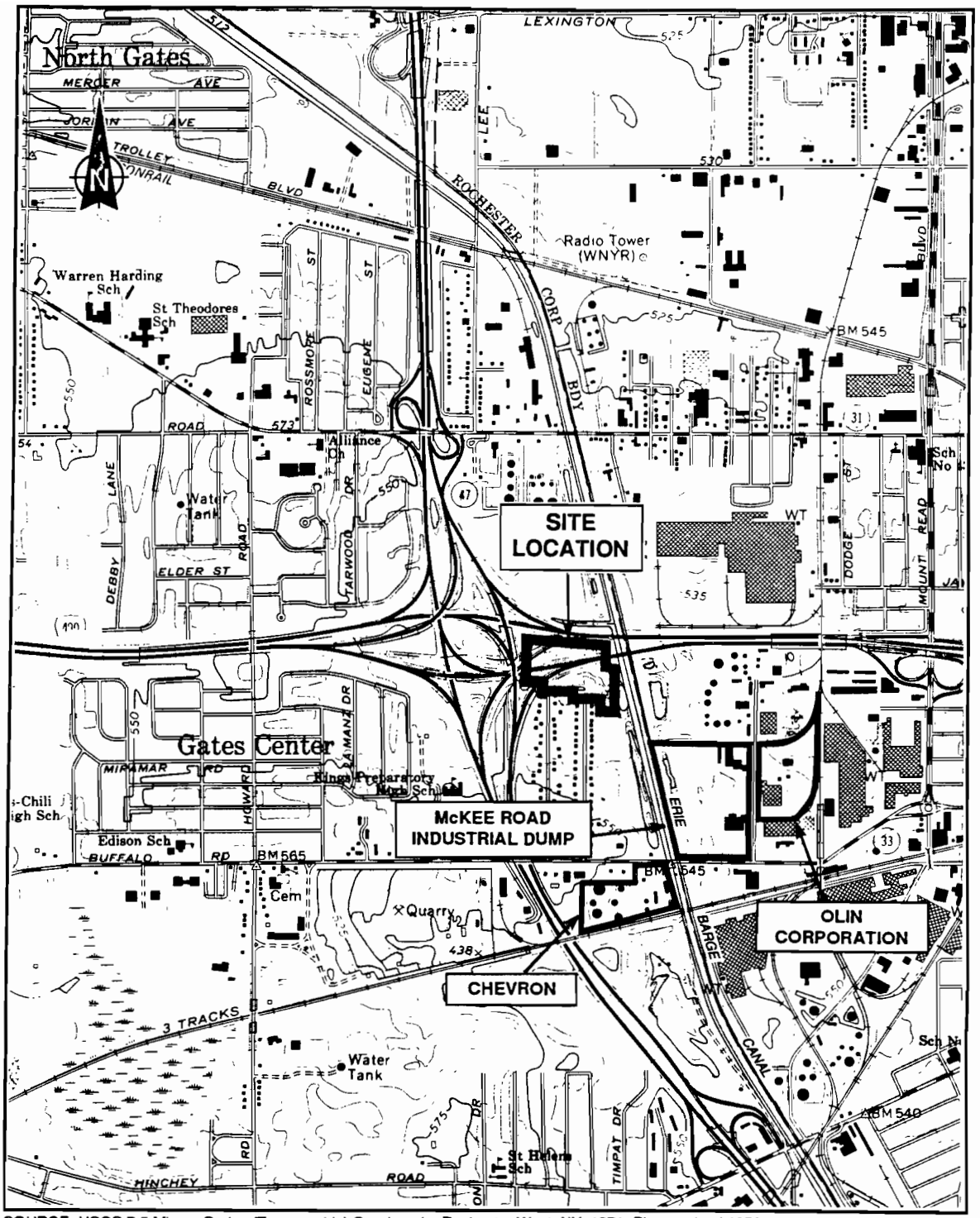
Site trespassers and nearby residents are the most likely potential receptors under existing land-use conditions. It is unlikely that under existing conditions the landfill area itself would be converted to residential use; therefore, risks to future potential residents were not evaluated. Homes exist adjacent to the Dearcop Farm property, but further development on Dearcop Drive and Varian Lane is not expected; therefore, the most plausible future change in land usage in the area might involve conversion of the vacant lot portion of the site to a more formal recreational use such as a playground or ballfield. The frequency of recreational exposures would probably increase if this were to occur, but such a conversion would require regrading of the site and probably excavation and replacement of clean fill; therefore, risks associated with direct contact with the primary medium of concern at the site (surface soils) would be reduced. Accordingly, additional exposure pathways that consider potential future land uses have not been included in the conceptual site model. The exposures and risks to both site trespassers and nearby residents by the pathways identified above are evaluated in this risk assessment, with the following exception: the vapor inhalation pathways were

screened against risk-based criteria in a preliminary risk evaluation for the site (E & E 1994). This evaluation indicated that these pathways were unlikely to pose significant risks; therefore, they were not quantitatively evaluated in this assessment.

1.4 ORGANIZATION OF THE RISK ASSESSMENT

This Human Health Risk Assessment has been prepared and organized in accordance with the United States Environmental Protection Agency's (EPA's) Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (EPA 1989b), other relevant EPA guidance, and NYSDEC guidance.

Section 1 reviews the site setting and presents the conceptual site model and potential exposure pathways. Section 2 reviews the available site characterization data, including the sampling plan, sampling and analytical methods, and data limitations, and identifies the chemicals of potential concern (COPCs) at the site. Section 3 assesses the potential exposure of receptors to the COPCs. The potential exposure pathways are reviewed, and exposure estimates derived, taking into consideration the site setting and various site characteristics. Section 4 provides toxicity assessments for the COPCs at the site. The section includes a review of toxicity assessment methodologies and a brief discussion of the toxicological properties of each chemical. Tables summarizing the quantitative indices of toxicity for the COPCs also are provided. Section 5 integrates the exposure and toxicity assessments from Sections 3 and 4 into an overall risk assessment. The main risks associated with the site are identified, along with the pathways and chemicals giving rise to those risks. Uncertainties in the risk assessment process and the risk estimates are discussed in Section 6, and a summary and conclusions are provided in Section 7.



SOURCE: USGS 7.5 Minute Series (Topographic) Quadrangle: Rochester West, NY, 1971, Photorevised 1978.

SCALE 1:24,000

0 ½ 1 Mile

0 .5 1 Kilometer

Figure 1-1
SITE LOCATION MAP, DEARCOP FARM SITE

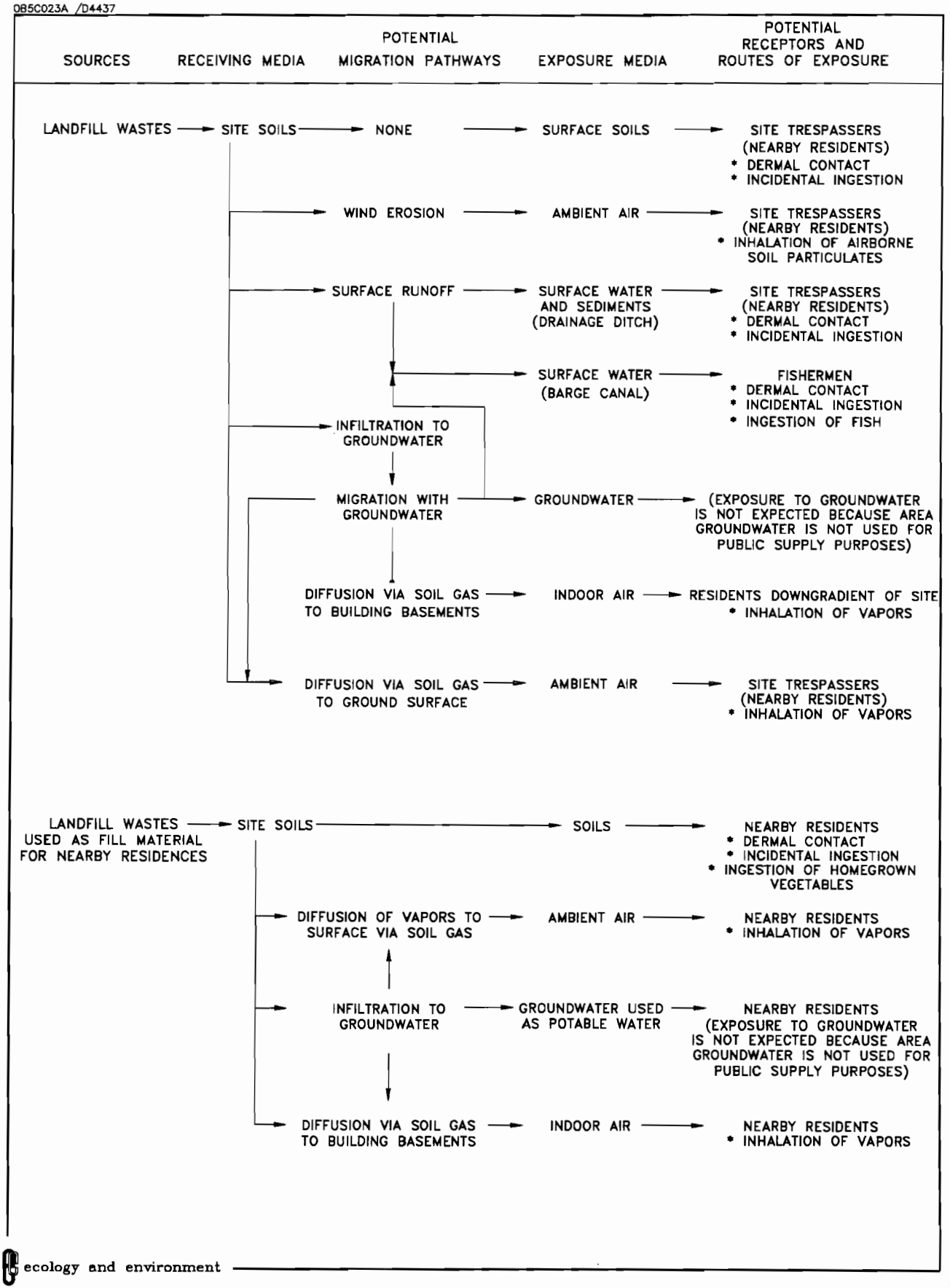


Figure 1-2 CONCEPTUAL SITE MODEL FOR DEARCOP FARM SITE

2. IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN (COPCs)

2.1 DATA COLLECTION

The objective of the RI was to identify and evaluate potential migration and exposure pathways by characterizing the nature and extent of contamination associated with the Dearcop Farm site, as well as its site topography, geology, hydrogeology, climate, and demographics. The investigative activities carried out to achieve this objective are discussed in Section 2 of the Phase I and Phase II RI reports, and a summary of the contamination found is presented in Section 3 of the RI reports.

During the Phase I RI, samples of surface soil, subsurface soil, soil gas, and groundwater were collected from locations on site and in the residential areas to the south. Sediment and surface water samples also were collected from site drainage ditches and the Barge Canal. COPCs were detected in soils, sediment, groundwater, and surface water. No background soil samples were taken during Phase I RI sampling.

In Phase II RI, additional soil, soil gas, surface water, sediment, and groundwater samples were collected. Vegetable and background surface soil samples were also collected. For surface soils, background samples SS-BG-2, SS-BG-2D, SS-BG-3, and SS-BG-4 were collected south of the site across State Route 33 approximately 100 to 250 feet from the highway. Sample SS-BG-1 was collected north of the site. No background samples were collected for subsurface soils, soil gas, groundwater, surface water, or vegetables.

The locations of most Phase I RI samples were selected in a directed fashion to investigate specific features of the site and to define the nature and extent of contamination. Phase II RI sampling concentrated primarily on addressing data gaps identified during the Phase I RI. Additional samples were collected from yards of residences that, based on the questionnaire distributed to area residences and observations made during the installation of

soil borings, were believed to contain debris. These samples were collected with a bias toward high-use areas (e.g., near swing sets, under picnic tables, in gardens).

Sampling of environmental media was carried out using standard EPA methodologies and quality assurance/quality control (QA/QC) procedures. The specific methods used are described in Section 2 of the Phase I report.

2.2 DATA EVALUATION

2.2.1 Data Validation

Analysis of the laboratory samples for Target Compound List (TCL) and Target Analyte List (TAL) substances was performed using methods and QA/QC procedures specified in the EPA contract laboratory program (CLP). Analyses were carried out by E & E's Analytical Services Center. E & E reviewed and validated the data packages using EPA functional guidelines for evaluating organic and inorganic analytes. Only data approved for use by this procedure were used in the risk assessment. Three composite surface soil samples were sent for third-party data validation because PAH contamination was detected in the surface soils.

2.2.2 Quantitation Limits

In accordance with EPA risk assessment guidance recommendations, the adequacy of the quantitation limits used in the analytical work for the Dearcop Farm site RI were evaluated by estimating the carcinogenic risk and/or noncarcinogenic hazard index for the COPCs, assuming that each chemical was present in the soil at its contract-required quantitation limit (CRQL) (EPA 1989b). The residential exposure scenario uses conservative standard default exposure assumptions; therefore, this scenario was used to evaluate the quantitation limits. The exposure assumptions used for this scenario are those specified in Section 3.3.3. The quantitation limits for soil, along with the corresponding cancer risks and hazard indices, are presented in Table 2-1.

Adequacy of detection limits is a common concern in risk assessments, particularly for highly toxic chemicals for which the concentrations corresponding to the 10^{-6} cancer risk level or a noncancer hazard index of 1 are very low. For the most part, CRQLs for carcinogenic and noncarcinogenic chemicals in soil appear to be adequate for the purposes of this risk assessment. Benzo(a)pyrene and dibenz(a,h)anthracene showed cancer risks greater

than 10^{-6}, but no chemicals had hazard indices greater than 1 when present in soils at their CRQLs. This indicates that the CRQLs for benzo(a)pyrene and dibenz(a,h)anthracene were not entirely adequate for risk assessment purposes at this site. Significant risks could exist, but they could be overlooked if these compounds were present at concentrations below, but approaching, their CRQLs.

The detection limits for some chemicals, including 1,1-DCA, naphthalene, cobalt, and lead, could not be quantitatively evaluated because confirmed toxicological indices (slope factors [SFs] and/or reference doses [RfDs]) were not available.

2.2.3 Data Qualifiers

Several types of data qualifiers were associated with a number of the analytical values reported and validated by the data evaluation process. The most commonly encountered data validation qualifiers, their meaning, and their effect on the use of the data in the risk assessment are summarized in Table 2-2. In accordance with the risk assessment guidance manual (EPA 1989b), if an analyte was found in a blank, values for the corresponding samples were included in the risk assessment only if the sample value was more than 10 times the blank value for common laboratory contaminants, or more than five times the blank value for other compounds.

Estimated value (J) flags were used to represent the best available estimates of the true concentrations present. The use of estimated values decreases the accuracy and confidence in quantitative estimates of exposures and risks obtained by using them. This will be noted in discussions of uncertainties. Nevertheless, their use provides the best available estimates of the concentrations actually present. U-flagged values (indicating that the chemical was not detected at the specified value) were evaluated on a case-by-case basis. If there was no reason to believe the substance was present in a sample, the U value was regarded as zero. If there was reason to believe it might be present, one-half of the quantitation limit (QL) for that substance was substituted for the U value. The presence of the chemical in a nearby sample (or if the chemical is a known degradation product of another detected compound) was considered evidence that the substance might be present in the U-flagged sample.

2.2.4 Background Concentrations

Most TAL metals are natural constituents of soils and groundwater at some concentration. Therefore, when evaluating data for risk assessment purposes, it is necessary to distinguish naturally occurring concentrations from those that may be due to contamination, and those due to on-site sources from those attributable to off-site sources. Metals concentrations in soils naturally tend to be highly variable. Data for a small number of background soil samples at a site often do not adequately reflect the range of metals concentrations that could occur naturally. Therefore, values reported by the United States Geological Survey (USGS) for background metals concentrations in surficial soils of the Eastern United States also were considered (Shacklette and Boerngen 1984). The 90th percentiles of the concentration distribution reported by these authors provide useful reference points for evaluating metals concentrations found in soils because only 10% of naturally occurring concentrations would exceed these values. Metals concentrations in site soils that did not exceed these values were considered within background concentrations. Higher concentrations were regarded as potentially due to contamination and were included in the risk assessment.

None of the chemicals detected in site media was eliminated from consideration in the risk assessment because of the chemicals' presence in background samples at similar concentrations. The maximum concentration of mercury (0.17 mg/kg) detected during the Phase I RI was only slightly higher than the highest background concentration (0.16 mg/kg); however, a maximum concentration of 2.0 mg/kg of mercury was detected in Phase II RI composite surface soil samples. Because these composite samples were taken with a bias toward high-use areas, mercury was retained as a COPC in surface soils.

2.3 SUMMARY OF ANALYTICAL RESULTS AND CHEMICALS OF POTENTIAL CONCERN

The chemicals detected in various environmental media in Phases I and II of the RI are summarized in Tables 2-3 through 2-17 as follows:

- Surface soil, Table 2-3;
- Composite surface soil, Table 2-4;
- Subsurface soil, Table 2-5;

- Composite subsurface soil, Table 2-6;
- Vegetables, Table 2-7;
- Groundwater, Table 2-8;
- Drainage ditch and quarry surface water, Table 2-9;
- Drainage ditch sediment, Table 2-10;
- Barge Canal surface water, Table 2-11;
- Barge Canal sediment, Table 2-12;
- Soil gas, Tables 2-13 and 2-14;
- Manhole waters, Table 2-15;
- Radionuclides in soil/sediment, Table 2-16; and
- Radionuclides in groundwater, Table 2-17.

Selection of COPCs was performed using media-specific criteria (see Table 2-18). In general, comparison of detected soil concentrations of analytes to site-specific and USGS background concentrations was the primary screening criterion. However, some chemicals that exceeded background concentrations but were well below (usually at least an order of magnitude) the applicable regulatory or risk-based criteria (maximum contaminant levels [MCLs], Ambient Water Quality Criteria [AWQC], EPA Region III risk-based concentrations [RBCs], or New York State Department of Environmental Conservation [NYSDEC] values) were not selected as COPCs to focus the risk assessment on the chemicals that might potentially pose a significant risk. The COPCs selected for each environmental medium are summarized in Table 2-19.

Table 2-1

CANCER RISKS AND HAZARD INDICES CORRESPONDING TO CRQL CONCENTRATIONS OF THE CHEMICALS OF POTENTIAL CONCERN

Chemical	Soil		
	CRQL (mg/kg)	Cancer Risk	Hazard Index
Acetone	0.01	—	1.3×10^{-6}
Barium	40	—	7.3×10^{-3}
Benzo(a)anthracene	0.33	2.6×10^{-7}	—
Benzo(a)pyrene	0.33	2.6×10^{-6}	—
Benzo(b)fluoranthene	0.33	2.6×10^{-7}	—
Benzo(k)fluoranthene	0.33	2.6×10^{-8}	—
Cadmium	1	—	2.6×10^{-2}
Chromium	2	—	2.6×10^{-5}
Chrysene	0.33	2.6×10^{-8}	—
Copper	5	—	1.7×10^{-3}
Dibenz(a,h)anthracene	0.33	2.6×10^{-6}	—
1,2-Dichloroethane	0.005	5.0×10^{-10}	—
Endrin	0.0033	—	1.4×10^{-4}
Ethylbenzene	0.005	—	6.4×10^{-7}
Heptachlor	0.0017	8.4×10^{-9}	4.4×10^{-5}
Heptachlor epoxide	0.0017	1.7×10^{-8}	1.7×10^{-3}
Indeno(1,2,3-cd)pyrene	0.33	2.6×10^{-7}	—
Lindane	0.0017	—	7.3×10^{-5}
Manganese	3	—	7.7×10^{-3}
Mercury	0.04	—	1.7×10^{-3}
Methylene chloride	0.1	8.2×10^{-10}	2.1×10^{-5}
2-Methylphenol	0.33	—	8.4×10^{-5}
4-Methylphenol	0.33	—	8.4×10^{-4}
N-Nitrosodiphenylamine	0.33	1.8×10^{-9}	—
Nickel	8	—	5.1×10^{-3}
PCBs	0.033	2.8×10^{-7}	—
Phenol	0.33	—	7×10^{-6}
Toluene	0.005	—	3.2×10^{-7}
1,1,1-Trichloroethane	0.005	—	7.1×10^{-7}

Table 2-1

CANCER RISKS AND HAZARD INDICES CORRESPONDING TO CRQL CONCENTRATIONS OF THE CHEMICALS OF POTENTIAL CONCERN

Chemical	Soil		
	CRQL (mg/kg)	Cancer Risk	Hazard Index
Trichloroethene	0.005	6.0×10^{-11}	—
Xylenes	0.005	—	3.2×10^{-8}
Zinc	4	—	1.7×10^{-4}

Key:

CRQL = Contract Required Quantitation Limit.

Table 2-2

DATA VALIDATION QUALIFIERS AND THEIR EFFECT ON DATA USE IN THE QUANTITATIVE RISK ASSESSMENT

Qualifier	Definition	Uncertain Identity?	Uncertain Concentration?	Include Data in Quantitative Risk Assessment?
Organic Chemical Data				
B	Analyte found in associated blank as well as in sample	No	Yes	Yes
E	Concentration exceeds calibration range of GC/MS instrument If Phase II RI groundwater samples were qualified with an E, diluted sample concentrations were used	No	Yes	Yes
Inorganic and Organic Chemical Data				
J	Value is estimated, either for a tentatively identified compound (TIC) or when a compound is present (spectral identification criteria are not met, but the value is < CRQL)	No for TCL chemicals Yes for TICs	Yes Yes	Yes Yes
U	Compound was analyzed for, but not detected	Yes	Yes	?

Key:

? = Determined on site-specific basis.
CRQL = Contract Required Quantitation Limit.
GC/MS = Gas Chromatography/Mass Spectrometry.
RI = Remedial Investigation.
TCL = Target Compound List.

Source: EPA 1989b.

Table 2-3

SUMMARY OF CONTAMINANTS DETECTED IN SURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[h]	Exceedance Frequency
Volatiles (µg/kg)											
2-Butanone	1/20	–	4.0	NA	–	300	0/20	47,000,000	0/20	4,000,000	0/20
Carbon Disulfide	3/20	2.0	3.0	NA	–	2,700	0/20	7,800,000	0/20	8,000,000	0/20
Chlorobenzene	1/20	–	2.0	NA	–	1,700	0/20	1,600,000	0/20	2,000,000	0/20
Toluene	3/20	1.0	18.0	NA	–	1,500	0/20	16,000,000	0/20	20,000,000	0/20
Trichloroethene	2/20	2.0	2.0	NA	–	700	0/20	58,000	0/20	64,000	0/20
Xylene (total)	1/20	–	1.0	NA	–	1,200	0/20	160,000,000	0/20	200,000,000	0/20
Semivolatiles (µg/kg)											
Acenaphthene	1/20	–	21.0	NA	–	50,000	0/20	4,700,000	0/20	5,000,000	0/20
Benzo(a)anthracene	1/20	–	200	NA	–	330	0/20	870	0/20	220	0/20
Benzo(a)pyrene[f]	1/20	–	280	NA	–	330	0/20	88	1/20	61	1/20
Benzo(b)fluoranthene	12/20	51.0	430	NA	–	1,100	0/20	870	0/20	220	1/20
Benzo(g,h,i)perylene	6/20	37.0	210	NA	–	50,000	0/20	NA	–	NA	–
Carbazole	1/20	–	56.0	NA	–	NA	–	32,000	0/20	8,300	0/20
Chrysene	1/20	–	260	NA	–	400	0/20	87,000	0/20	NA	–
Dibenz(a,h)anthracene	1/20	–	85.0	NA	–	330	0/20	88	0/20	14	1/20
Diethylphthalate	1/20	–	63.0	NA	–	7,100	0/20	63,000,000	0/20	60,000,000	0/20

Key at end of table.

Table 2-3

SUMMARY OF CONTAMINANTS DETECTED IN SURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[h]	Exceedance Frequency
Fluoranthene	11/20	22.0	390	NA	—	50,000	0/20	3,100,000	0/20	3,000,000	0/20
Fluorene	1/20	—	24.0	NA	—	50,000	0/20	3,100,000	0/20	3,000,000	0/20
Indeno(1,2,3-cd)pyrene	6/20	31.0	240	NA	—	3,200	0/20	870	0/20	NA	—
Phenanthrene	5/20	42.0	230	NA	—	50,000	0/20	NA	—	NA	—
Pyrene	12/20	33.0	370	NA	—	50,000	0/20	2,300,000	0/20	2,000,000	—
Pesticides (µg/kg)											
Alpha chlordane	2/20	19.0	86.0	NA	—	NA	—	490	0/20	540	0/20
PCBs (µg/kg)											
Aroclor 1254[f]	12/20	31.0	1,550	NA	—	100	5/20	83	7/20	79	6/20
Aroclor 1260[f]	11/20	26.0	945	NA	—	100	5/20	83	7/20	79	6/20
Inorganics (mg/kg)											
Aluminum	20/20	573	4,090	128,000	0/20	6,800[e]	0/20	230,000	0/20	NA	—
Arsenic	20/20	0.83	7.8	16.0	0/20	7.5[g]	1/20	23	0/20	80	0/20
Barium[f]	20/20	16.3	1,550	867	1/20	300[g]	1/20	5,500	0/20	4,000	0/20
Beryllium	4/20	0.30	0.39	1.81	0/20	0.41[e]	0/20	0.15	4/20	0.16	4/20
Cadmium[f]	19/20	0.48	8.8	NA	—	1.3[e]	11/20	39	0/20	80	0/20
Calcium	20/20	368	130,000	14,400	4/20	28,900[e]	3/20	NA	—	NA	—

Key at end of table.

Table 2-3

SUMMARY OF CONTAMINANTS DETECTED IN SURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[h]	Exceedance Frequency
Chromium	20/20	2.2	62.9	112	0/20	12.5[c]	10/20	78,000	0/20	80,000	0/20
Cobalt[f]	18/20	2.6	620	19.8	4/20	30.0[g]	3/20	NA	—	NA	—
Copper[f]	20/20	7.1	1,540	48.7	10/20	25.0[g]	10/20	2,900	0/20	NA	—
Iron	20/20	4,600	43,600	54,100	0/20	12,675[c]	9/20	NA	—	NA	—
Lead[f]	20/20	5.3	421	33.0	12/20	43.8[c]	7/20	NA	—	250	1/20
Magnesium	20/20	100	13,900	10,700	2/20	11,200[c]	1/20	NA	—	NA	—
Manganese	20/20	109	938	1,450	0/20	473[c]	10/20	390	12/20	20,000	0/20
Mercury	3/20	0.13	0.17	0.265	0/20	0.1	3/20	23	0/20	20	0/20
Nickel[f]	20/20	2.0	160	38.2	10/20	13.0[g]	11/20	1,600	0/20	2,000	0/20
Potassium	20/20	53.6	1,500	23,500	0/20	4,000[g]	0/20	NA	—	NA	—
Selenium	5/5[d]	0.22	0.71	0.941	0/5	2.0[g]	0/5	390	0/20	NA	—
Silver	17/20	0.75	22.4	NA	—	200	0/20	390	0/20	200	0/20
Sodium	4/20	114	3,720	17,400	0/20	3,000[g]	1/20	NA	—	NA	—
Vanadium	15/20	5.8	16.4	140	0/20	150[g]	0/20	550	0/20	600	0/20
Zinc[f]	20/20	14.3	858	104	6/20	48.6[c]	9/20	23,000	0/20	20,000	0/20
Cyanide	2/20	0.66	1.1	NA	—	NA	—	1,600	0/20	2,000	0/20

Key at end of table.

Table 2-3

SUMMARY OF CONTAMINANTS DETECTED IN SURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[h]	Exceedance Frequency
Asbestos and Other Fibers (%)											
Chrysotile	1/19	–	1.0	NA	–	NA	–	NA	–	NA	–
Cellulose	13/19	1.0	10.0	NA	–	NA	–	NA	–	NA	–

[a] Shacklette and Boerngen 1984.
[b] NYSDEC 1992.
[c] EPA 1993d, risk-based concentrations for residential soil.
[d] Although 19 samples were analyzed for selenium, only 5 of 19 values were not rejected through the quality assurance program.
[e] Site background.
[f] Selected as a COPC.
[g] Site background concentration is lower than NYSDEC-recommended soil cleanup goal or nondetect (potassium). Value reported is NYSDEC-recommended soil cleanup goal.
[h] Guidance derived from direct ingestion pathway (NYSDEC 1991a).

Key:

NA = Not available.

Table 2-4

SUMMARY OF CONTAMINANTS DETECTED IN COMPOSITE SURFACE SOIL SAMPLES

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[g]	Exceedance Frequency
Semivolatiles (µg/kg)											
Acenaphthene	12/12	700	26,000	NA	—	50,000	0/12	4,700,000	0/12	5,000,000	0/12
Acenaphthylene	1/12	—	860	NA	—	41,000	0/12	NA	—	300,000	0/12
Anthracene	12/12	44	5,900	NA	—	50,000	0/12	23,000,000	0/12	20,000,000	0/12
Benzo(a)anthracene[e]	12/12	220	11,000	NA	—	330	10/12	870	5/12	220	11/12
Benzo(a)pyrene[e]	12/12	180	8,000	NA	—	330	9/12	88	12/12	61	12/12
Benzo(b)fluoranthene[e]	12/12	190	6,300	NA	—	1,100	5/12	870	6/12	220	10/12
Benzo(g,h,i)perylene	12/12	180	6,100	NA	—	50,000	0/12	NA	—	NA	—
Benzo(k)fluoranthene[e]	12/12	130	4,700	NA	—	1,100	4/12	880	4/12	220	8/12
Chrysene[e]	12/12	180	6,100	NA	—	400	7/12	87,000	0/12	NA	—
Dibenz(a,h)anthracene[e]	11/12	230	4,800	NA	—	330	6/12	88	11/12	14	11/12
Fluoranthene	12/12	410	27,000	NA	—	50,000	0/12	3,100,000	0/12	3,000,000	0/12
Fluorene	12/12	13	3,000	NA	—	50,000	0/12	3,100,000	0/12	3,000,000	0/12
Indeno(1,2,3-cd)pyrene[e]	12/12	180	6,000	NA	—	3,200	2/12	870	6/12	NA	—
1-Methylnaphthalene	7/12	260	11,000	NA	—	NA	—	NA	—	NA	—
2-Methylnaphthalene	2/12	3,400	8,800	NA	—	36,400	0/12	NA	—	NA	—
Naphthalene[e]	12/12	350	18,000	NA	—	13,000	1/12	3,100,000	0/12	300,000	0/12

Key at end of table.

Table 2-4

SUMMARY OF CONTAMINANTS DETECTED IN COMPOSITE SURFACE SOIL SAMPLES

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[g]	Exceedance Frequency
Phenanthrene	12/12	130	17,000	NA	–	50,000	0/12	NA	–	NA	–
Pyrene	12/12	350	17,000	NA	–	50,000	0/12	2,300,000	0/12	2,000,000	0/12
Inorganics (mg/kg)											
Aluminum	12/12	2,540	6,480	128,000	0/12	6,800[d]	0/12	230,000	0/12	NA	–
Arsenic	12/12	2.5	13.6	16.0	0/12	7.5[f]	3/12	23	0/12	80	0/12
Barium	12/12	42.9	122	867	0/12	300[f]	0/12	5,500	0/12	4,000	0/12
Beryllium	12/12	0.22	0.55	1.81	0/12	0.41[d]	2/12	0.15	12/12	0.16	12/12
Cadmium[e]	10/12	0.63	2.5	NA	–	1.3[d]	3/12	39	0/12	80	0/12
Calcium	12/12	4,170	26,600	14,400	6/12	28,900[d]	0/12	NA	–	NA	–
Chromium	12/12	5.4	15.9	112	0/12	12.5[d]	4/12	78,000	0/12	80,000	0/12
Cobalt	12/12	4.0	9.4	19.8	0/12	30.0[f]	0/12	NA	–	NA	–
Copper[e]	12/12	18.9	777	48.7	5/12	25.0[f]	8/12	2,900	0/12	NA	–
Iron	12/12	6,530	15,900	54,100	0/12	12,675[d]	2/12	NA	–	NA	–
Lead[e]	12/12	31.5	237	33.0	11/12	43.8[d]	11/12	NA	–	250	0/12
Magnesium	12/12	1,200	5,410	10,700	0/12	11,200[d]	0/12	NA	–	NA	–
Manganese	12/12	165	1,240	1,450	0/12	473[d]	2/12	390	3/12	20,000	0/12
Mercury[e]	6/12	0.12	2.0	0.265	2/12	0.1	6/12	23	0/12	20	0/12
Nickel	12/12	2.5	17.3	38.2	0/12	13.0[f]	3/12	1,600	0/12	2,000	0/12

Key at end of table.

Table 2-4

SUMMARY OF CONTAMINANTS DETECTED IN COMPOSITE SURFACE SOIL SAMPLES

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[g]	Exceedance Frequency
Potassium	12/12	292	1,030	23,500	0/12	4,000[f]	0/12	NA	—	NA	—
Selenium	6/12	0.23	0.30	0.941	0/12	2.0[f]	0/12	390	0/12	NA	—
Silver	5/12	0.63	1.6	NA	—	200	0/12	390	0/12	200	0/12
Sodium	2/12	132	152	17,400	0/12	3,000[f]	0/12	NA	—	NA	—
Vanadium	12/12	8.6	17.0	140	0/12	150[f]	0/12	550	0/12	600	0/12
Zinc[e]	12/12	76.9	2,030	104	8/12	48.6[d]	12/12	23,000	0/12	20,000	0/12
Cyanide	5/12	0.59	0.78	NA	—	NA	—	1,600	0/12	2,000	0/12

[a] Shacklette and Boerngen 1984.
[b] NYSDEC 1992.
[c] EPA 1993d, risk-based concentrations for residential soil.
[d] Site background.
[e] Selected as a COPC.
[f] Site background concentration is lower than NYSDEC-recommended soil cleanup goal or nondetect (potassium). Value reported is NYSDEC-recommended soil cleanup goal.
[g] Guidance derived from direct ingestion pathway (NYSDEC 1991a).

Key:

ND = Not detected.
NA = Not available.

Table 2-5

SUMMARY OF CONTAMINANTS DETECTED IN SUBSURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Volatiles (µg/kg)											
Acetone[c]	4/37	85	260,000	NA	—	200	2/37	7,800,000	0/37	6,000,000	0/37
Benzene	1/37	—	2	NA	—	60	0/37	22,000	0/37	24,000	0/37
2-Butanone	10/37	2	44	NA	—	300	0/37	47,000,000	0/37	4,000,000	0/37
Carbon Disulfide	1/37	—	2	NA	—	2,700	0/37	7,800,000	0/37	8,000,000	0/37
Chlorobenzene	1/37	—	3	NA	—	1,700	0/37	1,600,000	0/37	2,000,000	0/37
1,1-Dichloroethane[c]	7/37	4	3,700	NA	—	200	1/37	7,800,000	0/37	8,000,000	0/37
1,2-Dichloroethane[c]	4/37	2	320,000	NA	—	100	3/37	700	1/37	7,700	1/37
Total 1,2-Dichloroethene	8/37	1	1,100	NA	—	NA	—	700,000	0/37	NA	—
Ethylbenzene[c]	2/37	3	110,000	NA	—	5,500	1/37	7,800,000	0/37	8,000,000	0/37
4-Methyl-2-pentanone	1/37	—	310	NA	—	1,000	0/37	3,900,000	0/37	4,000,000	0/37
Methylene chloride[c]	1/37	—	70,000	NA	—	100	1/37	85,000	0/37	93,000	0/37
Tetrachloroethene	2/37	4	162	NA	—	1,400	0/37	12,000	0/37	14,000	0/37
Toluene[c]	15/37	1	3,400,000	NA	—	1,500	2/37	16,000,000	0/37	20,000,000	0/37
Trichloroethene[c]	9/37	4	23,000	NA	—	700	1/37	58,000	0/37	64,000	0/37
1,1,1-Trichloroethane[c]	7/37	2	880,000	NA	—	800	2/37	7,000,000	0/37	7,000,000	0/37
Xylene (total)[e]	8/37	1	580,000	NA	—	1,200	2/37	160,000,000	0/37	200,000,000	0/37

Key at end of table.

Table 2-5

SUMMARY OF CONTAMINANTS DETECTED IN SUBSURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Semivolatiles (µg/kg)											
Acenaphthene	3/37	30	83	NA	—	50,000	0/37	4,700,000	0/37	5,000,000	0/37
Anthracene	6/37	22	98	NA	—	50,000	0/37	23,000,000	0/37	20,000,000	0/37
Benzo(a)anthracene[c]	9/37	50	2,200	NA	—	330	3/37	870	2/37	220	2/37
Benzo(a)pyrene[c]	8/37	41.5	3,100	NA	—	330	2/37	88	4/37	61	8/37
Benzo(b)fluoranthene[c]	10/37	40	8,300	NA	—	1,100	2/37	870	2/37	220	3/37
Benzo(g,h,i)perylene	10/37	29	5,000	NA	—	50,000	0/37	NA	—	NA	—
Benzo(k)fluoranthene[c]	8/37	59	2,400	NA	—	1,100	1/37	880	1/37	220	3/37
Bis(2-ethylhexyl)phthalate	3/37	28	247	NA	—	50,000	0/37	46,000	0/37	50,000	0/37
Butylbenzylphthalate	1/37	—	48	NA	—	50,000	0/37	16,000,000	0/37	20,000,000	0/37
Chrysene[c]	11/37	24	3,700	NA	—	400	3/37	87,000	0/37	NA	—
Dibenzo(a,h)anthracene[c]	5/37	19	2,000	NA	—	330	2/37	88	2/37	14	5/37
Dibenzofuran	4/37	29	58	NA	—	6,200	0/37	NA	—	NA	—
Diethylphthalate	3/37	—	50	NA	—	7,100	0/37	63,000,000	0/37	60,000,000	0/37
Di-n-butylphthalate	5/37	27	500	NA	—	8,100	0/37	7,800,000	0/37	8,000,000	0/37
Fluoranthene	9/37	32	4,700	NA	—	50,000	0/37	3,100,000	0/37	3,000,000	0/37
Fluorene	3/37	40	58.5	NA	—	50,000	0/37	3,100,000	0/37	3,000,000	0/37

Key at end of table.

Table 2-5

SUMMARY OF CONTAMINANTS DETECTED IN SUBSURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Indeno(1,2,3-cd)pyrene[c]	9/37	27	4,900	NA	–	3,200	1/37	870	1/37	NA	–
2-Methylnaphthalene	6/37	28	84	NA	–	36,400	0/37	NA	–	NA	–
2-Methylphenol[c]	1/37	–	3,900	NA	–	330	1/37	3,900,000	0/37	4,000,000	–
4-Methylphenol[c]	2/37	310	4,000	NA	–	900	1/37	390,000	0/37	4,000,000	–
Naphthalene	7/37	20	89.5	NA	–	13,000	0/37	3,100,000	0/37	300,000	0/37
N-nitrosodiphenylamine[c]	1/37	–	140,000	NA	–	NA	–	130,000	1/37	140,000	0/37
Phenanthrene	10/37	31	640	NA	–	50,000	0/37	NA	–	NA	–
Phenol[c]	2/37	200	540,000	NA	–	330	1/37	47,000,000	0/37	50,000,000	0/37
Pyrene	10/37	18	5,000	NA	–	50,000	0/37	2,300,000	0/37	2,000,000	–
Pesticides (µg/kg)											
Alpha chlordane	1/37	–	48.5	NA	–	NA	–	490	0/37	540	0/37
Alpha-BHC	1/37	–	55	NA	–	110	0/37	270	0/37	110	0/37
Beta-BHC	1/37	–	180	NA	–	200	0/37	660	0/37	3,900	0/37
4,4'-DDD	1/37	–	41	NA	–	2,900	0/37	2,700	0/37	NA	–
4,4'-DDE	1/37	–	4.6	NA	–	2,100	0/37	1,900	0/37	NA	–
4,4'-DDT	2/37	4.0	20	NA	–	2,100	0/37	1,900	0/37	NA	–
Dieldrin	1/37	–	29	NA	–	44	0/37	40	0/37	44	0/37

Key at end of table.

Table 2-5

SUMMARY OF CONTAMINANTS DETECTED IN SUBSURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Endosulfan I	1/37	–	123.5	NA	–	900	0/37	470,000	0/37	NA	–
Endosulfan sulfate	1/37	–	10	NA	–	1,000	0/37	NA	–	NA	–
Endrin[e]	2/37	24.8	200	NA	–	100	1/37	23,000	–	200,000	0/37
Gamma Chlordane	2/37	68.5	170	NA	–	540	0/37	490	0/37	540	0/37
Heptachlor[e]	3/37	22	180	NA	–	100	1/37	140	1/37	160	1/37
Heptachlor epoxide[e]	2/37	–	870	NA	–	20	2/37	70	2/37	770	2/37
Lindane[e]	3/37	4.7	280	NA	–	60	1/37	490	0/37	5,400	0/37
Methoxychlor	1/37	–	1,400	NA	–	10,000	0/37	390,000	0/37	80,000	0/37
PCBs (µg/kg)											
Aroclor 1248[e]	4/36	43	200,000	NA	–	10,000	1/36	83	3/36	79	3/36
Aroclor 1254[e]	9/36	16	3,300	NA	–	10,000	0/36	83	4/36	79	5/36
Aroclor 1260[e]	4/36	35	4,200	NA	–	10,000	0/36	83	2/36	79	3/36
Inorganics (mg/kg)											
Aluminum	37/37	372	11,300	128,000	0/37	30[c]	37/37	230,000	0/37	NA	–
Arsenic	37/37	0.50	7.0	16.0	0/37	7.5[c]	0/37	23	0/37	80	0/37
Barium[e]	37/37	14.0	1,150	867	1/37	300[c]	7/37	5,500	0/37	4,000	0/37
Beryllium	15/37	0.25	0.71	1.81	0/37	0.14[c]	15/37	0.15	15/37	0.16	15/37

Key at end of table.

Table 2-5

SUMMARY OF CONTAMINANTS DETECTED IN SUBSURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Cadmium[e]	37/37	0.75	111	NA	–	1[c]	26/37	39	1/37	80	1/37
Calcium	37/37	342	87,500	14,400	16/37	NA[c]	–	NA	–	NA	–
Chromium[e]	37/37	2.7	15,300	112	3/37	10[c]	20/37	78,000	0/37	80,000	0/37
Cobalt[e]	36/37	1.8	989	19.8	7/37	30[c]	4/37	NA	–	NA	–
Copper[e]	37/37	6.5	1,960	48.7	13/37	25[c]	14/37	2,900	0/37	NA	–
Iron	37/37	3,410	66,200	54,100	2/37	2,000[c]	37/37	NA	–	NA	–
Lead[e]	37/37	2.0	1,900	33.0	11/37	30[c]	11/37	NA	–	250	4/37
Magnesium	37/37	264	23,500	10,700	12/37	NA[c]	–	NA	–	NA	–
Manganese[e]	37/37	91.7	3,320	1,450	2/37	NA[c]	–	390	14/37	20,000	0/37
Mercury[e]	8/37	0.13	0.59	0.265	4/37	0.1	8/37	23	0/37	20	0/37
Nickel[e]	36/37	4.7	3,620	38.2	13/37	13[c]	21/37	1,600	1/37	2,000	1/37
Potassium	34/37	51.3	1,785	23,500	0/37	4,000[c]	0/37	NA	–	NA	–
Selenium	14/37	0.22	0.90	0.941	0/37	2[c]	0/37	390	0/37	NA	–
Silver	13/37	0.23	51.9	NA	–	200	0/37	390	0/37	200	0/37
Sodium	33/37	72.3	5,070	17,400	0/37	3,000[c]	1/37	NA	–	NA	–
Thallium	5/37	0.24	0.26	13.8	0/37	20[c]	0/37	NA	–	6,000	0/37
Vanadium	27/37	1.9	26.9	140	0/37	150[c]	0/37	550	0/37	600	0/37

Key at end of table.

Table 2-5

SUMMARY OF CONTAMINANTS DETECTED IN SUBSURFACE SOIL

Chemical	Frequency of Detection	Minimum	Maximum	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Zinc[e]	31/37	9.1	1,740	104	12/37	20[c]	29/37	23,000	0/37	20,000	0/37
Cyanide	3/37	0.66	8.2	NA	NA	NA	–	1,600	0/37	2,000	0/37
Asbestos and Other Fibers (%)											
Amosite	1/25	–	< 1.0	NA	–	NA	–	NA	–	NA	–
Cellulose	15/27	1.0	25.0	NA	–	NA	–	NA	–	NA	–
Chrysotile	6/27	TR	6.5	NA	–	NA	–	NA	–	NA	–
Other fibers	1/27	–	1.0	NA	–	NA	–	NA	–	NA	–

[a] Shacklette and Boerngen 1984.
[b] NYSDEC 1992.
[c] Value reported or site background.
[d] EPA 1993d, risk-based concentrations for residential soil.
[e] Selected as a COPC.
[f] Guidance derived from direct ingestion pathway (NYSDEC 1991a).

Key:

NA = Not available.
TR = Trace.

Table 2-6

SUMMARY OF CONTAMINANTS DETECTED IN COMPOSITE SUBSURFACE SOIL SAMPLE

Chemical	Frequency of Detection	Detected Concentration	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Volatiles (µg/kg)										
Diethylphthalate	1/1	110	NA	–	7,100	0/1	63,000,000	0/1	60,000,000	0/1
Pesticides (µg/kg)										
Alpha chlordane	1/1	12	NA	–	NA	–	490	0/1	540	0/1
4,4'-DDE	1/1	7.0	NA	–	2,100	0/1	1,900	0/1	NA	–
4,4'-DDT	1/1	29	NA	–	2,100	0/1	1,900	0/1	NA	–
Dieldrin	1/1	10	NA	–	44	0/1	40	0/1	44	0/1
Gamma Chlordane	1/1	12	NA	–	540	0/1	490	0/1	540	0/1
Heptachlor epoxide	1/1	4.8	NA	–	20	0/1	70	0/1	770	0/1
Inorganics (mg/kg)										
Aluminum	1/1	4,140	128,000	0/1	30[c]	1/1	230,000	0/1	NA	–
Arsenic	1/1	3.6	16.0	0/1	7.5[c]	0/1	23	0/1	80	0/1
Barium	1/1	31.1	867	0/1	300[c]	0/1	5,500	0/1	4,000	0/1
Beryllium	1/1	0.22	1.81	0/1	0.14[c]	1/1	0.15	1/1	0.16	1/1
Cadmium[e]	1/1	1.6	NA	–	1[c]	1/1	39	0/1	80	0/1
Calcium	1/1	2,140	14,400	0/1	NA[c]	–	NA	–	NA	–
Chromium	1/1	44.6	112	0/1	10[c]	1/1	78,000	0/1	80,000	0/1
Cobalt	1/1	9.0	19.8	0/1	30[c]	0/1	NA	–	NA	–

Key at end of table.

Table 2-6

SUMMARY OF CONTAMINANTS DETECTED IN COMPOSITE SUBSURFACE SOIL SAMPLE

Chemical	Frequency of Detection	Detected Concentration	USGS 90th Percentile Concentration for Eastern U.S. Soils[a]	Frequency of Detection Above USGS 90th Percentile Concentration for Eastern U.S. Soils	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III RBC for Residential Soil	Benchmark Health Risk Value[f]	Exceedance Frequency
Copper[e]	1/1	197	48.7	1/1	25[c]	1/1	2,900	0/1	NA	—
Iron	1/1	30,500	54,100	0/1	2,000[c]	1/1	NA	—	NA	—
Lead[e]	1/1	2,740	33.0	1/1	30[b]	1/1	NA	—	250	1/1
Magnesium	1/1	1,190	10,700	0/1	NA[c]	—	NA	—	NA	—
Manganese	1/1	692	1,450	0/1	NA[c]	—	390	1/1	20,000	0/1
Nickel	1/1	26.9	38.2	0/1	13[c]	1/1	1,600	0/1	2,000	0/1
Potassium	1/1	997	23,500	0/1	4,000[c]	0/1	NA	—	NA	—
Vanadium	1/1	8.4	140	0/1	150[c]	0/1	550	0/1	600	0/1
Zinc[e]	1/1	227	104	1/1	20[c]	1/1	23,000	0/1	20,000	0/1

[a] Shacklette and Boerngen 1984.
[b] NYSDEC 1992.
[c] Value reported or site background.
[d] EPA 1993d, risk-based concentrations for residential soil.
[e] Selected as a COPC.
[f] Guidance derived from direct ingestion pathway (NYSDEC 1991a).

Key:

NA = Not available.

Table 2-7

SUMMARY OF CONTAMINANTS DETECTED IN VEGETABLES (mg/kg dry weight)

Chemical	Frequency of Detection	Broccoli[b]	Typical Concentration in Broccoli[c]	Frequency of Exceedance of Typical Concentration	Tomato	Typical Concentration in Tomato[e]	Frequency of Exceedance of Typical Concentration
Aluminum	2/2	10.8	NA	—	11.7	20	0/1
Calcium	2/2	2,325	NA	—	113	NA	—
Chromium[a]	2/2	0.52	NA	—	0.49	0.074	1/1
Copper	2/2	4.7	10-15	0/1	0.58	7.4[f]	0/1
Iron	1/2	12.8	NA	—	—	NA	—
Lead[a]	2/2	0.50	0.05[d]	1/1	0.17	1.7[f]	0/1
Magnesium	2/2	624	NA	—	117	NA	—
Manganese	2/2	3.0	4.5[d]	0/1	0.49	12	0/1
Nickel[a]	2/2	0.55	0.29[d]	1/1	0.39	0.43-0.48	0/1
Potassium	2/2	3,980	NA	—	494	NA	—
Selenium[a]	1/2	0.08	0.005-<0.06[d]	1/1	—	—	—
Sodium	2/2	1,190	NA	—	755	NA	—
Zinc[a]	2/2	26.9	3[d]	1/1	3.2	21.5[f]	0/1
Cyanide	1/2	4.6	NA	—	—	—	—

Note: Calcium, iron, magnesium, potassium, and sodium are considered to be essential nutrients.

[a] Selected as a COPC.
[b] Reported concentrations are average of duplicates.
[c] Adriano 1986.
[d] Wet weight concentration.
[e] Kabata-Pendias and Pendias, 1992.
[f] Average of typical concentrations reported in Kabata-Pendias and Pendias, 1992.

Table 2-8

SUMMARY OF CONTAMINANTS DETECTED IN GROUNDWATER

Chemical	Frequency of Detection	Minimum	Maximum	Federal Maximum Contaminant Level[a]	Frequency of Detection Above Federal Maximum Contaminant Level	NYSDEC Class GA Groundwater Standard[b]	Frequency of Detection Above NYSDEC Class GA Groundwater Standard	EPA Region III RBC for Drinking Water[c]	Frequency of Detection Above EPA Region III RBC for Drinking Water
Volatiles (µg/L)									
Acetone	1/40	—	3	NA	—	50[j]	0/40	3,700	0/40
Carbon disulfide	5/40	2	21	NA	—	NA	—	21	0/40
Chloroethane[i]	2/40	41	56	NA	—	5	2/40	710	0/40
Benzene[i]	10/40	7	57.0	5.0	10/40	0.7	10/40	0.36	10/40
2-Butanone	1/40	—	14	NA	—	NA	—	22,000	0/40
Chlorobenzene[i]	1/40	—	11.0	100	0/40	5	1/40	39	0/40
Chloroform[i]	1/40	—	3.0	100	0/40	7.0	0/40	0.15	1/40
1,1-Dichloroethane[i]	25/40	5.0	4,300	NA	—	5.0	24/40	810	7/40
1,1-Dichloroethene[i]	15/40	1.0	310	7.0	10/40	5.0	10/40	0.044	15/40
Total 1,2-Dichloroethene[i]	26/40	2	2,300	70[d]	12/40	5.0	22/40	55	12/40
Ethylbenzene	8/40	1.0	5.0	700	0/40	5.0	0/40	1,300	0/40
Tetrachloroethene[i]	1/40	—	2.0	5.0	0/40	5.0	0/40	1.1	1/40
Toluene[i]	18/40	1.0	480	1,000	0/40	5.0	9/40	750	0/40
1,1,1-Trichloroethane[i]	18/40	1.0	2,100	200	2/40	5.0	10/40	1,300	2/40
1,1,2-Trichloroethane[i]	1/40	—	2	5	0/40	5.0	0/40	0.19	1/40

Key at end of table.

Table 2-8

SUMMARY OF CONTAMINANTS DETECTED IN GROUNDWATER

Chemical	Frequency of Detection	Minimum	Maximum	Federal Maximum Contaminant Level[a]	Frequency of Detection Above Federal Maximum Contaminant Level	NYSDEC Class GA Groundwater Standard[b]	Frequency of Detection Above NYSDEC Class GA Groundwater Standard	EPA Region III RBC for Drinking Water[c]	Frequency of Detection Above EPA Region III RBC for Drinking Water
Trichloroethene[i]	22/40	1.0	350	5.0	16/40	5.0	16/40	1.6	21/40
Vinyl chloride[i]	10/40	8	660	2.0	10/40	2.0	10/40	0.019	10/40
Total xylenes[i]	17/40	1.0	21.0	10,000	0/40	5.0	9/40	12,000	0/40
Semivolatiles (µg/L)									
Bis(2-ethylhexyl)phthalate[i]	5/40	1.0	20.0	6	2/40	50	0/40	4.8	2/40
Diethylphthalate	1/40	—	1.0	NA	—	NA	—	29,000	0/40
Di-n-octylphthalate	2/40	1.0	1.0	NA	—	NA	—	730	0/40
2-Methylphenol	2/40	—	1.0	NA	—	1.0 (total phenols cpds)	0/40	1,800	0/40
4-Methylphenol[i]	2/40	4.0	8	NA	—	1.0 (total phenols cpds)	2/40	180	0/40
Naphthalene	1/40	-	1.0	NA	—	NA	—	1,500	0/40
Phenol[i]	4/40	9.0	72.0	NA	—	1.0 (total phenols cpds)	4/40	22,000	0/40
Inorganics (µg/L)									
Aluminum	37/37	53.2	90,300	200[e]	23/37	NA	—	110,000	0/40
Antimony[i]	1/37	—	52.2	6	1/37	3	1/40	15	1/40

Key at end of table.

Table 2-8

SUMMARY OF CONTAMINANTS DETECTED IN GROUNDWATER

Chemical	Frequency of Detection	Minimum	Maximum	Federal Maximum Contaminant Level[a]	Frequency of Detection Above Federal Maximum Contaminant Level	NYSDEC Class GA Groundwater Standard[b]	Frequency of Detection Above NYSDEC Class GA Groundwater Standard	EPA Region III RBC for Drinking Water[c]	Frequency of Detection Above EPA Region III RBC for Drinking Water
Arsenic[i]	17/37	1.1	37.9	50	0/37	25	1/37	11	1/37
Barium	29/37	22.0	612	2,000	0/37	1,000	0/37	2,600	0/37
Beryllium[i]	1/37	–	4.5	4	1/37	3	1/37	0.016	1/37
Cadmium[i]	4/37	2.2	24.3	5	1/37	10	1/37	18	1/37
Calcium	37/37	37,900	1,400,000	NA	–	NA	–	NA	–
Chromium[i]	11/37	6.5	144	100	1/37	50	3/37	180[k]	0/37
Cobalt	5/37	5.4	91.3	NA	–	NA	–	NA	–
Copper[i]	13/37	3.6	341	1,000[e]	0/37	200	2/37	1,400	0/37
Iron	37/37	158	170,000	300[e]	27/37	300[g]	27/37	NA	–
Lead[i]	18/37	1.3	107	15[f]	4/37	25	4/37	NA	–
Magnesium	37/37	353	203,000	NA	–	NA	–	NA	–
Manganese[i]	31/37	5.7	5,760	50[e]	14/37	300[g]	3/37	180	5/37
Nickel[i]	9/37	7.3	144	100	2/37	NA	–	730	0/37
Potassium	37/37	1,060	80,500	NA	–	NA	–	NA	–
Selenium[i]	4/37	1.1	13.2	50	0/37	10	1/37	180	0/37
Sodium	33/37	20,000	6,000,000	NA	–	20,000	36/37	NA	–

Key at end of table.

Table 2-8

SUMMARY OF CONTAMINANTS DETECTED IN GROUNDWATER

Chemical	Frequency of Detection	Minimum	Maximum	Federal Maximum Contaminant Level[a]	Frequency of Detection Above Federal Maximum Contaminant Level	NYSDEC Class GA Groundwater Standard[b]	Frequency of Detection Above NYSDEC Class GA Groundwater Standard	EPA Region III RBC for Drinking Water[c]	Frequency of Detection Above EPA Region III RBC for Drinking Water
Thallium[i]	1/37	—	2.4	2.0	1/37	NA	—	NA	0/37
Vanadium	7/37	4.7	205	NA	—	NA	—	260	0/37
Zinc[i]	19/37	10.5	1,690	5,000[e]	0/37	300	2/37	11,000	0/37
Cyanide[i]	3/37	24.0	1,055	200	1/37	100	1/37	730[l]	1/37

a EPA 1993b, Drinking Water Regulations and Health Advisories.

b NYSDEC October 1993, Ambient Water Quality Standards and Guidance Values. Class GA groundwater is best suited as a potable water supply.

c EPA 1993d, risk-based concentrations for tap water.

d MCL for Total-1,2-Dichloroethene is based upon the MCL for the cis-isomer.

e Secondary MCL.

f Action level.

g Total for iron and manganese should not exceed 500 μg/L.

h The elevated concentrations of aluminum and iron suggest that some of the groundwater samples may have contained suspended sediment. Some of the other elevated metals concentrations identified as being of potential concern may be associated with these suspended sediments.

i Selected as a COPC.

j Guidance value.

k Standard is for chromium VI.

l Standard is for free cyanide.

Key:

NA = Not available.

Table 2-9

SUMMARY OF CONTAMINANTS DETECTED IN DRAINAGE DITCH AND QUARRY SURFACE WATER

Chemical	Frequency of Detection	Range of Detected Concentrations		NYSDEC Class D Surface Water Criteria[a]	Frequency of Detection Above NYSDEC Class D Surface Water Criteria
		Minimum	Maximum		
Volatiles (µg/L)					
Total 1,2-Dichloroethene	1/4	—	1.5	NA	—
Toluene	1/4	—	1.0	NA	—
Semivolatiles (µg/L)					
Bis(2-ethylhexyl)phthalate	3/4	1.0	4.0	NA	—
Diethylphthalate	1/4	—	3.0	NA	—
Inorganics (µg/L)					
Aluminum	2/4	158	1,230	NA	—
Arsenic	1/4	—	1.6	360[b]	0/4
Barium	4/4	51.1	89.7	NA	—
Calcium	4/4	80,600	128,000	NA	—
Copper	2/4	9.0	11.3	45.2 - 65.4[c]	0/4
Iron	3/4	235	967	300	2/4
Lead	3/4	1.9	11.8	290 - 478[c]	0/4
Magnesium	4/4	19,100	30,000	NA	—

Key at end of table.

Table 2-9

SUMMARY OF CONTAMINANTS DETECTED IN DRAINAGE DITCH AND QUARRY SURFACE WATER

Chemical	Frequency of Detection	Range of Detected Concentrations		NYSDEC Class D Surface Water Criteria[a]	Frequency of Detection Above NYSDEC Class D Surface Water Criteria
		Minimum	Maximum		
Manganese	4/4	9.4	600	NA	—
Mercury	1/4	—	0.33	0.2[d]	1/4
Potassium	4/4	2,490	3,790	NA	—
Selenium	1/4	—	1.6	NA	—
Sodium	4/4	56,600	533,000	NA	—
Zinc	2/4	52.2	260	733 - 1,020[c]	0/4

[a] NYSDEC, October 1993, Ambient Water Quality Standards and Guidance Values. Class D surface water is best suited for secondary contact. (Minimal contact expected, ingestion is unlikely.)

[b] Dissolved arsenic form.

[c] Criterion is hardness dependent, per sample.

[d] Guidance Value.

Key:

NA = Not available.

Table 2-10

SUMMARY OF CONTAMINANTS DETECTED IN DRAINAGE DITCH SEDIMENT

Chemical	Frequency of Detection	Minimum	Maximum	NYSDEC Sediment Criteria[a]	Frequency of Detection Above NYSDEC Sediment Criteria	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil
Volatiles (µg/kg)									
Carbon Disulfide	1/3	—	10.0	NA	—	2,700	0/3	7,800,000	0/3
Semivolatiles (µg/kg)									
Acenaphthene	4/6	850[e]	12,000	NA	—	50,000	0/6	4,700,000	0/6
Anthracene	5/6	31[e]	1,500	NA	—	50,000	0/6	23,000,000	0/6
Benzo(a)anthracene	5/6	163[e]	3,000	13	5/6	220	2/6	870	2/6
Benzo(a)pyrene	5/6	163[e]	2,200	13	5/6	61	4/6	88	5/6
Benzo(b)fluoranthene[d]	6/6	61.0	2,300	13	5/6	1,100	2/6	870	2/6
Benzo(g,h,i)perylene	5/6	143	1,600	NA	—	50,000	0/6	7,700	0/6
Benzo(k)fluoranthene[d]	4/6	170[e]	1,100	13	4/6	1,100	0/6	8,800	0/6
Carbazole	1/3	—	400	NA	—	NA	—	32,000	0/6
Chrysene[d]	6/6	53.0	2,000	13	6/6	400	2/6	87,000	0/6
Dibenzo(a,h)anthracene	5/6	100	1,100	NA	—	30	5/6	88	5/6
Dibenzofuran	1/3	—	280	NA	—	6,200	0/3	NA	—
Fluoranthene	6/6	110	5,900	NA	—	50,000	0/6	3,100,000	0/6
Fluorene	5/6	27	650	NA	—	50,000	0/6	3,100,000	0/6

Key at end of table.

Table 2-10

SUMMARY OF CONTAMINANTS DETECTED IN DRAINAGE DITCH SEDIMENT

Chemical	Frequency of Detection	Minimum	Maximum	NYSDEC Sediment Criteria[a]	Frequency of Detection Above NYSDEC Sediment Criteria	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil
Indeno(1,2,3-cd)pyrene	5/6	144[e]	1,500	13	5/6	3,200	0/6	870	2/6
1-Methylnaphthalene	3/6	250	1,000	NA	–	NA	–	NA	–
2-Methylnaphthalene	3/6	130	270	NA	–	36,400	0/6	NA	–
Naphthalene	4/6	255[e]	3,500	NA	–	13,000	0/6	3,100,000	0/6
Phenanthrene	6/6	68.0	3,600	NA	–	50,000	0/6	NA	–
Pyrene	6/6	94.0	5,700	NA	–	50,000	0/6	2,300,000	0/6
Pesticides/PCBs (µg/kg)									
alpha-Chlordane	2/3	2.9	5.7	0.01	2/3	NA	–	490	0/6
Aroclor-1254	2/3	23	33	0.008	2/3	1,000	0/3	83	0/3
4,4'DDT	1/3	–	7.8	0.1	1/3	2,100	0/3	1,900	0/3
Dieldrin	2/6	3.2	6.5	1	2/6	44.0	0/6	40	0/6
4,4'DDE	1/3	–	5.2	0.1	1/3	2,100	0/3	1,900	0/3
Endosulfan sulfate	1/3	–	16.0	NA	–	1,000	0/3	NA	–
Endrin ketone	1/3	–	21.0	NA	–	NA	–	NA	–
Heptachlor epoxide	3/6	4.0	16	0.3	3/6	20	0/6	70	0/6

Key at end of table.

Table 2-10

SUMMARY OF CONTAMINANTS DETECTED IN DRAINAGE DITCH SEDIMENT

Chemical	Frequency of Detection	Minimum	Maximum	NYSDEC Sediment Criteria[a]	Frequency of Detection Above NYSDEC Sediment Criteria	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil
Inorganics (mg/kg)									
Aluminum	6/6	2,090	5,530	NA	—	30[f]	6/6	230,000	0/6
Arsenic	6/6	0.79	5.3	5	1/6	7.5[f]	0/6	23	0/6
Barium	6/6	15.8	86.2	NA	—	300[f]	0/6	5,500	0/6
Beryllium	3/6	0.28[e]	0.42	NA	—	0.14	3/6	0.15	3/6
Cadmium[d]	6/6	1.0	2.4	0.8	6/6	1.0[f]	5/6	39	0/6
Calcium	6/6	16,400	38,500	NA	—	NA	—	NA	—
Chromium	6/6	5.0	15[e]	26	0/6	10[f]	3/6	78,000	0/6
Cobalt	6/6	5.7	10.3[e]	NA	—	30[f]	0/6	NA	—
Copper[d]	6/6	7.6	98.7[e]	19	4/6	25[f]	4/6	2,900	0/6
Iron	6/6	4,500	27,750[e]	24,000	1/6	2,000[f]	6/6	NA	—
Lead[d]	6/6	2.3	225	27	4/6	30[f]	4/6	NA	—
Magnesium	6/6	3,880	11,500	NA	—	NA[f]	—	NA	—
Manganese	6/6	161	565[e]	428	1/6	NA[f]	—	390	1/6
Mercury	2/6	2.9	3.0	0.11	2/6	0.1	2/6	23	0/6
Nickel	6/6	5.1	21.4[e]	22	0/6	13[f]	2/6	1,600	0/6

Key at end of table.

Table 2-10

SUMMARY OF CONTAMINANTS DETECTED IN DRAINAGE DITCH SEDIMENT

Chemical	Frequency of Detection	Minimum	Maximum	NYSDEC Sediment Criteria[a]	Frequency of Detection Above NYSDEC Sediment Criteria	NYSDEC Soil Cleanup Guidance[b]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[c]	Frequency of Detection Above EPA Region III RBC for Residential Soil
Potassium	3/6	292	325	NA	—	4,000[f]	0/6	NA	—
Selenium	2/6	0.24	0.35	NA	—	2[f]	0/6	390	0/6
Silver	3/6	—	1.3	NA	—	200	0/6	390	0/6
Sodium	6/6	109	1,340	NA	—	3,000[f]	0/6	NA	—
Vanadium	5/6	6.9	15.0	NA	—	150[f]	0/6	550	0/6
Zinc[d]	6/6	35.0	1,228[e]	85	4/6	20[f]	6/6	23,000	0/6

[a] For organics, Division of Fish and Wildlife Human Health Sediment Criteria, NYSDEC, November 1993. For inorganics, Division of Fish and Wildlife Sediment Criteria Guidance Document, NYSDEC, December 1989.

[b] NYSDEC 1992.

[c] EPA 1993d, risk-based concentrations for residential soil.

[d] Selected as a COPC.

[e] Average of duplicate and sample.

[f] Value reported or site background.

Key:

NA = Not available.

Table 2-11

SUMMARY OF CONTAMINANTS DETECTED IN BARGE CANAL SURFACE WATER

Chemical	Frequency of Detection	Range of Detected Concentrations		NYSDEC Class C Surface Water Criteria[a]	Frequency of Detection Above NYSDEC Class C Surface Water Criteria
		Minimum	Maximum		
Volatiles (µg/L)					
Carbon disulfide	1/3	—	130	NA	—
Total 1,2-Dichloroethene	2/3	1.0	2.0	NA	—
Semivolatiles (µg/L)					
Bis(2-Ethylhexyl)phthalate[c]	3/3	1.0	3.0	0.6	3/3
2-Chloropyridine	3/3	2.0	3.0	NA	—
Inorganics (µg/L)					
Aluminum[c]	5/5	175	494	100[e]	5/5
Barium	5/5	23.8[d]	43.4	NA	—
Calcium	5/5	43,400	83,700	NA	—
Copper	2/5	1.9	3.8	6.1 - 42.5[b]	0/5
Iron	5/5	279[d]	836	300	3/5
Lead	3/5	2.2	11.1	3.8 - 11.3[b]	0/5
Magnesium	5/5	9,840	19,300	NA	—

Key at end of table.

Table 2-11

SUMMARY OF CONTAMINANTS DETECTED IN BARGE CANAL SURFACE WATER

Chemical	Frequency of Detection	Range of Detected Concentrations		NYSDEC Class C Surface Water Criteria[a]	Frequency of Detection Above NYSDEC Class C Surface Water Criteria
		Minimum	Maximum		
Manganese	5/5	12.9[d]	46.1	NA	—
Potassium	5/5	1,195[d]	2,890	NA	—
Selenium[c]	3/5	1.4	1.8	1.0	3/5
Sodium	5/5	13,500	52,100	NA	—
Zinc	2/2	11.4	23.2	30	0/2

a NYSDEC October 1993, Ambient Water Quality Standards and Guidance Values. Class C surface water is suitable for fish propagation, and primary and secondary contact recreation.

b Standard dependent on hardness.

c Selected as a COPC.

d Average of duplicate and sample.

Key:

NA =Not available.

Table 2-12

SUMMARY OF CONTAMINANTS DETECTED IN BARGE CANAL SEDIMENT

Chemical	Frequency of Detection	Minimum	Maximum	NYSDEC Sediment Criteria[b]	Frequency of Detection Above NYSDEC Sediment Criteria	NYSDEC Soil Cleanup Guidance[c]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III Residential Soil
Volatiles (µg/kg)									
1,1-Dichloroethane	1/3	—	3.0	NA	—	200	0/3	7,800,000	0/3
Total 1,2-Dichloroethene	1/3	—	5.0	NA	—	300[f]	0/3	700,000	0/3
Trichloroethene	1/3	—	2.0	NA	—	700	0/3	58,000	0/3
Xylene (Total)	1/3	—	1.0	NA	—	1,200	0/3	160,000,000	0/3
Semivolatiles (µg/kg)									
Acenaphthene	2/5	1,200	5,900	7,300	0/5	50,000	0/5	4,700,000	0/5
Anthracene	4/5	42.0	1,500	NA	—	50,000	0/5	23,000,000	0/5
Benzo(a)anthracene	5/5	170	2,400	NA	—	330	2/5	870	1/5
Benzo(a)pyrene	3/5	190	2,000	NA	—	330	2/5	88	3/5
Benzo(b)fluoranthene	5/5	380	1,600	NA	—	1,100	1/5	870	1/5
Benzo(k)fluoranthene	2/5	330	1,200	NA	—	1,100	1/5	8,800	0/5
Benzo(g,h,i)perylene	5/5	170	1,400	NA	—	50,000	0/5	NA	—
Butylbenzylphthalate	1/3	—	73.0	NA	—	50,000	0/3	16,000,000	0/3
Chrysene	5/5	260	1,900	NA	—	400	2/5	87,000	0/5
Dibenzo(a,h)anthracene	5/5	91.0	1,100	NA	—	330	1/5	88	5/5
Fluoranthene	5/5	520	6,700	NA	—	50,000	0/5	3,100,000	0/5
Fluorene	4/5	53.0	800	NA	—	50,000	0/5	3,100,000	0/5
Indeno(1,2,3-cd)Pyrene	5/5	230	1,400	NA	—	3,200	0/5	870	1/5

Table 2-12

SUMMARY OF CONTAMINANTS DETECTED IN BARGE CANAL SEDIMENT

Chemical	Frequency of Detection	Minimum	Maximum	NYSDEC Sediment Criteria[b]	Frequency of Detection Above NYSDEC Sediment Criteria	NYSDEC Soil Cleanup Guidance[c]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III Residential Soil
Naphthalene	4/5	45.0	6,100	NA	—	13,000	0/5	3,100,000	0/5
Phenanthrene	5/5	230	4,600	1,390	1/5	50,000	0/5	NA	—
Pyrene	5/5	420	6,300	NA	—	50,000	0/5	2,300,000	0/5
Pesticides (μg/kg)									
4,4'-DDE	1/5	—	7.0	≤500	0/5	2,100	0/5	1,900	0/5
Heptachlor epoxide	1/5	—	8.9	0.3	1/5	20	0/5	70	0/5
PCBs (μg/kg)/									
Aroclor 1254	5/5	44.0	110	2,760	0/5	100	1/5	83	2/5
Inorganics (mg/kg)									
Aluminum	5/5	2,470	13,000	NA	—	30[e]	5/5	230,000	0/5
Arsenic	5/5	3.2	6.2	5	1/5	7.5[e]	0/5	23	0/5
Barium	5/5	4.32	104	NA	—	300[e]	0/5	5,500	0/5
Beryllium	4/5	0.38	0.86	NA	—	0.14[e]	4/5	0.15	4/5
Cadmium	5/5	1.6	2.3	0.8	5/5	1[e]	5/5	39	0/5
Calcium	5/5	27,900	56,900	NA	—	NA[e]	—	NA	—
Chromium	5/5	23.4	57.4	26	3/5	10[e]	5/5	78,000	0/5
Cobalt	5/5	4.7	19.4	NA	—	30.0[e]	0/5	NA	—
Copper[a]	5/5	34.0	75.6	19	5/5	25.0[e]	5/5	2,900	0/5
Iron	5/5	8,670	39,900	24,000	2/5	2,000[e]	5/5	NA	—

Table 2-12

SUMMARY OF CONTAMINANTS DETECTED IN BARGE CANAL SEDIMENT

Chemical	Frequency of Detection	Minimum	Maximum	NYSDEC Sediment Criteria[b]	Frequency of Detection Above NYSDEC Sediment Criteria	NYSDEC Soil Cleanup Guidance[c]	Frequency of Detection Above NYSDEC Soil Cleanup Guidance	EPA Region III RBC for Residential Soil[d]	Frequency of Detection Above EPA Region III Residential Soil
Lead	5/5	66.8	1,220	27	5/5	30[e]	5/5	NA	—
Magnesium	5/5	6,660	22,000	NA	—	NA[e]	—	NA	—
Manganese	5/5	299	681	428	2/5	NA[c]	—	390	2/5
Mercury[a]	4/5	0.23	0.82	0.11	4/5	0.1	4/5	23	0/5
Nickel	5/5	12.8	44.9	22	2/5	13.0[e]	4/5	1,600	0/5
Potassium	3/5	536	586	NA	—	4,000[e]	0/5	NA	—
Selenium	1/5	—	0.44	NA	—	2.0[e]	0/5	390	0/5
Silver	2/5	1.3	1.4	NA	—	200	0/5	390	0/5
Sodium	3/5	180	522	NA	—	3,000[e]	0/5	NA	—
Vanadium	2/5	18.0	26.3	NA	—	150[e]	0/5	550	0/5
Zinc[a]	3/5	224	444	85	3/5	20[e]	5/5	23,000	0/5

[a] Selected as COPC.
[b] Division of Fish and Wildlife Sediment Criteria Guidance Document (December 1989).
[c] NYSDEC 1992.
[d] EPA 1993d.
[e] Value reported or site background.
[f] Goal is for 1,2-DCE(trans).

Note: To determine the COPCs in Barge Canal sediments, concentrations of contaminants detected in the on-site portion of the drainage ditch and concentrations detected in the drainage ditch prior to its entrance onto the Dearcop Farm site were compared with concentrations detected in the Barge Canal sediments. If the contaminants were detected at higher concentrations in off-site samples than in on-site samples, the contaminants were not considered site related and therefore were not selected as COPCs.

Table 2-13

SUMMARY OF CONTAMINANTS DETECTED IN SOIL GAS

Chemical	Frequency of Detection	Range of Detected Contaminants		NYS Annual Guideline Concentration[a]	EPA Region III RBC for Air[b]	Indoor Air Dilution Factor[c]
		Minimum	Maximum			
Volatiles ($\mu g/m^3$)						
Benzene	8/196	11.0	5,000	0.12	0.22	5.0×10^{-4}
1,1-Dichloroethane	28/196	10.0	70,000	500	520	5.0×10^{-4}
1,2-Dichloroethane	2/196	53.0	63.0	0.039	0.069	5.0×10^{-4}
1,1-Dichloroethene	3/196	14.0	400	NA	0.036	5.0×10^{-4}
cis-1,2-Dichloroethene	18/196	15.0	5,000	1,900	37	5.0×10^{-4}
trans-1,2-Dichloroethene	19/196	13.0	15,000	360	73	5.0×10^{-4}
Ethylbenzene	8/196	33.0	5,000	1,000	1,000	5.0×10^{-4}
Methylene chloride	18/196	6.0	40,000	27	3.8	5.0×10^{-4}
Toluene	9/196	52.0	5,000	2,000	420	5.0×10^{-4}
1,1,1-Trichloroethane	30/196	1.0	5,000	1,000	1,000	5.0×10^{-4}
Trichloroethene	55/196	9.9	51,000	0.45	1	5.0×10^{-4}
Vinyl chloride	9/195	19.0	5,000	0.02	0.021	5.0×10^{-4}
Xylenes (total)	3/196	2,300	5,000	300	7,300	5.0×10^{-4}

Key at end of table.

Table 2-13

SUMMARY OF CONTAMINANTS DETECTED IN SOIL GAS

Chemical	Maximum Estimated Indoor Air Concentration	Frequency Above NYS Annual Guideline Concentration	Frequency Above EPA RBC for Air	Outdoor Air Dilution Factor	Estimated Maximum Outdoor Air Concentration	Frequency Above NYS Annual Guideline Concentration	Frequency Above EPA Region III RBC for Air
Volatiles ($\mu g/m^3$)							
Benzene	2.5	3	2	1×10^{-6}	5×10^{-3}	0/196	0/196
1,1-Dichloroethane	35.0	0	0	1×10^{-6}	7×10^{-2}	0/196	0/196
1,2-Dichloroethane	0.032	0	0	1×10^{-6}	6.3×10^{-5}	0/196	0/196
1,1-Dichloroethene	0.20	—	2	1×10^{-6}	4×10^{-4}	0/196	0/196
cis-1,2-Dichloroethene	2.5	0	0	1×10^{-6}	5×10^{-3}	0/196	0/196
trans-1,2-Dichloroethene	7.5	0	0	1×10^{-6}	1.5×10^{-2}	0/196	0/196
Ethylbenzene	2.5	0	0	1×10^{-6}	5×10^{-3}	0/196	0/196
Methylene chloride	20.0	0	3	1×10^{-6}	4×10^{-2}	0/196	0/196
Toluene	2.5	0	0	1×10^{-6}	5×10^{-3}	0/196	0/196
1,1,1-Trichloroethane	2.5	0	0	1×10^{-6}	5×10^{-3}	0/196	0/196
Trichloroethene	25.5	19	12	1×10^{-6}	5.1×10^{-2}	0/196	0/196
Vinyl chloride	2.5	9	5	1×10^{-6}	5×10^{-3}	0/195	0/195
Xylenes (total)	2.5	300	0	1×10^{-6}	5×10^{-3}	0/196	0/196

[a] NYSDEC 1991b.

[b] EPA 1993d, risk-based concentrations for ambient air.

[c] EPA 1992a, Assessing potential indoor air impacts for Superfund sites.

Key:

NA = Not available.

Table 2-14

SUMMARY OF CONTAMINANTS DETECTED IN SOIL GAS SAMPLES FROM THE CURRENT RESIDENTIAL AREA AND BORDERING LOCATIONS

Chemical	Frequency of Detection	Range of Detected Contaminants		NYS Annual Guideline Concentration[a]	EPA Region III RBC for Air[b]	Indoor Air Dilution Factor[c]	Maximum Estimated Indoor Air Concentrations	Frequency Above NYS Annual Guideline Concentration	Frequency Above EPA Region III RBC for Air
		Minimum	Maximum						
Volatiles ($\mu g/m^3$)									
1,1-Dichloroethane	2/60	10	140	500	520	5×10^{-4}	0.07	0/60	0/60
trans-1-2-Dichloroethene	2/60	38	58	360	73	5×10^{-4}	0.029	0/60	0/60
Ethylbenzene	1/60	—	240	1,000	1,000	5×10^{-4}	0.12	0/60	0/60
Methylene chloride	1/60	—	70	27	3.8	5×10^{-4}	0.035	0/60	0/60
1,1,1-Trichloroethane	2/60	16	30	1,000	1,000	5×10^{-4}	0.015	0/60	0/60
Trichloroethene	10/60	13	580	0.45	1	5×10^{-4}	0.10	0/60	0\60

[a] NYSDEC 1991b.

[b] EPA 1993d, risk-based concentrations for ambient air.

[c] EPA 1992a, Assessing potential indoor air impacts for Superfund sites.

Table 2-15

SUMMARY OF CONTAMINANTS DETECTED IN MANHOLE WATERS

Chemical	Frequency of Detection	Minimum	Maximum
Volatiles (μg/L)			
Benzene	2/4	4	17
Bromodichloromethane	3/4	1	4
Chloroform	4/4	2	48
Ethylbenzene	1/4	—	3
Toluene	3/4	1	4
Xylene	2/4	2	10
Semivolatiles (μg/L)			
Bis(2-ethylhexyl)phthalate	2/4	3	4
Butylbenzylphthalate	1/4	—	1
1,4-Dichlorobenzene	3/4	3	6
Diethylphthalate	2/4	20	29
Di-n-butylphthalate	1/4	—	5
4-Methylphenol	2/4	7	9
Phenol	1/4	—	3
Inorganics (μg/L)			
Aluminum	3/4	334	558
Arsenic	1/4	—	1.9
Barium	4/4	40.2	90.9
Calcium	4/4	69,600	103,000
Chromium	1/4	—	8.3
Copper	4/4	22.8	120
Iron	4/4	395	2,790
Lead	4/4	1.8	16.3
Magnesium	4/4	16,500	24,100
Manganese	4/4	55.9	190
Mercury	2/4	3.2	3.4
Nickel	3/4	9.3	57.1

Table 2-15

SUMMARY OF CONTAMINANTS DETECTED IN MANHOLE WATERS

Chemical	Frequency of Detection	Minimum	Maximum
Potassium	4/4	4,770	12,600
Selenium	2/4	1.5	2.1
Silver	2/4	24.9	26.8
Sodium	4/4	48,800	269,000
Vanadium	1/4	—	4.8
Zinc	4/4	40.5	214
Cyanide	1/4	—	10.0

Table 2-16

SUMMARY OF RADIONUCLIDES DETECTED IN SOIL/SEDIMENT SAMPLES (pCi/g dry)

	Frequency of Detection	Minimum[a]	Maximum[a]	Residential Soil PRG at 10^{-6} Cancer Risk	Frequency Above 10^{-6} Cancer Risk Soil PRG
Gross alpha	12/14	3.9	36	NA	—
Gross beta	14/14	4.3	120	NA	—
Actinium-228	14/14	0.327	1.9	0.014	14/14
Bismuth-214	14/14	0.159	4.2	0.0079	14/14
Cesium-137	2/12	0.138	0.96	28	0/12
Lead-212	14/14	0.26	1.7	0.15	14/14
Lead-214	14/14	0.24	4.7	0.065	14/14
Potassium-40	10/10	0.383	14	0.077	10/10
Radium-226	7/14	0.697	8.2	2.3	3/14
Radium-228	14/14	0.327	1.9	7.9	0/14
Thallium-208	14/14	0.32	1.6	0.0032	14/14
Thorium-228	3/6	0.353	0.893	37	0/6
Thorium-234	2/10	0.724	3.1	11	0/10

[a] Value shown does not include reported analytical uncertainty.

Key:

NA = Not available.
PRG = Preliminary remediation goals for soil under residential land use.

Table 2-17

SUMMARY OF RADIONUCLIDES DETECTED IN GROUNDWATER (pCi/L)

	Frequency of Detection	Minimum[a]	Maximum[a]	Federal Maximum Contaminant Level	Frequency Above Federal MCL	NYSDEC Class GA Groundwater Standard or Relevant Standard[b]	Frequency Above NYSDEC Standard	Drinking Water PRG at 10^{-6} Cancer Risk	Frequency Above 10^{-6} Cancer Risk Drinking Water PRG
Gross alpha	1/20	–	26	15	1/20	15	–	NA	–
Gross beta	18/20	8.0	260	4 mrem	–[c]	1,000	0/20	NA	–
Radium-226	12/20	0.52	16	NA	0/20	3	1/20	0.0042	12/20
Radium-228	9/20	1.3	28	NA	1/20	3.0	3/20	0.018	9/20
Radium-226 plus Radium-228	20/20	1.82	44	5	–	5	4/20	0.022	20/20
Barium-140	1/20	–	20	NA	–	2	1/20	47	1/20
Potassium-40	4/20	43.2	108	NA	–	NA	–	1.2	4/20
Thorium-228	1/20	–	10.3	NA	–	7,000	0/20	0.0074	1/20

[a] Value shown does not include reported analytical uncertainty.

[b] Applicable NYSDEC Class GA groundwater standards (from 6 NYCRR 703, Water Quality Standards, Surface Waters and Groundwater) are used for four radioanalytical parameters and indicated as such in table. Because NYSDEC has no Class GA groundwater standard for the remaining radionuclides, the relevant standard used is the radionuclide concentration listed in 6 NYCRR 380.9, Prevention and Control of Environmental Pollution by Radioactive Materials, Table of Concentrations of Radioactive Material, Schedule 2. The Class GA Groundwater Standards are maximum allowable concentrations. The 380.9 standards apply to allowable concentrations in excess of natural background concentrations.

[c] Dose equivalent for gross beta results were not calculated because the results did not exceed NYSDEC Class GA standard.

Key:

PRG = Preliminary remediation goal for residential drinking water.

Table 2-18

SUMMARY OF MEDIA-SPECIFIC CRITERIA USED TO SELECT COPCs

Media	Site Background Concentration[a]	90th Level of Inorganics in Eastern U.S. Soils[b]	State Groundwater or Surface Water Standards or Federal MCL[c]	Region III Risk-Based Criteria[d]	NYS Annual Guideline Concentration[e]	NYS Recommended Soil Cleanup Goal[f]	New York State Sediment Criteria[g]
Surface soil	X	X	—	X	—	X	—
Subsurface soil	—	X	—	X	—	X	—
Surface water	—	—	X	—	—	—	—
Sediment	—	—	—	X	—	X	X
Groundwater	—	—	X	X	—	—	—
Soil gas	—	—	—	—	X	—	—

[a] Site Background Concentration: as discussed in Section 2.2.4.
[b] Shacklette and Boerngen 1984.
[c] NYSDEC October 1993, EPA 1993b.
[d] EPA 1993d.
[e] NYSDEC 1991b.
[f] NYSDEC 1992.
[g] NYSDEC 1989, November 1993.

Key:

X = Used.
— = Not used.
MCL = Maximum contaminant level.
NYS = New York State
NYSDEC = New York State Department of Environmental Conservation.

Table 2-19

SUMMARY OF CONTAMINANTS OF POTENTIAL CONCERN IN EACH MEDIUM

Chemical	Drainage Ditch Sediment[a]	Surface Soil	Subsurface Soil	Groundwater	Barge Canal Water[b]	Barge Canal Sediment[b]	Vegetables[c]
Volatiles							
Acetone			X				
Benzene				X			
Chlorobenzene				X			
Chloroethane				X			
Chloroform				X			
1,1-Dichloroethane			X	X			
1,2-Dichloroethane			X				
1,1-Dichloroethene				X			
1,2-Dichloroethene				X			
Ethylbenzene			X				
Methylene chloride			X				
Tetrachloroethene				X			
Toluene			X	X			
Trichloroethene			X	X			
1,1,1-Trichloroethane			X	X			
1,1,2-TCA				X			
Vinyl chloride				X			

Table 2-19

SUMMARY OF CONTAMINANTS OF POTENTIAL CONCERN IN EACH MEDIUM

Chemical	Drainage Ditch Sediment[a]	Surface Soil	Subsurface Soil	Groundwater	Barge Canal Water[b]	Barge Canal Sediment[b]	Vegetables[c]
Xylenes			X	X			
Semivolatiles							
Bis(2-ethylhexyl) phthalate				X	X		
Benzo(a)anthracene		X	X				
Benzo(a)pyrene		X	X				
Benzo(b)fluoranthene	X	X	X				
Benzo(k)fluoranthene		X	X				
Chrysene	X	X	X				
Dibenzo(a,h)anthracene		X	X				
Indeno(1,2,3-cd)pyrene		X	X				
2-Methyl phenol			X				
4-Methyl phenol			X	X			
Naphthalene		X					
Phenol			X	X			
N-Nitrosodiphenylamine			X				
Pesticides							
Endrin			X				
Heptachlor			X				

Table 2-19

SUMMARY OF CONTAMINANTS OF POTENTIAL CONCERN IN EACH MEDIUM

Chemical	Drainage Ditch Sediment[a]	Surface Soil	Subsurface Soil	Groundwater	Barge Canal Water[b]	Barge Canal Sediment[b]	Vegetables[c]
Heptachlor epoxide			X				
Lindane			X				
PCBs							
Aroclor 1248			X				
Aroclor 1254		X	X				
Aroclor 1260		X	X				
Metals							
Aluminum					X		
Antimony				X			
Arsenic				X			
Barium		X	X				
Beryllium				X			
Cadmium	X	X	X	X			
Chromium			X	X			X
Cobalt		X	X				
Copper	X	X	X	X		X	
Lead	X	X	X	X			X
Manganese			X	X			

Table 2-19

SUMMARY OF CONTAMINANTS OF POTENTIAL CONCERN IN EACH MEDIUM

Chemical	Drainage Ditch Sediment[a]	Surface Soil	Subsurface Soil	Groundwater	Barge Canal Water[b]	Barge Canal Sediment[b]	Vegetables[c]
Mercury		X	X			X	
Nickel		X	X	X			X
Selenium				X	X		X
Sodium				X			
Zinc	X	X	X	X		X	X
Cyanide				X			

[a] To determine COPCs for the drainage ditch sediments, concentrations detected in the on-site portion of the drainage ditch were compared with concentrations detected in the drainage ditch prior to its entry onto the Dearcop Farm site. If the highest concentrations were detected in the drainage ditch prior to its entrance onto the Dearcop Farm site, the contaminant was not considered site-related and therefore, was not selected as a COPC.

[b] Because of the lack of a predominant flow direction and the presence of other hazardous-waste sites near the Dearcop Farm site, the presence of contaminants in the Barge Canal water and the Barge Canal sediment do not appear to be site-related.

[c] No formal screening criteria are available to determine COPCs for vegetables. COPCs were selected based on exceedances of U.S. typical concentrations of these metals in vegetables.

3. EXPOSURE ASSESSMENT

3.1 EXPOSURE SETTING/POTENTIALLY EXPOSED POPULATIONS

The Dearcop Farm site is located in an urban area west of the Barge Canal (see Figure 1-1). A biking/jogging path is located between the site and the Barge Canal, and the I-390/I-490 interchange is situated on the northern 10 acres of the original 16-acre landfill. The eastern portion and southern border of the site are wooded, but the central portion of the site (the vacant lot area) is sparsely vegetated. Foundry sand, slag, scrap metal, wood, glass, and other debris are visible on the surface in this area. The site is bordered to the south and southwest by a medium-density residential area; the nearest residences are located immediately adjacent to the site on Dearcop Drive and Varian Lane. Two Class 2 inactive hazardous wastes sites, Olin Chemicals Corporation and McKee Road Industrial Dump, are situated southeast of the site on the eastern side of the Barge Canal. One Class 2A site, Chevron USA Tank Farm, is located approximately 0.5 miles south of the site. These sites are discussed in Section 1.4 of the Phase I RI.

Results of the Phase I and Phase II RI sampling indicate that VOCs, semivolatiles, and metals were detected in site soils and groundwater. In addition, several VOCs were detected in soil gas samples. Some waste materials disposed at the Dearcop Farm site were apparently used as fill in the adjacent residential area. Results of soil sampling in the residential areas indicate elevated levels of PAHs and metals.

The site can be entered from the south from Dearcop Drive and Varian Lane and from a bike path. Measures have been taken to reduce access to the site. "No trespassing" and warning signs have been posted around the perimeter, and in 1990 NYSDEC installed a chain-link fence at the ends of Dearcop Drive and Varian Lane and along the bike path. However, during the Phase I and II RI investigations, trespassers were observed on site

several times. In January 1993, to further restrict trespasser access, the New York State Department of Transportation (NYSDOT) installed a chain-link fence along the west side of the bike path adjacent to the site. There is evidence (e.g., worn paths, beer bottles) that trespassing still occurs.

Under current land-use conditions, site trespassers could potentially be exposed to contaminants in site surface soils and air. Residents living nearby could be exposed to site-related contaminants in apparently site-derived fill material in their yards. While it is possible that nearby residents might be exposed to fugitive dust from the site, this pathway is not likely to be significant because the residential area is not downwind from the site under prevailing wind conditions and a tree line between the site and the residential area serves as a windbreak when the residential area is downwind from the site.

Potential exposure risks to workers if excavation is conducted on the Dearcop Farm site were qualitatively assessed. Focus was placed primarily on excavation activities that might be associated with sewer lines from a nearby Monroe County Pure Waters pump station (including the sewer line that runs from the end of Varian Lane past the pump station and across the western portion of the site) and on the Buckeye Pipeline located along the Barge Canal outside of the Dearcop Farm property.

Five surface soil samples were evaluated to qualitatively assess potential risks to workers excavating on site. Maximum concentrations in Phase I RI soil samples SS-1, SS-2, SS-3, SS-4, and SS-14 were compared with eastern United States soils data from Shacklette and Boerngen, recommended NYSDEC soil cleanup goals, and EPA Region III risk-based concentrations (RBCs) for commercial ingestion of soil. These soil samples were located near the sewer line. Maximum metals concentrations were compared to the Shacklette and Boerngen data. If the maximum chemical concentration detected did not exceed the 90th percentile of Shacklette and Boerngen data, then the chemical was considered present at background concentrations. Several metals did not exceed the Shacklette and Boerngen data. The maximum concentrations of cadmium, copper, nickel, and zinc exceeded the NYSDEC-recommended value but were below the Region III EPA RBC. Lead was detected at a maximum concentration of 206 mg/kg, which exceeds the NYSDEC-recommended soil cleanup goal but does not exceed the benchmark human direct ingestion soil concentration (NYSDEC 1991a). Given their proximity to areas that might be excavated, several subsurface soil samples also were evaluated to determine if a potential health threat exists for

workers who may be excavating along the sewer lines or the Buckeye Pipeline. Maximum concentrations of chemicals detected in subsurface soil from TP-4, MW-9S, MW-1D, and MW-2D were compared to Shacklette and Boerngen data, NYSDEC-recommended soil cleanup levels, and EPA Region III RBCs for commercial ingestion of soil. None of the chemicals detected was found to exceed risk-based concentrations; therefore, it does not appear that excavation along existing sewer lines or along the Buckeye Pipeline would present a significant increased risk of adverse noncarcinogenic health effects or excess cancer risks for workers.

A future residential scenario was not developed for the landfill area because it is unlikely that homes will be built on the landfill itself. In addition, because Dearcop Drive and Varian Lane are surrounded on three sides by I-390, I-490, and the Barge Canal, additional development along Dearcop Drive and Varian Lane is not expected.

3.2 POTENTIAL EXPOSURE PATHWAYS

A schematic depiction of the potential exposure pathways is shown in the conceptual site model (Figure 1-2). These potential exposure pathways also are briefly discussed in Section 1.3.

3.2.1 Sources and Receiving Media

There appear to be two primary sources of environmental contamination associated with the Dearcop Farm site: the landfill wastes on site and the landfill wastes reportedly used as fill material in the yards of nearby residences. In addition, 1,000 gallons of unknown substances from DuPont reportedly were burned in an open pit on site. DuPont wastes disposed of at the site included acids, heavy metals, waste oil sludges, halogenated organics, and other compounds. The disposal method used for the combustible materials apparently was open burning. The former solvent burning area reportedly is where the I-490 eastbound/westbound highway median currently exists (EA Science and Technology 1988). This area was covered with clean fill prior to construction of I-490; therefore, direct contact with residues from the incineration of the DuPont wastes is unlikely.

3.2.2 Contaminant Fate and Transport

The fate and transport of contaminants in the environment are influenced by a variety of site- and chemical-specific factors. Environmental fate and transport processes for the COPCs at the Dearcop Farm site are discussed in Section 5 of the Phase I RI and are summarized briefly in this section. The majority of the Dearcop Farm site is covered with fill material that is not described in soil surveys; however, the native soil that underlies the fill material is mapped as the Hilton Loam. The Hilton Loam is characterized by deep, moderately well-drained, medium-textured, and moderately coarse-textured soils.

The VOCs are characterized by moderate-to-high vapor pressures, high water solubility, and little tendency for adsorption by soil and sediments; therefore, VOCs are generally considered to be highly mobile in the environment. At the surface, VOCs can volatilize to the atmosphere. In the subsurface, VOCs can migrate downward with infiltrating precipitation, eventually reaching groundwater, or migrate upward in soil gas to the atmosphere. Most organic contaminants undergo biotransformation or biodegradation in soil and groundwater when environmental conditions are favorable. Chlorinated methanes (chloroform, methylene chloride) and chlorinated ethenes (tetrachloroethene [perchloroethylene, PCE], trichloroethene [TCE], dichloroethene [DCE], and vinyl chloride) undergo sequential reductive dehalogenation under anoxic conditions (see Figure 5-1 of the Phase I RI report).

Most priority pollutant PAHs can be characterized as having low vapor pressure, low water solubility, low Henry's Law constants, high octanol-water partition coefficients, and high organic carbon partition coefficients (K_{oc}s). High K_{oc}s indicate that most PAHs are strongly sorbed to organic matter in the soils. Combined with low water solubilities, the rates of transport of most PAHs from the unsaturated zone via infiltration to the saturated zone will be extremely low. Low vapor pressures, low Henry's Law constants, and low K_{oc}s indicate that most PAHs will not readily volatilize from surface water or from surface soils. Exceptions to this generalization are some of the lower molecular weight PAHs such as acenaphthene, fluorene, fluoranthene, naphthalene, and pyrene, which have water solubilities greater than 100 μg/L. Although these compounds have high K_{oc}s (10^3 or greater) relative to other PAHs, their high solubility indicates that they are relatively mobile and may migrate to groundwater.

PCBs have low vapor pressures, but can volatilize from surface soils to the atmosphere. In general, PCBs are strongly adsorbed to soils and other organic matter and are relatively immobile in the subsurface. PCBs with higher levels of chlorination (Aroclors 1248, 1254, and 1260) are resistant to aerobic biodegradation but can degrade slowly by anaerobic processes. Bioconcentration factors for PCBs typically range from 10^4 to 10^6.

In general, pesticides have low water solubilities and a tendency to adsorb to soils, and therefore also are relatively immobile in the subsurface. Some pesticides are persistent in the environment and may bioaccumulate in aquatic environments.

Metals are also persistent in the environment, but their chemical and physical forms can change depending on environmental conditions. Metals in soils and sediment may be in a metallic form, sorbed or chelated by organic matter or oxides, sorbed on the exchange sites of soil colloids, or dissolved in soil water. Most metals are immobile in soil that has normal pH ranges, and they become significantly leachable only if acidic solutions percolate through the soils. Other environmental factors that influence metal mobility include soil clay content, organic content, oxidation-reduction potential, carbonate content, and groundwater chemistry.

Speciation of metals is also an important factor in their mobility. If the metals are present as oxides or hydroxides, they will remain relatively immobile in soils and sediments. If they are present as soluble salts, the most likely reaction that may occur is the hydrolysis of metals to oxides or hydroxides, or precipitation of low solubility sulfates or carbonates.

Contaminants bound to surface soils may be dispersed by surface runoff or by wind erosion. Surface runoff from the site does not discharge directly to the Barge Canal; rather, it enters a drainage ditch that discharges into the Barge Canal. The central portion of the site is sparsely vegetated and is covered with debris. Wind erosion of these soils is possible; however, given the prevailing wind direction and the presence of a tree line along the site's southern border that partially protects the site from the prevailing wind, residential exposure to these contaminants is unlikely. Site trespasser exposure to wind-borne particles is expected to be more significant.

3.2.3 Exposure Pathways Selected for Analysis

As shown in Figure 1-2, the following exposure pathways are potentially complete under existing site conditions for on-site contaminants at the Dearcop Farm site:

- Direct contact (incidental ingestion through hand-to-mouth contact and dermal contact) with contaminated surface soils on site by site trespassers;

- Inhalation of resuspended soil particles by site trespassers;

- Direct contact (incidental ingestion through hand-to-mouth contact and dermal contact) with contaminated surface soils (contaminated site media reportedly used as fill) in residential yards by nearby residents; and

- Consumption of vegetables grown in yards with fill material by nearby residents.

Future residential construction in the general vicinity of the Dearcop Farm site is not expected because Dearcop Drive and Varian Lane are surrounded on three sides by I-390, I-490, and the Barge Canal.

Groundwater at the site is classified GA by NYSDEC, indicating that it is a potential drinking water source; however, no existing residential wells receive groundwater from the site area, and the area is served by a public water supply system. Therefore, groundwater is not presently used as a source of potable water, nor is it expected to be used for this purpose in the future.

The potentially complete exposure pathways and the potential receptors are summarized in Table 3-1. The pathway/receptor combinations selected for quantitative evaluation also are identified, along with the reasons for their selection.

In addition to the exposure pathways evaluated in this risk assessment, two other studies were performed to evaluate two potential exposure scenarios for nearby residents. An indoor air quality survey was conducted to determine whether the contaminants (in particular, vinyl chloride) found in the groundwater from monitoring well MW-9S at the Dearcop Farm site were migrating into nearby residences through the soil. The results of this survey are presented in the Indoor Air Quality Sampling Report (E & E 1994b).

The second additional study was conducted by NYSDEC. This study was designed to further characterize and define PAH and metals concentrations in surface and subsurface soils in the residential area. The results of this study can be found in the Draft Phase III Remedial Investigation Residential Lot Soil Sampling Report, prepared by NYSDEC in September 1994 (NYSDEC 1994).

3.3 QUANTIFICATION OF EXPOSURE

This section describes how the quantitative exposure estimates were obtained. Section 3.3.1 describes how source media contaminant concentrations used in the exposure assessment calculations were selected or derived. Section 3.3.2 describes the contaminant migration models used to estimate exposure point concentrations, and Section 3.3.3 describes the exposure estimation calculations for each exposure pathway and route of exposure.

3.3.1 Exposure Media Contaminant Concentrations

Under existing conditions, the media of concern at the Dearcop Farm site are surface soil and air (release of soil vapors). For each source medium, the average contaminant concentrations were used to estimate exposure point concentrations for the average exposure case. When there was a sufficient number of samples (i.e., greater than 10) the 95th percent upper confidence limit (UCL) on the arithmetic mean was used to estimate the exposure point concentration for the RME case; otherwise, the maximum detected concentration was used to evaluate the RME case (EPA 1992d).

Soil Contaminant Concentrations

Phase I soil/sediment samples were analyzed for radionuclides. These data were not used in the human health risk assessment because there is evidence that the radionuclides previously identified as COPCs are present at essentially background concentrations, believed to be due to local naturally occurring radioactive material (NORM), or are associated with discrete areas of low-level, on-site NORM (areas of blue sandy material suspected to be glass fines). In addition, the on-site NORM does not belong to an acknowledged NORM waste sector (e.g., phosphate waste, metal mining waste) and most typical NORM exposure scenarios (e.g., downwind exposure to resuspended particulates, on-site exposure to indoor radon) do not apply to the inactive Dearcop Farm site where the NORM is present in very low concentrations in small, localized areas (EPA 1993a). Background surface soil samples taken during the Phase II RI and analyzed for radionuclides indicate that the detected radionuclides are present within background concentrations for this area; therefore, risks were not estimated for exposure to soils containing radionuclides.

Several soil samples were taken during the Phase I and Phase II RI. The concentrations detected in the Phase I RI residential surface soil samples and the Phase II RI residential

composite surface soil samples were used as exposure point concentrations to evaluate direct contact exposures for nearby residents. The Phase II RI surface soil samples were taken with a bias toward high-use areas such as swing sets and gardens. Surface soil samples collected on the Dearcop Farm site were used to estimate exposure point concentrations for site trespassers who could potentially be exposed to on-site contamination. Because excavation is not anticipated, subsurface soil concentrations were not used to estimate potential risks associated with dermal contact and soil ingestion.

Surface Water and Sediment Contaminant Concentrations

Surface water and sediment samples were collected from the drainage ditch and the Barge Canal during Phase I and Phase II RI sampling. During the Phase I RI, elevated levels of mercury and PCBs were detected in Barge Canal sediment. During the Phase II RI, additional sampling was conducted to aid in determining whether the contamination detected during the Phase I RI was site derived. The Barge Canal does not maintain a predominant flow direction; therefore, the samples could not be evaluated on an upstream/downstream basis. However, concentrations present in both the Barge Canal surface water and the sediment were compared with those media on site. For the two Phase I RI COPCs in the Barge Canal sediment (mercury and PCBs), sediment concentrations on site were compared with concentrations detected in Barge Canal sediment samples. Concentrations on site were lower than those detected in the Barge Canal sediment, which suggests that the Dearcop Farm site may not be the primary source of these contaminants. Elevated levels of PAHs also were detected in Barge Canal sediment; however, these concentrations cannot be attributed specifically to the Dearcop Farm site because these concentrations also were lower on site. It is possible that sediment containing relatively high levels of contaminants has migrated off site and into the canal, leaving sediment with relatively lower levels of contaminants on site; however, there is no apparent evidence to support this.

COPCs detected in the Barge Canal sediment were compared with concentrations of the same contaminants in sediment samples taken on site and sediment samples collected from the drainage ditch prior to its entrance onto the site. Results of this evaluation indicate that benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene were detected in sample SED-1 (a sample collected from the drainage ditch prior to its entrance on site) at higher concentrations than the levels detected in

the Barge Canal. This suggests that the site may not be a source of the PAHs detected in the Barge Canal sediment. Benzo(k)fluoranthene was present in the Barge Canal sediment at a concentration (1,200 μg/kg) slightly above the maximum concentration detected in sample SED-1 (1,100 μg/kg).

Three COPCs—aluminum, bis(2-ethylhexyl)phthalate, and selenium—were detected in the Barge Canal surface water. Bis(2-ethylhexyl)phthalate is a common laboratory contaminant. Concentrations of aluminum and selenium cannot be specifically attributed to the Dearcop Farm site because, as discussed earlier, the Barge Canal does not maintain a predominant flow direction and three other hazardous waste sites are located near the Barge Canal. The Barge Canal is the primary surface water receptor for all four of these sites; therefore, it would be difficult to attribute the presence of these contaminants to a particular site unless the chemical were a fingerprint contaminant. Several metals also were selected as Barge Canal sediment COPCs. However, concentrations of chromium, nickel, manganese, aluminum, and beryllium were detected at higher levels in Barge Canal sediment than in sediment on site. These data suggest that the contaminants detected in the Barge Canal sediment may not be site derived; therefore, any potential health risks that these chemicals pose to fishermen or swimmers in the Barge Canal cannot be specifically attributed to activities at the Dearcop Farm site.

Because contact with sediment is not expected to be frequent and the presence of contaminants in the Barge Canal surface water and sediment cannot be definitively attributed to the Dearcop Farm site, these data were not used for purposes of the quantitative risk assessment; they were used only to determine whether contaminants detected in these media could be specifically attributed to activities at the Dearcop Farm site.

Vegetable Contaminant Concentrations

Two vegetable samples were collected from residences where debris was noted in the soils and where elevated metals concentrations in soil were found. Both samples were used in calculating exposure point concentrations for vegetables. No background vegetable samples were taken. The concentrations of each chemical detected in the vegetables were carried through the quantitative risk assessment. The results of this evaluation are presented and discussed in Section 5.

Exposure Point Particulate Concentrations

Particulate concentrations for current exposure (site trespassers) were derived from the site surface soil concentrations using techniques discussed in Appendix A. In addition to contaminated particulates, volatilization of contaminants also may be of concern. The vapor inhalation pathways were screened against risk-based criteria in a preliminary risk evaluation for the site (E & E 1994a). This evaluation indicated that these pathways were unlikely to pose significant risks; therefore, they were not quantitatively evaluated in this risk assessment.

The exposure point concentrations used to evaluate the various exposure scenarios are provided in the risk estimation tables in Appendix B.

3.3.2 Contaminant Migration Modeling Methods

This section describes the modeling methods used to estimate the exposure point air concentrations that were used to evaluate exposures by the air pathways identified in Section 3.2.3. Details of the models and the parameter values used in the calculations are provided in Appendix A.

Particulate Emissions

Fugitive dust emissions due to wind erosion were estimated using the methods described in *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites* (EPA 1985). The emissions were estimated for the central portion of the site, which is sparsely vegetated and where evidence of surficial debris was discovered. Therefore, wind erosion is likely to occur in this area.

The inhalable particulate (PM10) emissions from the Dearcop Farm site due to wind erosion were estimated using the model for an "unlimited reservoir" of erodible soil as provided by the EPA (1985). The resulting total emissions were used in a near-field air dispersion "box model" to estimate COPC concentrations in the breathing zones on site (Gas Research Institute 1988) (see Appendix A).

Airborne Particle Concentrations On and Near the Site

After soil particles leave the ground as a result of wind erosion, they mix with the ambient air moving across the source area to produce a concentration of particles in the breathing zone. When a receptor is located on or near source areas such as those at the site

(where the downwind distance to the receptor is similar to, or less than, the size of the source area), the most important factors determining the breathing zone concentrations are the source strength (source area multiplied by average flux), wind speed, and vertical dispersion, which is a function of the distance to the receptor. Airborne soil particle concentrations to which receptors are potentially exposed were estimated by a near-field air dispersion "box model" that uses these factors to estimate the breathing-zone particle concentrations.

3.3.3 Exposure Estimation Methods

The exposure estimates described in this section combine the estimates of exposure media (soil, air, and vegetables) contaminant concentrations developed in the previous section with estimates of the frequency and duration of exposure that receptor populations are likely to experience. Estimates of various physiological parameters (e.g., breathing rate, body weight, and average life expectancy) also are included to estimate the average daily intake or absorbed dose of contaminants received by the receptor populations.

The equations used to estimate the exposure for each pathway and route of exposure are presented in tables later in this report. The parameter values used to evaluate the equations, the rationale for their selection, and a reference source also are provided. In most cases, standard default exposure factors from the EPA's Supplemental Risk Assessment Guidance for Superfund (EPA 1993e) or other EPA guidance were used. Exposure factors not specified in guidance documents were taken from the EPA's Exposure Factors Handbook (EPA 1989a) or were based on professional judgment.

The parameter values were all selected to correspond to the average and reasonable maximum exposure (RME) that an individual in the receptor population might experience. For the first exposure route, all parameters will be described and discussed in the text; for subsequent pathways, only the key parameters for that pathway and those parameters not previously mentioned will be described.

Scenario 1: Site Trespasser Exposure

Pathway 1A - Incidental Ingestion of Chemicals in Surface Soils (Table 3-2)

The contaminant concentrations in the surface soils (CS) are described in Section 3.3.1 and presented in Appendix B. The ingestion rate (IR) is the amount of soil a receptor might ingest through hand-to-mouth contact. Exposure frequency (EF) is the number of days

per year during which exposure is estimated to occur. For adolescents, an average exposure frequency of once per month and an RME exposure frequency of three times per month were estimated based on discussions with individuals who performed fieldwork during Phase I and Phase II RI sampling. They found that paths appeared worn and bottles were present, but not enough to indicate that the site is used regularly as a gathering place. Exposure duration (ED) is the total number of years during which exposure could occur. The body weight (BW) used is the average body weight assumed for the age group of the receptors. Averaging time (AT) is the period over which the estimated exposure is averaged. For noncarcinogens, the averaging time is equal to the exposure duration; for carcinogens, it is taken as the standard life expectancy of 70 years because the carcinogenic potency slope factors (described in Section 4.3.2) are based on lifetime exposures.

Pathway 1B - Dermal Contact with Chemicals in Soil (Table 3-3)

The absorption factor (ABS) is a chemical-specific value that describes the fraction of the applied dose of a chemical in soil that is absorbed through the skin. The current EPA guidance document on dermal exposure assessment (EPA 1992b) recommends ABS values for only two of the chemicals detected in soil at the Dearcop Farm site. The maximum recommended ABS values were used for PCBs (0.06) and cadmium (0.01). Dermal exposures to other chemicals in soil were not assessed quantitatively. The skin areas (SAs) that might come in contact with the soil were EPA-recommended default values (EPA 1992b). The soil-to-skin adherence factor (AF) is an estimate of the amount of soil that might adhere to the skin and serve as a source of exposure.

Pathway 1C - Inhalation of Airborne Soil Particles (Table 3-4)

The contaminant concentrations in air (CA) were modeled from source area soil concentrations as described in Section 3.3.2 and Appendix A. The inhalation rate (InhR) is the receptor's estimated breathing rate for a moderate level of activity. The exposure time (ET) is the number of hours per day the receptor is exposed to ambient air on site. The ET was estimated at one hour per day because the receptor is not expected to be located downwind of the open field area that could serve as a source of fugitive dust for the entire time spent on site.

Scenario 2: Nearby Residents Exposure

Pathway 2A - Incidental Ingestion of Chemicals in Surface Soils (Table 3-5)

The exposure parameters used in evaluating this pathway are the same as those described for Pathway 1A.

Pathway 2B - Dermal Contact with Chemicals in Soil (Table 3-6)

The exposure parameters used in evaluating this pathway are the same as those described for Pathway 1B.

Pathway 2C - Ingestion of Home-Grown Vegetables (Table 3-7)

The contaminant concentrations in the home-grown vegetables (CF) are described in Section 3.3.1. The IR is the amount of vegetable that a receptor might ingest per meal. The fraction ingested from a contaminated source is a pathway-specific value that expresses the fraction of contaminated food (FI).

3.3.4 Exposure Estimates

Estimates of the potential exposures to site contaminants that could occur were developed by combining the information and estimation methods presented in the previous two sections. Derivation of the exposure point contaminant concentrations were described in Section 3.3.3. The exposure estimates obtained by this process are given as lifetime average daily intakes (LADIs) for carcinogenic effects and as chronic daily intakes (CDIs) or subchronic daily intakes (SDIs) for noncarcinogenic effects for each complete pathway and exposure case in the risk estimation tables in Appendix B (see Tables B-2 through B-19). The exposure estimates are combined with toxicity estimates for each chemical (from Section 4) to obtain risk estimates.

Table 3-1

SUMMARY OF POTENTIALLY COMPLETE EXPOSURE PATHWAYS AND POTENTIAL RECEPTORS

Potentially Exposed Population	Exposure Routes, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Current Land Use			
Site Trespassers (Nearby Residents and Others)	Incidental ingestion of surface soils and dust via hand-to-mouth contact	Yes	Site is adjacent to a residential area, and access is not effectively restricted. Nearby residents and others can enter the site and contact contaminated soils.
	Dermal contact with contaminated surface soils	Yes	Site entry by foot is not completely restricted. Trespassers could contact contaminated soils.
	Inhalation of COPCs on airborne soil particles	Yes	The contaminated central portion of the site is not well vegetated; therefore, a potential for fugitive dust emission from the site exists.
	Incidental ingestion and dermal contact with the contaminated surface water and sediment	No	Elevated concentrations of metals and PAHs were detected in surface water and drainage ditch sediments. However, these concentrations do not appear to be site-related.
	Inhalation of airborne soil vapors on site	No	Contaminant modeling indicates that air concentrations are below levels of concern.
	Ingestion and dermal contact with contaminated groundwater	No	Area is served by a public water supply system.
Fishermen	Incidental ingestion and dermal contact with surface water and ingestion of fish	No	Contaminants detected in Barge Canal surface water do not appear to be site-related.

Table 3-1

SUMMARY OF POTENTIALLY COMPLETE EXPOSURE PATHWAYS AND POTENTIAL RECEPTORS

Potentially Exposed Population	Exposure Routes, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Nearby Off-site Residents	Inhalation of airborne soil particles off site	No	This pathway is unlikely to be significant because of the prevailing wind direction and the trees and vegetation surrounding the site.
	Inhalation of airborne soil vapors from site-related fill placed in residential yards (ambient air)	No	COPCs in soil include volatile and semivolatile chemicals that can migrate to the surface via soil gas and disperse into ambient air. However, contaminant modeling indicates that air concentrations are below levels of concern.
	Incidental ingestion or dermal contact with surface soils	Yes	Contaminated soil/debris from the site was reportedly used as fill in nearby residential yards.
	Inhalation of vapors via soil gas to building basements	No	Contaminant modeling indicates that air concentrations are below levels of concern.
	Ingestion of contaminated groundwater as drinking water	No	Area is served by a public water supply system.
	Showering or bathing with contaminated groundwater	No	Area is served by a public water supply system.
	Ingestion of home-grown vegetables	Yes	Site-derived fill material was reportedly used in nearby residential backyards, including gardens.

Source: Ecology and Environment, Inc. 1994.

Table 3-2

CURRENT SITE TRESPASSER EXPOSURE: PATHWAY 1A - INCIDENTAL INGESTION OF CHEMICALS IN SOIL (ADOLESCENT SITE TRESPASSERS)

Equation:

$$\text{Intake (mg/kg-day)} = \frac{\text{CS x IR x CF x EF x ED}}{\text{BW x AT}}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
IR = Ingestion Rate (mg soil/day)
CF = Conversion Factor (10^{-6} kg/mg)
EF = Exposure Frequency (day/years)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged, in days)

Variable	Receptor	Case	Value (Rationale/Source)
CS	Adolescent	Average	Average concentration in site soil
		RME	UCL concentration in site soil
IR	Adolescent	Average	50 mg/day (EPA 1989a)
		RME	100 mg/day (EPA 1991a)
EF	Adolescent	Average	12 days/year (once/month; professional judgment)
		RME	36 days/year (three times/month; professional judgment)
ED	Adolescent	Average/RME	10 years (entire duration of age group 6-16)
BW	Adolescent	Average/RME	42 kg (average body weight for age group 6-16; EPA 1989a)
AT	Adolescent	Average/RME	Pathway-specific period of exposure for noncarcinogenic (i.e., ED x 365 days/year), and 70-year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

Key:

RME = Reasonable Maximum Exposure.
UCL = Upper 95th percent confidence limit on the arithmetic mean.

Source: Ecology and Environment, Inc. 1994.

Table 3-3

CURRENT SITE TRESPASSER EXPOSURE: PATHWAY 1B - DERMAL CONTACT WITH CHEMICALS IN SOIL (ADOLESCENT SITE TRESPASSERS)

Equation:

$$\text{Absorbed Dose (mg/kg-day)} = \frac{\text{CS x ABS x CF x SA x AF x EF x ED}}{\text{BW x AT}}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
ABS = Absorption Factor (Unitless)
CF = Conversion Factor (10^{-6} kg/mg)
SA = Skin Surface Area Available for Contact (cm^2/event)
AF = Soil to Skin Adherence Factor (mg/cm^2)
EF = Exposure Frequency (events/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged, in days)

Variable	Receptor	Case	Value (Rationale/Source)
CS	Adolescent	Average	Average concentration in on-site soil
		RME	UCL concentration in on-site soil
ABS	Adolescent	Average/RME	Chemical specific values (EPA 1992c).
SA	Adolescent	Average	3,100 cm^2 (EPA 1992c)
		RME	3,800 cm^2 (EPA 1992c)
AF	Adolescent	Average	0.2 mg/cm^2 (EPA 1992c)
		RME	1.0 mg/cm^2 (EPA 1992c)
EF	Adolescent	Average	12 days/year (professional judgment)
		RME	36 days/year (professional judgment)
ED	Adolescent	Average/RME	10 years (entire duration of age group 6-16)
BW	Adolescent	Average/RME	42 kg (median body weight for age group 6-16; EPA 1989a)
AT	Adolescent	Average/RME	Pathway-specific period of exposure for non-carcinogenic effects (i.e., ED x 365 days/year); and 70-year lifetime for carcinogenic effects (i.e. 70 years x 365 days/year)

Key:

RME = Reasonable Maximum Exposure.
UCL = Upper 95th percent confidence limit on the arithmetic mean.

Source: Ecology and Environment, Inc. 1994.

Table 3-4

CURRENT SITE TRESPASSER EXPOSURE: PATHWAY 1C - INHALATION OF SOIL PARTICULATES (ADOLESCENT SITE TRESPASSERS)

Equation:

$$\text{Intake (mg/kg-day)} = \frac{\text{CA x InhR x ET x EF x ED}}{\text{BW x AT}}$$

where:

CA = Contaminant concentration in air (mg/m^3)
InhR = Inhalation rate (m^3/hour)
ET = Exposure time (hours/day)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged, in days)

Variable	Receptor	Case	Value (Rationale/Source)
CA	Adolescent	Average	Modeled value based on average concentrations in on-site soils
		RME	Modeled value based on UCL on-site soil concentrations
InhR	Adolescent	Average	1.4 m^3/hour (EPA 1989a)
		RME	1.7 m^3/hour (EPA 1989a)
ET	Adolescent	Average/RME	1 hour/day (professional judgment)
EF	Adolescent	Average	12 days/year (professional judgment)
		RME	36 days/year (professional judgment)
ED	Adolescent	Average/RME	10 years (entire duration of 6- to 16-year-old age group)
BW	Adolescent	Average/RME	42 kg (average [EPA 1989a])
AT	Adolescent	Average/RME	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70-year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

Key:

RME = Reasonable maximum exposure.
UCL = Upper 95th percent confidence limit on the arithmetic mean.

Source: Ecology and Environment, Inc. 1994.

Table 3-5

CURRENT RESIDENTIAL EXPOSURE: PATHWAY 2A - INCIDENTAL INGESTION OF CHEMICALS IN SOIL (ADULT AND CHILD RESIDENTS)

Equation:

$$\text{Intake (mg/kg-day)} = \frac{\text{CS x IR x CF x EF x ED}}{\text{BW x AT}}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
IR = Ingestion Rate (mg soil/day)
CF = Conversion Factor (10^{-6} kg/mg)
EF = Exposure Frequency (day/years)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged, in days)

Variable	Receptor	Case	Value (Rationale/Source)
CS	Adult/Child	Average	Average concentration in on-site soil
		RME	UCL concentration in on-site soil
IR	Adult	Average	50 mg/day (age groups greater than 6 years old; EPA 1993e)
		RME	100 mg/day (age groups greater than 6 years old; EPA 1993e)
	Child	Average	100 mg/day (children 0-6 years old; EPA 1993e)
		RME	200 mg/day (children 0-6 years old; EPA 1993e)
EF	Adult/Child	Average/RME	243 days/year (8 months/year)
ED	Adult	Average	7 years (EPA 1993e)
		RME	24 years (EPA 1993e)
	Child	Average	2 years (EPA 1993e)
		RME	6 years (entire duration of 1-6 year old age group)
BW	Adult	Average/RME	70 kg (average; EPA 1993e)
	Child	Average/RME	15 kg (children 1 through 6 years old; 50th percentile; EPA 1993e)
AT	Adult/Child	Average/RME	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70-year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

Key:

RME = Reasonable Maximum Exposure.
UCL = Upper 95th percent confidence limit on the arithmetic mean.

Source: Ecology and Environment, Inc. 1994.

Table 3-6

CURRENT RESIDENTIAL EXPOSURE: PATHWAY 2B - DERMAL CONTACT WITH CHEMICALS IN SOIL (ADULT AND CHILD RESIDENTS)

Equation:

$$\text{Absorbed Dose (mg/kg-day)} = \frac{\text{CS x ABS x CF x SA x AF x EF x ED}}{\text{BW x AT}}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
ABS = Absorption Factor (Unitless)
CF = Conversion Factor (10^{-6} kg/mg)
SA = Skin Surface Area Available for Contact (cm^2/event)
AF = Soil-to-Skin Adherence Factor (mg/cm^2)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged, in days)

Variable	Receptor	Case	Value (Rationale/Source)
CS	Adult/Child	Average	Average concentration in on-site soil
		RME	UCL concentrations in on-site soil
ABS	Adult/Child	Average/RME	Chemical-specific value (EPA 1992c)
SA	Adult	Average	5,000 cm^2 (EPA 1992c)
		RME	5,800 cm^2 (EPA 1992c)
	Child	Average	1,744 cm^2 (EPA 1992c)
		RME	2,000 cm^2 (EPA 1992c)
AF	Adult/Child	Average	0.2 mg/cm^2 (EPA 1992c)
		RME	1.0 mg/cm^2 (EPA 1992c)
EF	Adult/Child	Average/RME	243 days/year (8 months/year)
ED	Adult	Average	7 years (50th percentile time at one residence [EPA 1993e])
		RME	24 years (national upper bound time [90th percentile] at one residence; EPA 1993e)
	Child	Average	2 years (1993e)
		RME	6 years (duration of age group)
BW	Adult	Average/RME	70 kg (EPA 1993e)
	Child	Average/RME	15 kg (children 1-6 years old; 50th percentile; EPA 1989b)

Key at end of table.

Table 3-6

CURRENT RESIDENTIAL EXPOSURE:
PATHWAY 2B - DERMAL CONTACT WITH CHEMICALS IN SOIL
(ADULT AND CHILD RESIDENTS)

Variable	Receptor	Case	Value (Rationale/Source)
AT	Adult/Child	Average/RME	Pathway-specific period of exposure for noncarcinogenic (i.e., ED x 365 days/year), and 70-year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

Key:

RME = Reasonable Maximum Exposure.
UCL = Upper 95th percent confidence limit on the arithmetic mean.

Source: Ecology and Environment, Inc. 1994.

Table 3-7

CURRENT RESIDENTIAL EXPOSURE: FOOD PATHWAY PATHWAY 2C - INGESTION OF CONTAMINATED VEGETABLES (ADULT AND CHILD RESIDENTS)

Equation:

$$\text{Intake (mg/kg-day)} = \frac{CF \times IR \times FI \times EF \times ED}{BW \times AT}$$

where:

CF = Contaminant Concentration in Food (mg/kg)
IR = Ingestion Rate (kg/meal)
FI = Fraction Ingested from Contaminated Source (unitless)
EF = Exposure Frequency (meals/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged, in days)

Variable	Receptor	Case	Value (Rationale/Source)
CF	Adult/Child	Average	Concentration in vegetable
		RME	Concentration in vegetable
IR	Adult	Average	0.081 kg/meal (raw tomato) (Pao *et al.* 1982)
		RME	0.728 kg/meal (raw tomato) (Pao *et al.* 1982)
	Child	Average	0.112 kg/meal (cooked broccoli) (Pao *et al.* 1982)
		RME	0.680 kg/meal (cooked broccoli) (Pao *et al.* 1982)
FI	Adult/Child	Average	0.25 (EPA 1989a)
		RME	0.40 (EPA 1989a)
EF	Adult/Child	Average	60 days/year (professional judgment)
		RME	90 days/year (professional judgment)
ED	Adult	Average	9 years (50th percentile at one residence [EPA 1993e])
		RME	30 years (national upper bound time [90th percentile] at one residence; EPA 1993e)
	Child	Average	2 years (EPA 1993e)
		RME	6 years (entire duration of 1- to 6-year-old age group)
BW	Adult	Average/RME	70 kg (EPA 1993e)
	Child	Average/RME	15 kg (children 1 through 6 years old; 50th percentile; EPA 1986b)

Key at end of table.

Table 3-7

CURRENT RESIDENTIAL EXPOSURE: FOOD PATHWAY
PATHWAY 2C - INGESTION OF CONTAMINATED VEGETABLES
(ADULT AND CHILD RESIDENTS)

Variable	Receptor	Case	Value (Rationale/Source)
AT	Adult/Child	Average/RME	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70-year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

Key:

RME = Reasonable Maximum Exposure.

Source: Ecology and Environment, Inc. 1994.

4. TOXICITY ASSESSMENT

4.1 INTRODUCTION

The purpose of the toxicity assessment is to develop toxicity data for the COPCs at the Dearcop Farm site and to provide an estimate of the relationship between the extent of exposure to a contaminant and the likelihood and/or severity of adverse effects. The toxicity assessment will be accomplished in two steps: hazard identification and dose-response assessment.

Hazard identification is a qualitative description of the potential toxic properties of the COPCs at the site. Brief health effects summaries for the COPCs also are presented.

The dose-response evaluation is a process that results in a quantitative estimate or index of toxicity for each contaminant at the site. For carcinogens, the index is the slope factor (SF). For noncarcinogens, it is the reference dose (RfD). Procedures used to develop quantitative indices of toxicity and to incorporate toxicological information into the risk estimation process, as well as the quantitative indices of toxicity, are presented in Section 4.3. Uncertainties in the toxicity assessment process are discussed in Section 6.2.

4.2 HEALTH EFFECTS SUMMARIES

The health effects summaries describe the potential toxic properties of the COPCs at the Dearcop Farm site. For carcinogens, the weight-of-evidence category also is included. In most cases, the information in the summaries is drawn from the Public Health Statement in the Agency for Toxic Substances and Disease Registry's (ATSDR) toxicological profile for the chemical (ATSDR 1989-1993).

Acetone

Acetone is a clear, colorless liquid with a mintlike odor. It is a naturally occurring, volatile metabolite that has been identified in such plants as onions, grapes, apples, tomatoes, and morning glory. Acetone is a component of human breath and can also be emitted by volcanoes and forest fires. It is used as a solvent and as a chemical intermediate.

Most acetone used as a solvent or emitted from natural sources will ultimately be released to the air. If released to soil, it can both volatilize and leach to groundwater. Acetone also biodegrades readily.

The most likely human exposure to acetone is occupational. However, it is possible that the general population could be exposed to acetone through inhalation of automobile exhaust, solvent vapors, tobacco, and wood smoke; dermal or inhalation contact with consumer products such as nail polish remover; or by ingestion of food or water that contains acetone.

Acetone is a skin irritant and severe eye irritant. Following inhalation exposure, nasal effects, eye irritation, respiratory system effects, nausea, vomiting, and muscle weakness can occur. Kidney damage, metabolic changes, and coma can result from ingestion of large amounts of acetone. Increased liver and kidney weights and kidney toxicity are the critical, or most sensitive, toxic effects of acetone in animals and serve as the basis of the EPA's RfD.

The EPA has placed acetone in Group D, not classifiable as to human carcinogenicity.

Barium

Barium is a naturally occurring element that makes up 0.05% of the earth's crust. Barium compounds are used commercially in the metallurgic, paint, glass, ceramic, and electronics industries and for medicinal purposes.

Background levels of barium in the environment are very low. Barium can enter the body by inhalation or ingestion of food or water containing barium or its compounds. Little is known about the human health effects of barium. Most of the reported data come from studies of short-term exposure to large amounts of barium. Ingestion of barium can cause many effects, including breathing difficulty; increased blood pressure; changes in heart

rhythm, blood, and nerve reflexes; stomach irritation; swelling of the brain; and damage to the liver, kidney, heart, and spleen.

The critical, or most sensitive, effect from oral exposures seen in animal studies is a significant increase in blood pressure. Other long-term effects are changes in function and chemistry of the heart and reduced life span. Adverse effects associated with inhalation of barium dusts have not been well characterized. Smaller litter size and increased miscarriage rates in rats have been reported as the critical effects from inhalation of barium. There is no reliable information to determine whether barium can cause cancer in animals or people; therefore, EPA has not assigned barium a weight-of-evidence classification for carcinogenicity.

Cadmium

Cadmium is a naturally occurring element present in trace amounts in the earth's crust. Although it has other industrial applications, cadmium is used mostly in metal plating and the manufacture of pigments, batteries, and plastics.

Humans are exposed to small quantities of cadmium because it is widely distributed in air, water, soil, and food. Cadmium can enter the body by absorption from the stomach or intestines after ingestion of food or water containing cadmium, or by absorption from the lungs after inhalation of cadmium-containing dust, mists, or fumes. Food and cigarette smoke are probably the largest sources of cadmium for the general public. Very little cadmium enters the body through the skin.

Cadmium can cause a number of adverse health effects. Ingestion of high doses causes severe irritation to the stomach, leading to vomiting and diarrhea. Inhalation can lead to severe irritation of the lungs and may cause death. People have committed suicide by drinking water containing high levels of cadmium. There is very strong evidence that the kidney is the main target organ of cadmium toxicity following chronic exposure. Long-term ingestion of cadmium has caused kidney damage and fragile bones in humans. Long-term human exposure by the inhalation route may cause kidney damage and lung diseases such as emphysema. The critical, or most sensitive, effect of cadmium exposure is significant proteinuria, which is indicative of abnormal kidney function.

Long-term inhalation of air containing cadmium by workers is associated with an increased risk of lung cancer. Laboratory rats that breathe cadmium have increased cancer

rates. Studies of humans or animals have not demonstrated increased cancer rates from eating or drinking cadmium. The EPA classifies cadmium as a Group B1, probable human inhalation carcinogen, based on occupational studies.

Chromium

Chromium is a naturally occurring element used industrially in the manufacture of steel and other alloys. Its compounds are used in refractory brick for the metallurgical industry, and in metal plating (chromium VI), the manufacture of pigments (both chromium III and chromium VI), leather tanning (chromium III), and other processes. Exposure to chromium can result from inhalation of air containing chromium-bearing particles and ingestion of contaminated water or food. Chromium is considered an essential nutrient that helps to maintain normal glucose, cholesterol, and fat metabolism. The minimum daily requirement of chromium for optimal health has not been established, but ingestion of 20 to 500 μg/day has been estimated to be safe and adequate.

Two major forms of chromium found in the environment differ in their potential adverse health effects. Chromium VI is an irritant; short-term, high-level exposure can result in adverse effects at the site of contact, causing ulcers of the skin, irritation and perforation of the nasal mucosa, and irritation of the gastrointestinal tract. Minor to severe damage to the mucous membranes of the respiratory tract and to the skin have resulted from occupational exposure to as little as 0.1 mg/m^3 chromium VI compounds. The critical effect associated with inhalation of chromium VI is atrophy of the nasal mucosa. Chronic oral exposure to chromium VI also may cause adverse effects in the kidney and liver. Long-term occupational exposure to low levels of chromium VI compounds has been associated with lung cancer. EPA has classified chromium VI as a Group A human inhalation carcinogen.

Inhalation of the second form, chromium III, does not result in these effects and is the form thought to be an essential nutrient. The only effect observed in toxicological studies of chromium III was a decrease in liver and spleen weights in rats. Chromium III has not been assigned a weight-of-evidence classification for carcinogenicity by the EPA.

Cobalt

Cobalt occurs naturally in many different chemical forms. Pure cobalt is a steel-gray, shiny, hard metal that does not dissolve in water. Natural sources of cobalt include soil and

dust, seawater, volcanic eruptions, and forest fires. In the United States, cobalt is used to make alloys, large appliances, and colored pigments, and as a drier for paint and porcelain enameling used on steel bathroom fixtures. Man-made sources include by-products of the burning of coal and oil; exhaust from cars, trucks, and aircraft; industrial processes that use the metal or its compounds; and sewage sludge from cities.

Cobalt stimulates production of red blood cells in humans and is also used as treatment for anemia. Cobalt has been found to be a dermal sensitizer in humans. Individuals are sensitized following dermal or inhalation exposure, but ingestion of cobalt also may trigger dermatitis. The most sensitive indicators of cobalt toxicity by inhalation exposure are the effects on the respiratory system in both humans and animals. Interrelationships have been found to exist between cobalt and nickel sensitization, and it is possible that in people sensitized by nickel, exposure to cobalt may result in an allergic reaction. Indicators of cobalt toxicity following oral exposure include an increase in hemoglobin in both humans and animals and dermatitis in sensitized individuals. Following chronic occupational exposure, cobalt was found to be the cause of hard metal disease. Observed symptoms include respiratory irritation, wheezing, asthma, pneumonia, and fibrosis. These effects have been found to occur at exposure levels ranging from 0.003 to 0.893 mg/mg^3 over a period of 2 to 17 years. Work-related asthma was found in hard metals workers who were occupationally exposed to levels of cobalt ranging from 0.007 to 0.893 mg/mg^3. The only known essential nutritional function of cobalt is as a cofactor of vitamin B12, and there is no evidence that the intake of cobalt is limiting in the human diet; therefore, no recommended daily allowance (RDA) is deemed necessary for cobalt. The acceptable daily intake of cobalt in humans ranges from approximately 0.002 to 0.008 mg/kg/day in adults and 0.01 to 0.06 mg/kg/day in children.

The EPA has not assigned cobalt a weight-of-evidence classification for carcinogenicity.

Copper

Copper is a naturally occurring element that is used to make electrical wiring and water pipes and is a component of alloys such as bronze and brass. Copper compounds are used as fungicides to prevent plant disease, in water treatment, and in wood, leather, and fabric preservatives.

Copper may enter the body by inhalation, by ingestion of water or food containing copper, and by dermal contact. Copper is an essential element at low-dose levels but may induce toxic effects at high-dose levels. The critical, or most sensitive, effect is gastrointestinal irritation. The National Academy of Science has recommended 2 to 3 mg/day of copper as a safe and adequate daily intake. Long-term overexposure to copper dust can irritate the nose, mouth, and eyes and cause headaches, dizziness, nausea, and diarrhea. Ingestion of high concentrations of copper can cause vomiting, diarrhea, stomach cramps, and nausea. Liver and kidney damage and possibly death may result from long-term exposure.

Very young children are particularly susceptible to liver damage from ingestion of high concentrations of copper. In general, the seriousness of health effects of copper increase as the level and duration of exposure increase. Copper is not known to cause cancer or birth defects.

The EPA has placed copper in Group D, not classifiable as to human carcinogenicity.

Cyanide

Cyanides are naturally occurring substances found in a number of foods and plants and are produced by certain bacteria, fungi, and algae. Cyanides have industrial uses as intermediates in a variety of processes, and widespread use contributes to the numerous sources from which cyanide is released to the environment.

Cyanides are readily absorbed via the inhalation, oral, and dermal routes of exposure. Inhalation of hydrogen cyanide gas (HCN) is reportedly the quickest route of entry, resulting in the most rapid onset of toxic effects. Following absorption, cyanide is distributed throughout the body. It exerts acute toxic effects through interference with the electron transport chain, preventing the use of oxygen by cells.

At high concentrations, cyanide is acutely lethal. Inhalation of 270 ppm (300 mg/m^3) HCN results in nearly immediate death, whereas inhalation of 135 ppm (150 mg/m^3) is fatal after 30 minutes of exposure. Symptoms of acute exposure to HCN include tachycardia accompanied by palpitation, vertigo, buzzing in the ears, headache, epigastric burning, vomiting, general weakness, tremor, sensory obtusion, dyspnea, and loss of consciousness. The severity and rapidity of the onset of effects depends on the route, dose, and duration of exposure and the cyanide compound administered. Small doses of cyanide, like those

routinely inhaled by smokers, are metabolized by the body to thiocyanate and excreted in the urine without apparent adverse effects.

The nervous system is the major target organ in cyanide poisoning. Symptoms of chronic exposure to cyanide in humans and laboratory animals include enlargement of the thyroid gland, which is thought to result from an iodine imbalance caused by thiocyanate, the principal metabolite of cyanide. Evidence of possible developmental effects following exposure to concentrations of cyanide below fetotoxic doses is not conclusive.

Cyanides have not been associated with carcinogenic effects in humans or animals.

The EPA has placed cyanide in Group D, not classifiable as to human carcinogenicity.

1,1-Dichloroethane (1,1-DCA)

1,1-DCA is a man-made liquid chemical that is used industrially as a solvent and in the manufacture of other chemicals. When 1,1-DCA is released to surface water or surface soil, the chemical will evaporate into air. Although its water solubility is low, 1,1-DCA can migrate from soil into groundwater. Some 1,1-DCA found in the environment is a break-down product of 1,1,1-trichloroethane. Human exposure to 1,1-DCA can result from breathing contaminated air or eating or drinking contaminated food or water.

Relatively little information is available on the health effects of 1,1-DCA in humans or animals. 1,1-DCA was once used as a surgical anesthetic gas, although this use was discontinued when it was discovered that irregular heartbeats were induced at anesthetic doses. Exposure to high levels of 1,1-DCA in air has caused death in animals. Long-term exposure to high levels of 1,1-DCA has caused kidney damage in laboratory animals. In addition, exposure of pregnant rats to 1,1-DCA in air resulted in delayed development in the offspring. There is no evidence of similar harmful health effects in humans. There is no observed critical effect following exposure to 1,1-DCA.

One laboratory study suggests 1,1-DCA may cause increased tumors in rats and mice, but the results are inconclusive. There is no evidence that 1,1-DCA is carcinogenic in humans. In light of the results of animal studies, EPA has classified 1,1-DCA as a Group C, possible human carcinogen.

1,2-Dichloroethane (1,2-DCA)

1,2-DCA is a man-made liquid chemical used primarily in the synthesis of other solvents, particularly those that remove grease, glue, and dirt. In the past, 1,2-DCA was also found in commercial and household cleaning agents. When released to surface soil or surface water, 1,2-DCA evaporates readily into air, where it is broken down by sunlight. In the subsurface, 1,2-DCA migrates in soil gas and in groundwater. 1,2-DCA does not break down rapidly in soil, groundwater, and surface water.

Humans are exposed to 1,2-DCA primarily by breathing air containing its vapors or by drinking contaminated water. 1,2-DCA can also enter the body through the skin.

The lungs, heart, liver, and kidneys are the organs primarily affected in both humans and animals exposed to 1,2-DCA. Short-term exposure to 1,2-DCA in air may result in an increased susceptibility to infection and liver, kidney, and/or blood disorders. Effects seen in animals after long-term exposure to 1,2-DCA include liver, kidney, and/or heart disease, and death.

1,2-DCA has caused increased numbers of tumors in laboratory animals when administered in high doses in the diet or on the skin, and is classified as a Group B2, probable human carcinogen.

Endrin

Endrin is a man-made insecticide that was used primarily on cotton and grains. There are currently severe restrictions on the use of endrin in the United States. Endrin aldehyde is a very small component of technical-grade endrin and is also a degradation product of endrin that is produced at high temperatures and over long periods of time.

Endrin is very persistent in the environment. It is insoluble in water and adsorbs tightly to soil particles, making it nearly immobile in the subsurface. Small amounts of endrin may volatilize from soil or be carried by dust particles in the air.

Humans could be exposed to endrin by breathing contaminated air, drinking contaminated water, or through direct contact with contaminated soil or crops. The most likely route of possible exposure is ingestion of contaminated food products.

Endrin can be absorbed by the body through the skin, the lungs, and the stomach. In general, it is quickly metabolized and the metabolites are quickly eliminated from the body.

Endrin has been found to accumulate in the bodies of laboratory animals, primarily in the fatty tissues.

Exposure to endrin in humans can affect the central nervous system and, in some cases, causes death. In mild poisoning cases, however, recovery is usually rapid with no permanent effects.

The critical, or most sensitive, effects of endrin that serve as the basis of the EPA's RfD are convulsions and liver lesions. Endrin has exhibited no evidence of carcinogenicity in either humans or animals; however, the available studies are regarded by the EPA as inadequate and inconclusive.

The EPA has placed endrin in Group D, not classifiable as to human carcinogenicity.

Ethylbenzene

Ethylbenzene is a colorless liquid with a gasoline-like odor. Ethylbenzene occurs naturally in coal tar and petroleum, and it is found in many synthetic products, including paints, inks, and insecticides. Gasoline contains about 2% ethylbenzene by weight.

Ethylbenzene evaporates easily into the air from soil or water. People living in urban areas or near factories or highways may be exposed to ethylbenzene in the air. Indoor air, on average, contains more ethylbenzene than outside air due to buildup from household products such as cleaning products and paints. Tobacco smoke also contains ethylbenzene.

Ethylbenzene can enter the body through inhalation of vapors; through dermal contact with gasoline, paint vapors, or glue vapors; or through ingestion of food or water containing its residues.

Humans exposed to high levels of ethylbenzene have exhibited signs of dizziness and lethargy. Low-level exposure has been associated with eye and throat irritation. No deaths have been reported in humans exposed to ethylbenzene.

Short-term exposure to high concentrations of ethylbenzene in air is associated with liver, kidney, and nervous system damage, and death in laboratory animals. However, these results are unclear because of conflicting results and weaknesses in many of the studies. The critical, or most sensitive, effects that serve as the basis of the EPA's RfD are liver and kidney toxicity in rats.

There are no data on long-term health effects in humans exposed to ethylbenzene. One long-term study showed increased tumors in rats treated with ethylbenzene, but the study was flawed.

The EPA has placed ethylbenzene in Group D, not classifiable as to human carcinogenicity, because of limited laboratory data and lack of adequate human data.

Heptachlor/Heptachlor Epoxide (Lindane—gamma-BHC)

Heptachlor, a wide-spectrum, man-made pesticide, is a primary component of the commercial product chlordane. Chlordane was registered for use in the United States until 1988, when carcinogenicity concerns led to it being banned. Chlordane and heptachlor were used on more than 20 types of crops and in household applications to eliminate termites.

Heptachlor persists in the environment. In the environment and in the body, heptachlor is converted to heptachlor epoxide by chemical and microbial reactions. Heptachlor epoxide is more persistent than its parent compound. Since heptachlor was used on food crops and in homes, there have been residual levels in soils, ambient air, and indoor air in many parts of the United States.

Heptachlor and heptachlor epoxide can be absorbed by the body through dermal contact with contaminated media, inhalation of particles in ambient air, and ingestion of contaminated food or soils. Heptachlor epoxide can also pass directly from a mother's blood to an unborn baby through the placenta.

There are little data on the adverse health effects of heptachlor and heptachlor epoxide exposure in humans. Symptoms associated with human overexposure to these compounds include headache, dizziness, lack of coordination, irritability, weakness, and convulsions. The critical, or most sensitive, effect of heptachlor/heptachlor epoxide is increased liver weight in rats exposed to the chemicals. Chronic oral treatment with heptachlor has resulted in significant increases in hepatocellular carcinomas in mice.

The EPA has classified heptachlor and heptachlor epoxide as Group B2, probable human carcinogens.

Hexachlorocyclohexane

Hexachlorocyclohexane (HCH) is a man-made chemical that occurs in eight isomers. The isomers alpha (α), beta (β), gamma (γ), and delta (δ) are all solids that were used

primarily as pesticides. The γ isomer, also called lindane, was the active component. Commercial lindane contains a mixture of the isomers. The HCH insecticides were used on fruit, vegetable, and forest crops. Lindane is also used as a human medicine for head and body lice and scabies (Kwell). Since the late 1970s, HCH has not been used as a pesticide in the United States and manufacture of lindane has stopped. Lindane is still imported for use in consumer products including dog dips, shampoos, lotions, sprays, and creams.

Although HCH is no longer used as a pesticide, former widespread use of HCH has left α, β, γ, and δ isomers in the air, water, and soil. In general, lindane is persistent in the environment but will biodegrade slowly in soil and aerated water.

Human exposure can occur with contact with contaminated air, water, or food. HCH is found in meat and milk, as well as fruit and vegetables. In the body, HCH is absorbed rapidly from the digestive tract. In addition, lindane can cross the skin when used in lotions, creams, and shampoos.

Most data on human exposure are from occupational studies. Exposure to high concentrations of HCH can cause lung irritation, heart disorders, and blood disorders. Accidental and suicidal poisonings have caused death in some cases. The critical effects of lindane exposure are liver and kidney toxicity.

Long-term exposure to high doses of HCH has caused convulsions, kidney disease, liver disease, and death in laboratory animals. HCH was removed from use as an insecticide because α, β, and γ isomers cause liver cancer with long-term exposure of mice. EPA has classified γ and δ HCH as Group B2, probable human carcinogens, based on animal data.

Lead

Lead is a naturally occurring metal that is used in the manufacture of storage batteries and the production of ammunition and miscellaneous metal products (e.g., sheet lead, solder, and pipes). Other uses for lead are in the manufacturing of lead compounds, including gasoline additives and pigments. In recent years, the quantity of lead used in paints, gasoline additives, ammunition, and solder has been reduced because of lead's toxic effects.

Lead can enter the body via ingestion and inhalation. Although it may also enter the body through the skin, dermal absorption of inorganic lead compounds is less significant than absorption through other routes. Children appear to be the segment of the population at greatest risk from toxic effects of lead. Children absorb about 50% of ingested lead; adults

absorb only 5% to 15%. Initially, lead travels in the blood to the soft tissues (heart, liver, kidney, brain, etc.), then it is gradually redistributed to the bones and teeth, where it tends to remain. Children retain a larger fraction of the absorbed lead, about 57%, in the blood and soft tissue compartments, whereas in adults, roughly 95% of the total body burden of lead is found in bones and teeth.

The most serious effects associated with markedly elevated blood lead levels include neurotoxic effects such as irreversible brain damage. Health effects are the same for inhaled and ingested lead. At blood lead levels of 40 to 100 micrograms per deciliter (μg/dl), children have exhibited nerve damage, permanent mental retardation, colic, anemia, brain damage, and death. Chronic kidney disease is also evident at these levels. For most adults, such damage does not occur until blood lead levels exceed 100 to 120 μg/dl. At these levels, damage to the male reproductive system; miscarriages; anemia; severe digestive system symptoms; decreased reaction time; weakness in fingers, wrists, or ankles; and some increased risk of heart and circulatory system disease may be exhibited.

None of the epidemiology studies conducted to explore the relationship between lead exposure and increased cancer risk found any relationship. However, animal studies have shown increased kidney cancer and central nervous system cancer in rats and mice. The EPA has classified lead as a Group B2, probable human carcinogen.

Manganese

Manganese is a naturally occurring element used in the steel industry, in metallurgical processing, and as a component of dry cell batteries. Manganese is an essential element for humans and is a cofactor for a number of enzymatic reactions. A World Health Organization (WHO) committee concluded that an intake of 2 to 3 mg/day was adequate for adults.

Following inhalation of manganese dust, absorption into the bloodstream occurs only if particles are sufficiently small to penetrate deeply into the lungs. Long-term inhalation of manganese dust may result in a neurological disorder characterized by irritability, difficulty in walking, and speech disturbances. Short-term inhalation exposure has been associated with respiratory disease.

There are few reports of negative health effects in humans exposed to manganese in drinking water or food. Laboratory studies of animals exposed to manganese in water or food have demonstrated adverse health effects, including changes in brain chemical levels, low

birth weights in rats when mothers were exposed during pregnancy, slower than usual testes development, decreased rate of body weight gain, and weakness and muscle rigidity in monkeys. The critical effect of manganese exposure in humans is central nervous system alterations.

There are no human carcinogenicity data for manganese exposure. The data from some animal studies have shown increases in tumors in a small number of animals at high doses of manganese, but the data are inadequate to judge whether manganese can cause cancer. The EPA has placed manganese in Group D, not classifiable as to human carcinogenicity.

Mercury

Mercury is a naturally occurring element that exists in three oxidation states—metallic mercury (Hg^{o}), mercurous mercury (Hg_2^{++}), and mercuric mercury (Hg^{++})—and a variety of chemical forms. The most important with respect to human exposure are compounds of methyl mercury, mercuric mercury, and elemental mercury vapor.

Uptake of inorganic mercury and methyl mercury compounds occurs primarily through ingestion, with the major source of human exposure to methyl mercury being through the consumption of fish and shellfish. Mercury can also readily enter the body through inhalation of mercury vapor.

All forms of mercury, once absorbed, are distributed to tissues throughout the body via the bloodstream. The critical, or most sensitive, effects of inorganic mercury are kidney damage and central nervous system damage. Long-term exposure to all forms of mercury can permanently damage the brain, kidneys, and developing fetus. The form of mercury and route of exposure determine which health effects will be most severe. Mercury vapor and methyl mercury readily cross the blood-brain and placental barriers.

Prenatal life is very sensitive to methyl mercury poisoning, with effects in infants ranging from slowed mental and coordination development to a severe form of cerebral palsy. To date, these effects have been found to be irreversible. Depending upon the form, level of mercury taken in, and duration of exposure, effects on the adult nervous system can range from a reversible feeling of burning, or pins and needles, and feeling "out-of-sorts" to irreversible brain damage leading to permanent tremors and shakiness and constriction of the visual field.

Mercury has not been found to be carcinogenic in animals or humans and has been placed in Group D, not classifiable as to human carcinogenicity, by the EPA.

Methylene Chloride (Dichloromethane, MC)

Methylene chloride (MC) is a man-made liquid chemical that is widely used as an industrial solvent and as a paint stripper. Because MC evaporates easily, it is released into the environment mainly in air, where it is broken down by sunlight. MC released to water or soil tends to volatilize to air, but may migrate to groundwater. MC is formed during water chlorination, and small amounts of MC may be found in some public drinking water supplies.

Absorption into the body occurs readily following exposure by breathing vapors or accidental ingestion. Occupational worker exposure to high levels of MC in air has resulted in drowsiness, fatigue, lack of appetite, and light headedness. Other effects include impaired reaction time and coordination, numbness or tingling of fingers and toes, and intoxication. The critical, or most sensitive, effect of MC exposure is liver damage observed in rats.

Chronic exposure of laboratory animals to high concentrations of MC by inhalation has resulted in an increased incidence of liver and lung cancer in mice and rats. MC has not been shown to cause cancer in occupationally exposed humans. Based on results from animal studies, the EPA has classified MC as a Group B2, probable human carcinogen.

2-Methylphenol

2-Methylphenol (O-cresol) is a colorless, crystalline compound. Crude cresol (commercial cresol) is a mixture of aromatic compounds containing about 20% of o-cresol, 40% of m-cresol, and 30% of p-cresol with small amounts of phenol and xylenols. Cresols can be either solid or liquid, depending on how pure they are. Pure cresols are solid; mixtures tend to be liquid. The cresols are used in synthetic resins, explosives, and petroleum products, and the photographic, paint, and agricultural industries. They have been used for years as antiseptics, disinfectants, and insecticides. Cresols are present in wood and tobacco smoke, and are used as deodorizers, to dissolve substances, and as starting chemicals for making other chemicals.

People are most likely to be exposed to cresols by breathing, eating, or drinking media contaminated with them. Smokestacks of factories, electrical power plants, and oil refineries may send cresols into the air, and people who live close to these places may breathe

them. Acute exposures by all routes of absorption may cause muscular weakness, gastroenteric disturbances, severe depression, collapse, and death. While the effects are primarily on the central nervous system, edema of the lungs and injury of the kidneys, liver, pancreas, and spleen may also occur. Repeated exposures may result in digestive disturbances, damage to the liver and kidneys, and skin eruptions. Cresol has a marked corrosive action on tissues and may cause burns and dermatitis. In most cases, death is caused by respiratory failure. Most of the cresols that enter the body are quickly changed to other substances and leave the body in the urine within one day. Cresol is absorbed through the skin, open wounds, and mucous membranes of the gastroenteric and respiratory tracts. The rate of absorption through the skin depends primarily upon the size of the area exposed and secondarily on the concentration of the chemical applied. The major route of excretion of the cresols is with the urine, but considerable amounts may be excreted with bile and traces with exhaled air. The critical, or most sensitive, effect of 2-methylphenol exposure is decreased body weights and neurotoxicity in rats.

The EPA has classified 2-methylphenol as a Group C, possible human carcinogen.

4-Methylphenol

4-methylphenol (p-cresol) is produced through coal tar distillation, petroleum cracking, and synthesis from other chemicals. 4-methylphenol has been used to enhance fragrance in soaps, detergents, lotions, and perfumes in concentrations averaging 0.005%, 0.001%, 0.001%, and 0.04%, respectively. 4-methylphenol has been used as a synthetic flavoring substance in certain foods, although its usage has been regulated as a food additive by the EPA. Available information indicates that the most likely routes for human exposure to cresols are inhalation and dermal absorption. Cresols are absorbed across the mucous membranes of the respiratory and gastrointestinal tracts and through the skin or open wounds.

Cresols cause effects similar to those observed for phenol, including irritation, corrosion, hemorrhages, cytoplasmic destruction of the gastrointestinal tract, kidney tubule damage, nodular pneumonia, and hepatocellular necrosis. 4-methylphenol has also been found to induce hair depigmentation in mice when topically applied. There is limited evidence that 4-methylphenol causes cancer: three cases of cancer in humans exposed to cresols were reported by the Carcinogenic Assessment Group. Two cases of transitional cell sarcoma of the bladder were found in workers occupationally exposed to cresols or creosote,

and a case of squamous cell carcinoma of the vocal cords was found in petroleum refinery workers who had been exposed to cresol, dichloroethane, and chromic acid for several decades. Data also suggest that the three cresol isomers exhibit strong inhibitory potencies in mammalian tissue cultures. Therefore, it is likely that these compounds would be teratogenic in the placental barrier; however, no empirical data are available.

EPA has classified 4-methylphenol as a Group C, possible human carcinogen.

Nickel

Nickel is a naturally occurring metal found in small quantities in the earth's crust. Nickel is used industrially in making various steels and alloys and in electroplating. Exposure to nickel and nickel compounds may occur through inhalation of dust and particles, ingestion of food and drinking water containing nickel, and by absorption through the skin. Very small amounts of nickel have been shown to be essential nutrients for some species of animals and may be essential to humans. The critical, or most sensitive, effect of ingestion of nickel has been found to be decreased body and organ weight in rats.

Inhalation exposure to high levels of nickel and nickel compounds may have adverse effects on the lungs. Exposure by oral and inhalation routes can also affect the immune system, kidneys, and blood. Inhalation of nickel at concentrations greater than 0.001 mg/m^3 in air may cause immune system depression, lung irritation, and pulmonary disease. Death may result at concentrations greater than 0.1 mg/m^3.

Inhalation of nickel refinery dust has caused cancer of the lungs, nasal cavity, and voice box in humans. Nickel refinery dust and nickel subsulfide are classified as Group A human inhalation carcinogens.

N-Nitrosodiphenylamine

N-nitrosodiphenylamine is a blue-green crystalline substance that can be formed naturally or industrially. It is used in the rubber industry as a vulcanizing retarder.

If released to the environment, N-nitrosodiphenylamine would be expected to adhere to soil and sediments. It is unlikely to leach to groundwater.

Exposure of humans to N-nitrosodiphenylamine is generally unlikely. If exposure were to occur, the most probable location would be at a rubber plant, where inhalation, direct skin contact, or inadvertent ingestion of the substance could occur.

Information regarding the effects of human exposure to N-nitrosodiphenylamine is currently unavailable. However, studies have shown that animals fed N-nitrosodiphenylamine for long periods of time had swelling, changes in body weight, and bladder tumors. Death has also occurred following ingestion of high concentrations of N-nitrosodiphenylamine. There are no studies regarding the toxicity of N-nitrosodiphenylamine following inhalation exposure.

Based upon available animal studies in which bladder tumors were found in rats, N-nitrosodiphenylamine is classified as a Group B2, probable human carcinogen.

Phenol

Phenol, a white, crystalline mass or hygroscopic, translucent, needle-shaped crystal, is one of the many aromatic compounds present in coal tar. Phenol is used in the production or manufacture of a large variety of aromatic compounds, including explosives, fertilizers, coke, illuminating gas, lampblack, paints, paint removers, rubber, asbestos goods, wood preservatives, synthetic resins, textiles, drugs, pharmaceutical preparations, perfumes, bakelite, and other plastics (phenol-formaldehyde resins). Phenol also is used in the petroleum, leather, paper, soap, toy, tanning, dye, and agricultural industries. With few exceptions, human exposure in industry has been limited to accidental contact of phenol with the skin or to inhalation of phenol vapors.

The signs of phenol-induced acute illness in experimental animals resemble those observed in humans. In humans, phenol usually exerts a predominant action upon the central nervous system resulting in sudden collapse. In other mammals, the primary effects occur in the motor centers in the spinal cord, resulting in marked twitching and severe convulsions. Following absorption of a toxic dose, the heart rate first increases, then becomes slow and irregular. The blood pressure increases slightly at first, then decreases. The toxic effects of phenol are related directly to the amount of "free" phenol in the blood. In an acute intoxication, death is usually due to respiratory failure. An oral dose of 1 gram of phenol may be lethal to humans; however, some patients have survived ingestion of 65 grams of pure phenol or 120 grams of crude phenol. Swallowing phenol causes intense burning of the mouth and throat followed by stomach pain. In many cases, collapse occurs a few minutes after the phenol is swallowed.

Prolonged oral or subcutaneous administration can cause damage to the lungs, liver, kidneys, heart, and genitourinary tract. In animals, prolonged inhalation of vapors has induced respiratory difficulties, lung damage, loss of weight, and paralysis. Phenol is readily absorbed through the skin and from the stomach, uterus, intraperitoneal cavity, and subcutaneous tissues of man and animals.

Severe chronic poisoning in humans is characterized by systemic disorders such as digestive disturbances (including vomiting, difficulty in swallowing, ptyalism, diarrhea, and anorexia); by nervous disorders, with headache, fainting, vertigo, and mental disturbances; and possibly by skin eruptions. The disease is usually fatal when there is extensive damage to the liver and kidneys.

The critical effect of exposure to phenol is reduced fetal body weight in rats. The EPA has placed phenol in Group D, not classifiable as to human carcinogenicity.

Polychlorinated Biphenyls (PCBs)

PCBs are a group of man-made chemicals composed of 209 individual compounds. They have been used widely in heat transfer fluids, lubricants, and dielectric materials in transformers, capacitors, and other electrical equipment because of their insulating and flame-resistant properties. The industrial manufacture of PCBs in the United States was stopped in 1977 in response to the discovery that PCBs could accumulate and persist in the environment and might cause adverse health effects. Although PCBs are no longer manufactured in the United States, people can be exposed to PCBs spilled or leaked from older transformers, capacitors, and other kinds of equipment, and to low levels of PCBs widespread throughout the environment. PCBs bind tightly to soils and can be found in high concentrations in some freshwater and marine sediment. Some freshwater fish have bioconcentrated PCBs, and eating fish from contaminated areas may be a potentially significant source of human exposure.

PCBs can enter the body when fish, other foods, or water containing PCBs are ingested, when air that contains PCBs is breathed, or when skin contact with PCBs occurs. Skin irritations characterized by acne-like lesions and rashes and liver effects were the only significant adverse health effects reported in PCB-exposed workers. Epidemiological studies of workers occupationally exposed to PCBs thus far have not found any conclusive evidence of an increased incidence of cancer in these groups.

Effects of PCBs in experimentally exposed animals include liver damage, skin irritations, low birth weights and other reproductive problems, immunosuppression, and death. Some strains of rats and mice that were fed certain PCB mixtures throughout their lives showed increased incidence of cancer of the liver and other organs. Based on these animal studies, EPA has classified PCBs as Group B2, probable human carcinogens.

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs contain only carbon and hydrogen and consist of two or more fused benzene rings in linear, angular, or cluster arrangements. PAHs are formed during the incomplete burning of fossil fuel, garbage, or any organic matter. PAHs produced by burning may be carried into the air on dust particles and distributed into water and soil. In general, PAHs do not evaporate easily and do not dissolve in water.

Exposure to PAHs may occur by inhaling airborne particles, drinking water, or accidentally ingesting soil or dust containing PAHs. In addition, smoking tobacco or eating charcoal-broiled food are common routes of exposure to PAHs.

Some PAHs are known carcinogens, and potential health effects caused by PAHs are usually discussed in terms of an individual PAH compound's carcinogenic or noncarcinogenic effects. Little attention has been paid to noncarcinogenic effects of PAHs. Rapidly growing tissues, such as the intestinal lining, bone marrow, lymphoid organs, blood cells, and testes seem to be especially susceptible targets to noncarcinogenic effects. Concentrations of 150 mg/kg or more administered to laboratory animals have been shown to inhibit body growth.

Exposure to benzo(a)pyrene (B[a]P) and other carcinogenic PAHs can cause cancer at the point of exposure. B(a)P is used as the surrogate for evaluation of the toxicity of all of the Class B2 carcinogenic PAHs because only B(a)P has been assigned a slope factor by EPA. Animals exposed to high levels of B(a)P in air develop lung tumors; when exposed via the dietary route, they develop stomach tumors; and when B(a)P is painted on skin, animals develop skin tumors. Although RfDs and SFs for dermal exposure to other chemicals are routinely extrapolated from oral-route values, it is inappropriate to use the oral SF of B(a)P to evaluate carcinogenic risks from dermal exposure because dermal exposure to B(a)P directly causes skin cancer.

The EPA has classified benzo(a)pyrene as a Group B2, probable human carcinogen.

Toluene

Toluene is used as a solvent in the production of a variety of products and as a constituent in the formulation of gasoline and aviation fuels. Toluene can enter and affect the body if it is inhaled, comes in contact with the eyes or skin, or is swallowed.

Exposure to toluene can cause many central nervous system effects. Toluene may cause fatigue, weakness, confusion, headache, dizziness, drowsiness, and irritation of the eyes, respiratory tract, and skin. These symptoms have been reported in association with occupational exposure to airborne concentrations of toluene ranging from 50 ppm (189 mg/m^3) to 1,500 ppm (5,660 mg/m^3). Symptoms generally increase in severity with increased exposures. The critical, or most sensitive, effects of toluene that serve as the basis of the EPA RfD are changes in liver and kidney weights in rats.

Toluene does not appear to cause cancer in animals or humans. No increased risk of cancer was detected in studies of occupationally exposed humans. Similarly, toluene did not cause cancer in rats and mice exposed via inhalation.

The EPA has placed toluene in Group D, not classifiable as to human carcinogenicity.

1,1,1-Trichloroethane (1,1,1-TCA)

1,1,1-TCA is a man-made chemical that has many industrial and household uses. It is used as a cleaning solvent to remove oil or grease from manufactured metal parts, and as a solvent to dissolve other substances such as glue and paint. Consumer products such as spot removers may contain 1,1,1-TCA. Much of the 1,1,1-TCA produced in the United States is emitted into the atmosphere as a result of evaporation during use. 1,1,1-TCA released onto or into the ground can migrate into groundwater.

Inhalation is the major route of exposure in humans, but humans can also be exposed by consuming contaminated food and water and by skin contact with 1,1,1-TCA contaminated soil and water. 1,1,1-TCA is readily absorbed into the body following exposure by inhalation or ingestion. It readily leaves the body with exhaled air.

Inhalation of high levels of 1,1,1-TCA for a short time by humans has resulted in central nervous system effects such as dizziness, light headedness, and loss of balance and coordination. These health effects are readily reversible when exposure stops. Studies in animals and humans have shown that mild liver effects result from long-term exposure. Kidney damage has also been reported in animal studies.

Cancer studies were performed on mice and rats dosed orally. No consistent pattern of an increased incidence of cancer was found, but the study was of limited value because of the death of many of the animals. It is not known whether 1,1,1-TCA causes cancer in humans. The EPA has placed 1,1,1-TCA in Group D, not classifiable as to human carcinogenicity.

Trichloroethene (TCE)

TCE is a man-made chemical widely used as a cleaning agent and solvent for degreasing operations. Most TCE released into surface water or surficial soil will rapidly evaporate into the air. In the subsurface, TCE is moderately to highly mobile and can migrate to groundwater. TCE biodegrades very slowly in subsurface soils and groundwater. Microbial degradation products include dichloroethylene and vinyl chloride.

Humans are most likely to be exposed to TCE in air. TCE also may occur in drinking water supplies and consumer products, including metal cleaners, spot removers, rug cleaning fluids, paints, and paint removers. TCE may cause adverse health effects following exposure via inhalation, ingestion, or skin or eye contact. Exposure to high levels of TCE can cause central nervous system effects, including drowsiness, dizziness, headache, blurred vision, lack of coordination, mental confusion, flushed skin, tremors, nausea, vomiting, fatigue, irregular heartbeat, and, in some cases, death. In the past, TCE was used as an anesthetic, but that use was discontinued when it was found to cause irregular heartbeats. Chronic exposure to TCE can cause liver damage and skin reactions, as well as central nervous system effects.

Exposure of laboratory animals to TCE has been associated with an increased incidence of a variety of tumors, including kidney, liver, and lung cancers. However, it is uncertain whether people exposed to TCE have a higher risk of cancer. TCE was considered a Group B2, probable human carcinogen; however, that designation is currently undergoing review.

Xylenes

Xylenes are natural components of coal tar and petroleum; however, the majority of xylenes used commercially are man-made. Xylenes are used in solvent mixtures and cleaning agents and are components of gasoline and other fuels. There are three isomers of xylene

(ortho-, meta-, and para-xylene), which can occur as a mixture, and are referred to herein as xylene.

Xylene evaporates easily and is widespread in the environment. Xylene is released from industrial sources, automobile exhaust, use of xylene as a solvent, and chemical waste disposal sites. Xylene can be detected in air in cities and industrial areas and in some public drinking water supplies.

Exposure to xylene may occur by breathing xylene vapors, or eating or drinking xylene-contaminated food or water. Xylene is rapidly absorbed following inhalation or ingestion. Short-term human exposure to high levels of xylene causes irritation of the skin, eyes, nose, and throat; increased reaction time to a visual stimulus; impaired memory; stomach discomfort; and possible changes in the liver and kidneys. Xylene may be fatal if large enough concentrations are inhaled or ingested. There are no studies regarding the long-term effects of inhalation or ingestion of xylene by humans.

Exposure of laboratory animals to xylene in air resulted in changes in the cardiovascular system, changes in liver weights, and hearing loss. Decreased body weight and increased numbers of birth defects in unborn rats were observed at high concentrations of xylene. The effects of long-term exposure to low concentrations of xylene has not been well studied in animals. The critical, or most sensitive, effects are decreased body weight in laboratory animals, hyperactivity, and increased mortality.

Oral exposure to xylene has not been found to cause increased cancer in rats or mice. There are no human carcinogenicity data. The EPA has placed xylene in Group D, not classifiable as to human carcinogenicity.

Zinc

Zinc is a naturally occurring element that can be found in a variety of compounds. Zinc has many industrial uses, including galvanizing steel and manufacturing zinc-containing alloys such as brass. Zinc is an essential nutrient, and an inadequate amount of zinc in the diet will lead to adverse health effects.

People are exposed to low concentrations of zinc every day in air, water, soil, and food. Sources of zinc exposure include the drinking of water containing elevated levels of zinc and the breathing of air containing elevated levels of zinc from galvanizing, smelting,

welding, or brass foundry operations. Drinking water is thought to be the most significant exposure route to zinc at hazardous waste sites.

Zinc appears to be toxic only at levels at least 10 times higher than the recommended daily allowance. Symptoms of overexposure may include severe diarrhea, stomach cramping, nausea, and vomiting. Serious damage to the digestive system can occur if too much zinc is ingested over a long period of time. Ingesting too much zinc can cause deficiency in other nutrients such as iron (anemia) and copper. Anemia is the critical, or most sensitive, effect caused by zinc overexposure. Inhalation of zinc fumes or dusts has been associated with a condition called "metal fume fever" characterized by flulike symptoms, including throat irritation, body aches, weakness, and fatigue.

Zinc is not thought to cause cancer or birth defects. The EPA has placed zinc in Group D, not classifiable as to human carcinogenicity.

4.3 QUANTITATIVE INDICES OF TOXICITY

Quantitative indices of toxicity were compiled for the dose-response assessment to be used in estimating the relationship between the extent of exposure to a contaminant and the potential increased likelihood and/or severity of adverse effects. The methods for deriving indices of toxicity and estimating potential adverse effects are presented below.

4.3.1 Categorization of Chemicals as Carcinogens or Noncarcinogens

For the purpose of this risk assessment, COPCs were classified into two groups: potential carcinogens and noncarcinogens. The risks posed by these two types of compounds are assessed differently because noncarcinogens generally exhibit a threshold dose below which no adverse effects occur, whereas no such threshold can be proven to exist for carcinogens.

As used here, the term *carcinogen* means any chemical for which there is sufficient evidence that exposure may result in continuing uncontrolled cell division (cancer) in humans and/or animals. Conversely, the term *noncarcinogen* means any chemical for which the carcinogenic evidence is negative or insufficient. These definitions are dynamic; compounds may be reclassified any time additional evidence becomes available that shifts the weight-of-evidence one way or the other.

COPCs have been classified as carcinogens or noncarcinogens based on weight-of-evidence criteria contained in the EPA's Carcinogenicity Evaluation Guidelines (EPA 1986). Table 4-1 summarizes the five EPA weight-of-evidence categories. According to these EPA guidelines, chemicals in the first two groups, A and B (B1 or B2), are considered human carcinogens or probable human carcinogens based on sufficient evidence and should be the subject of nonthreshold carcinogenic risk estimation procedures. Depending upon the quality of the data, Group C chemicals also may be subjected to these procedures. The remaining chemicals in Groups D and E are defined as noncarcinogens and should be subjected to threshold-based toxicological risk estimation procedures.

Exposure to some chemicals may result in both carcinogenic and noncarcinogenic effects. In these cases, both the carcinogenic and noncarcinogenic effects were evaluated and considered in the risk assessment process.

4.3.2 Assessment of Carcinogens

In contrast to noncarcinogenic effects for which thresholds are thought to exist, scientists have been unable to demonstrate experimentally a threshold for carcinogenic effects. This has led to the assumption by federal regulatory agencies (e.g., EPA, Food and Drug Administration [FDA], and Occupational Safety and Health Administration [OSHA]) that any exposure to a carcinogen theoretically entails some finite risk of cancer. However, depending on the potency of a specific carcinogen and the level of exposure, such a risk could be vanishingly small.

Scientists have developed several mathematical models to estimate low-dose carcinogenic risks from observed high-dose risks. Consistent with current theories of carcinogenesis, EPA has selected the linearized multistage model based on prudent public health policy (EPA 1986). In addition to using the linearized multistage model, the EPA uses the upper 95% confidence limit for doses or concentrations in animal or human studies to estimate low-dose SFs. By using these procedures, the regulatory agencies are unlikely to underestimate the actual slope factors (formerly called carcinogenic potency factors) for humans.

Using SFs, lifetime excess cancer risks can be estimated by:

$$\text{Risk} = \Sigma \, \text{LADI}_j \times \text{SF}_j$$

where:

$LADI_j$ = exposure route-specific lifetime average daily intake

SF_j = route-specific slope factor.

Using the multistage model, the carcinogenic risks for the oral, dermal, and inhalation routes of exposure are calculated as follows:

$$Risk = LADI_oSF_o + LADI_dSF_o + LADI_iSF_i$$

where:

o = oral route

d = dermal route

i = inhalation route.

SFs for the COPCs for oral and inhalation exposure routes are presented in Table 4-2. The preferred source of SFs is EPA's Integrated Risk Information System (IRIS) database, which contains confirmed values reflecting the consensus judgment of the agency. The second choice is EPA's Health Effects Assessment Summary Tables (HEAST), which contain information taken from final documents prepared by the EPA Office of Health and Environmental Assessment.

Currently, IRIS reports units risks, but not SFs, for inhalation of carcinogens. For Table 4-2, inhalation route unit risks from IRIS have been converted to SFs using the following equation:

$$\frac{SF_1}{(mg/kg/day)^{-1}} = \frac{Unit\ Risk}{(\mu g/m^3)} \times \frac{70\ kg \times 1{,}000\ \mu g/mg}{20\ m^3/day}$$

The EPA weight-of-evidence classification for the chemical and the type of cancer that may be associated with exposure to the chemical are also included on Table 4-2.

4.3.3 Assessment of Noncarcinogens

Risks associated with noncarcinogenic effects (e.g., organ damage, immunological effects, birth defects, and skin irritation) are usually assessed by comparing the estimated average daily intake to the acceptable daily dose, now called the RfD by EPA. The RfD is selected by identifying the lowest reliable no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL) in the scientific literature, then applying a suitable uncertainty factor (usually ranging from 10 to 1,000) to allow for differences between the study conditions and the human exposure situation to which the RfD is to be applied. NOAELs and LOAELs can be derived from either human epidemiological studies or animal studies; however, they are usually based on laboratory experiments on animals in which relatively high doses are used. Consequently, uncertainty or safety factors are applied when deriving RfDs to compensate for data limitations inherent in the underlying experiments and for the lack of precision created by extrapolating from high doses in animals to lower doses in humans. The application of uncertainty factors in the derivation of RfDs is explained in RAGS-HHEM (EPA 1989b) and outlined below.

The RfD is derived from the NOAEL (or LOAEL) for the critical toxic effect by consistent application of uncertainty factors (UFs) and a modifying factor (MF). The uncertainty factors generally are multiples of 10 (although values less than 10 are sometimes used), with each factor representing a specific area of uncertainty inherent in the extrapolation from the available data. The bases for application of different uncertainty factors are explained below.

- A UF of 10 is used to account for variation in the general population and is intended to protect sensitive subpopulations (e.g., elderly, children).

- A UF of 10 is used when extrapolating from animals to humans. This factor is intended to account for the interspecies variability between humans and other mammals.

- A UF of 10 is used when a NOAEL derived from a subchronic instead of a chronic study is used as the basis for a chronic RfD.

- A UF of 10 is used when a LOAEL is used instead of a NOAEL. This factor is intended to account for the uncertainty associated with extrapolating from LOAELs to NOAELs

In addition to the UFs listed above, a modifying factor (MF) is applied:

- An MF ranging from >0 to 10 is included to reflect a qualitative professional assessment of additional uncertainties in the critical study and in the entire data base for the chemical not explicitly addressed by the preceding uncertainty factors. The default value for the MF is 1.

To calculate the RfD, the appropriate NOAEL (or the LOAEL if a suitable NOAEL is not available) is divided by the product of all of the applicable uncertainty factors and the modifying factor. That is:

$$RfD = \frac{NOAEL \text{ or } LOAEL}{(UF_1 \times UF_2 \ldots \times MF)}$$

Oral RfDs are typically expressed as one significant figure in units of mg/kg/day.

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a portion of the lifetime, in the case of a subchronic RfD, or during the entire lifetime, in the case of a chronic RfD. The RfD is used as a reference point for gauging the potential effects of other exposures. Usually, exposures that are less than the RfD are not likely to be associated with health risks. As the frequency of exposures exceeding the RfD increases and as the size of the excess increases, the probability increases that adverse health effects may be observed in a human population. Nonetheless, a clear distinction that would categorize all exposures below the RfD as "acceptable" (risk-free) and all exposures in excess of the RfD as "unacceptable" (causing adverse effects) cannot be made (HEAST 1991). Noncarcinogenic risks are usually assessed by calculating a hazard quotient, which is the ratio of the estimated exposure to the RfD as follows:

$$HQ = \frac{ADI}{RfD}$$

where:

HQ =Hazard Quotient
ADI =Average Daily Intake (exposure)
RfD =Reference Dose (acceptable daily intake).

A hazard quotient greater than 1 indicates that adverse effects may be possible, whereas a value less than 1 means that adverse effects would not be expected. The higher the hazard quotient is above 1, the more likely it is that adverse effects could occur.

The EPA is in the process of developing subchronic RfDs based on potential noncarcinogenic effects associated with exposure durations ranging from a few weeks to seven years. Short-term exposures can occur when an activity resulting in exposure is performed for a limited period of time or when a chemical degrades or disperses to negligible concentrations within a short period. The hazard quotient for subchronic exposure is obtained by dividing the estimated average daily dose by the subchronic RfDs. Exposures of greater than 7 years duration (adult and adolescent exposures) were evaluated using chronic RfDs. Exposures of 7 years duration or less (exposures to young children 0 to 6 years of age) were evaluated using subchronic RfDs.

Chronic and subchronic RfDs for the oral and inhalation exposure routes are presented in Table 4-3. As with the SFs, the preferred source for RfDs was IRIS and the secondary source was HEAST. For some chemicals, following recommendations in the HEAST manual, RfDs were obtained from the EPA's Environmental Criteria and Assessment Office's (ECAO's) Superfund Health Risk Technical Support Center.

Currently, all of the sources listed above report reference concentrations (RfCs) rather than RfDs for the inhalation route. For Table 4-3, inhalation route RfCs were converted to RfDs using the following equation:

$$\frac{RfD}{(mg/kg\text{-}day)} = \frac{RFC}{(mg/m^3)} \times \frac{20m^3/day}{70kg}$$

Other entries in the table that have not been discussed previously are as follows: the confidence level indicates the degree of confidence that should be placed in the RfD value and is usually obtained from the Integrated Risk Information System (IRIS) entry for a chemical; the critical effect is the effect or target organ affected by the smallest dose of the chemical that produces any adverse effect and that serves as the basis for the RfD; and the RfD source is the reference for the RfD. The fourth choice is to use values from other EPA documents, and the fifth choice is to use values derived directly from the general literature. The RfD basis is the vehicle in which the chemical was administered or the medium of exposure in the study(ies) that served as the basis for the RfD.

4.3.4 Route Extrapolation of Reference Doses and Slope Factors

Once substances have been absorbed via the oral or dermal routes, their distribution, metabolism, and elimination patterns (biokinetics) are usually similar. For this reason, and because dermal route RfDs and SFs are usually not available, oral route RfDs and SFs are commonly used to evaluate exposures to substances by the dermal route. When this is done, the oral RfDs and SFs are adjusted to account for differences in a chemical's absorption between the oral and dermal routes of exposure. This approach is not appropriate and is not used if the adverse effect occurs at the point of exposure. For example, the oral route RfD for copper is based on gastrointestinal tract irritation; therefore, dermal and inhalation route RfDs for copper cannot be extrapolated from the oral RfD. Although inhalation route biokinetics differ more from oral route kinetics than do the dermal route kinetics, oral RfDs and SFs may also be used to evaluate inhalation exposures (except in the case of exposure point effects) if inhalation route RfDs and SFs are not available, and *vice versa*. Extrapolation of toxicological indices from one route to another is inappropriate if the critical effect for either route is at the point of contact.

4.3.5 Assessment of Lead

There are no verified or EPA consensus toxicological indices available for lead in either IRIS or HEAST. The absence of authoritative toxicological indices reflects the

scientific community's inability to agree on a threshold dose for lead's noncarcinogenic effects or to satisfactorily estimate its carcinogenic potency, despite a large body of scientific literature on its toxicological effects (IRIS 1994).

Because there currently are no EPA-approved toxicological indices for lead, risks associated with potential exposures to lead cannot be quantitatively assessed. Consequently, for the purposes of this risk assessment, lead concentrations found in soils were assessed by comparison with background soil concentrations. The concentrations of lead in surface soil on and around the site range from 5.3 mg/kg to 820 mg/kg, whereas subsurface soil lead concentrations range from 2.0 mg/kg to 2,740 mg/kg. Five background surface soil samples also were collected. The concentrations of lead in these samples range from 22.9 mg/kg to 43.8 mg/kg. The majority of the lead concentrations in surface soil on and around the site fall within this background range. The maximum concentration of lead detected in surface soil samples (820 mg/kg) was detected in a sample collected from a vegetable garden. This concentration is similar to the concentration detected in a subsurface soil sample collected from the same location (774 mg/kg), 2 to 4 feet below ground surface. The presence of lead in these soil samples is believed to be directly related to the fill materials observed at this location. Because there are so few soil samples that contain lead at concentrations that substantially exceed the concentrations detected in background samples, it appears that the isolated high concentrations of lead that were found may be due to the presence of fill material.

Table 4-1

FIVE EPA WEIGHT-OF-EVIDENCE CATEGORIES FOR CHEMICAL CARCINOGENICITY

Group	Description
A	Human Carcinogen: sufficient evidence from epidemiological studies to support a causal association between exposure and cancer
B	Probable Human Carcinogen:
B1	• At least limited evidence of carcinogenicity to humans from epidemiological studies
B2	• A combination of sufficient evidence of carcinogenicity in animals and inadequate evidence of carcinogenicity in humans
C	Possible Human Carcinogen: limited evidence of carcinogenicity in animals in the absence of human data
D	Not Classified: inadequate evidence of carcinogenicity in animals
E	No Evidence of Carcinogenicity for Humans: no evidence of carcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies

Source: EPA 1986.

Table 4-2

TOXICITY VALUES FOR POTENTIAL CARCINOGENIC EFFECTS

Chemical	Route	Slope Factor (SF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type of Cancer	SF Basis/SF Source
Acetone	Oral	ND	D	NA	NA/IRIS and HEAST
	Inhalation	ND	D	NA	NA/IRIS and HEAST
Barium	Oral	ND	--	NA	NA/IRIS and HEAST
	Inhalation	ND	--	NA	NA/IRIS and HEAST
Benzo(a)anthracene	Oral	0.73	B2	NA	NA/extrapolated from B(a)P
	Inhalation	0.61	B2	NA	NA/extrapolated from B(a)P
Benzo(a)pyrene	Oral	7.3	B2	Stomach	Diet/IRIS
	Inhalation	6.1	B2	Respiratory Tract	Inhalation/HEAST (1991)
Benzo(b)fluoranthene	Oral	0.73	B2	NA	NA/extrapolated from B(a)P
	Inhalation	0.61	B2	NA	NA/extrapolated from B(a)P
Benzo(k)fluoranthene	Oral	0.073	B2	NA	NA/extrapolated from B(a)P
	Inhalation	0.061	B2	NA	NA/extrapolated from B(a)P
Cadmium	Oral	ND	--	NA	NA/IRIS
	Inhalation	6.3	B1	Lung, trachea, bronchus	Inhalation/IRIS
Chromium (III)	Oral	ND	NA	NA	NA/IRIS and HEAST
	Inhalation	ND	NA	NA	NA/IRIS and HEAST
Chrysene	Oral	0.0073	B2	NA	NA/extrapolated from B(a)P
	Inhalation	0.0061	B2	NA	NA/extrapolated from B(a)P
Cobalt	Oral	ND	--	NA	NA/IRIS and HEAST
	Inhalation	ND	--	NA	NA/IRIS and HEAST
Copper	Oral	ND	D	NA	NA/IRIS and HEAST
	Inhalation	ND	D	NA	NA/IRIS and HEAST

Key at end of table.

Table 4-2

TOXICITY VALUES FOR POTENTIAL CARCINOGENIC EFFECTS

Chemical	Route	Slope Factor (SF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type of Cancer	SF Basis/SF Source
Dibenzo(a,h)anthracene	Oral Inhalation	7.3 6.1	B2 B2	NA NA	NA/extrapolated from B(a)P NA/extrapolated from B(a)P
1,1-Dichloroethane	Oral Inhalation	ND ND	C C	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
1,2-Dichloroethane	Oral Inhalation	9.1E-02 9.1E-02	B2 B2	Circulatory system Circulatory system	Gavage/IRIS Gavage/IRIS
Endrin	Oral Inhalation	ND ND	D D	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Ethylbenzene	Oral Inhalation	ND ND	D D	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Heptachlor	Oral Inhalation	4.5 4.5	B2 B2	Liver Liver	Diet/IRIS Diet/HEAST
Heptachlor epoxide	Oral Inhalation	9.1 9.1	B2 B2	Liver Liver	Diet/IRIS Diet/IRIS
Indeno(1,2,3-cd)pyrene	Oral Inhalation	0.73 0.61	B2 B2	NA NA	NA/extrapolated from B(a)P NA/extrapolated from B(a)P
Lead	Oral Inhalation	ND ND	B2 B2	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Lindane	Oral Inhalation	ND ND	— —	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Manganese	Oral Inhalation	ND ND	D D	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Mercury (inorganic)	Oral Inhalation	ND ND	D D	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST

Key at end of table.

Table 4-2

TOXICITY VALUES FOR POTENTIAL CARCINOGENIC EFFECTS

Chemical	Route	Slope Factor (SF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type of Cancer	SF Basis/SF Source
Methylene Chloride	Oral Inhalation	7.5E-03 1.6E-03	B2 B2	Liver Liver	Inhalation/IRIS Inhalation/IRIS
2-Methylphenol	Oral Inhalation	ND ND	C C	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
4-Methylphenol (p-Cresol)	Oral Inhalation	ND ND	C C	NA NA	NA/IRIS NA/IRIS
Naphthalene	Oral Inhalation	ND ND	D D	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Nickel	Oral Inhalation	ND 8.4E-01	– A	NA Lung	NA/IRIS Occupational/IRIS
N-Nitrosodiphenylamine	Oral Inhalation	4.9E-03 ND	B2 B2	Bladder NA	Drinking water/IRIS NA/IRIS and HEAST
Phenol	Oral Inhalation	ND ND	— —	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Polychlorinated biphenyls (PCBs)	Oral Inhalation	7.7 7.7	B2 B2	Liver NA	Diet/IRIS NA/extrapolated from oral
Toluene	Oral Inhalation	ND ND	D D	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST
Trichloroethene[a]	Oral Inhalation	1.1E-02 6.0E-03	B2 B2	Liver Lung	Gavage/IRIS Inhalation/ECAO SCHRTSC
1,1,1-Trichloroethane	Oral Inhalation	ND ND	D D	NA NA	NA/IRIS and HEAST NA/IRIS and HEAST

Key at end of table.

Table 4-2

TOXICITY VALUES FOR POTENTIAL CARCINOGENIC EFFECTS

Chemical	Route	Slope Factor (SF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type of Cancer	SF Basis/SF Source
Xylene(s)	Oral	ND	D	NA	NA/IRIS and HEAST
	Inhalation	ND	D	NA	NA/IRIS and HEAST
Zinc	Oral	ND	D	NA	NA/IRIS and HEAST
	Inhalation	ND	D	NA	NA/IRIS and HEAST

[a] Toxicity values currently under review.

Key:

HEAST = Health Effects Assessment Summary Tables.
IRIS = Integrated Risk Information System.
NA = Not applicable.
ND = Not determined.
ECAO SHRTSC = Environmental Criteria and Assessment Office Superfund Health Risk Technical Support Center.

Source: Ecology and Environment, Inc. 1994.

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Acetone	Oral	Chronic	1E-01	Low	Increased liver and kidney weight - nephrotoxicity	Gavage/IRIS	UF = 1,000 MF = 1
		Subchronic	1E+00	NS	Increased liver and kidney weight - nephrotoxicity	Gavage/HEAST	UF = 100
	Inhalation	Chronic	1E-01	NS	NA	Extrapolated from oral	
		Subchronic	1E+00	NS	NA	Extrapolated from oral	
Barium	Oral	Chronic	7E-02	Medium	Increased blood pressure	Drinking water/IRIS	UF = 3 MF = 1
		Subchronic	7E-02	NS	Increased blood pressure	Drinking water/HEAST	UF = 3
	Inhalation	Chronic	1E-04	NS	Fetotoxicity	Inhalation/HEAST	UF = 1,000
		Subchronic	1E-03	NS	Fetotoxicity	Inhalation/HEAST	UF = 100
Benzo(a) anthracene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Benzo(a)pyrene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
Benzo(b) fluoranthene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
Benzo(k) fluoranthene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Cadmium	Oral	Chronic	5E-04	High	Significant proteinuria	Drinking water/IRIS	UF =10 MF = 1
		Subchronic	5E-04	NS	Significant proteinuria	Extrapolated from chronic	
	Inhalation	Chronic	5E-04	NS	NA	Extrapolated from oral	
		Subchronic	5E-04	NS	NA	Extrapolated from oral	
Chromium (III)	Oral	Chronic	1E+00	Low	None observed	Diet/IRIS	UF = 100 MF = 10
		Subchronic	1E+01	NS	None observed	Diet/HEAST	UF = 1,000
	Inhalation	Chronic	6E-07	NS	Nasal musoca atrophy	Inhalation/HEAST	UF = 300
		Subchronic	1.1E-06	Low	Nasal mucosa atrophy	Inhalation/ECAO SHRTSC	UF = 100
Chrysene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Cobalt	Oral	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Copper	Oral	Chronic	3.7E-02	NS	Local GI Irritation	Derived from drinking water standard/ HEAST	
		Subchronic	3.7E-02	NS	Local GI Irritation	Derived from drinking water standard/ HEAST	
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Dibenzo(a,h) anthracene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
1,1-Dichloroethane	Oral	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	1E-01	NS	None observed	Inhalation/HEAST	UF = 1,000
		Subchronic	1E+00	NS	None observed	Inhalation/HEAST	UF = 100
1,2-Dichloroethane	Oral	Chronic	ND	NS	Increased mortality	NA/IRIS and HEAST	
		Subchronic	ND	NS	Increased mortality	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
Endrin	Oral	Chronic	3E-04	Medium	Liver lesions, convulsions	Diet/HEAST	UF = 100 MF = 1
		Subchronic	3E-04	NS	Liver lesions, convulsions	Diet/HEAST	UF = 100

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Ethylbenzene	Oral	Chronic	1E-01	Low	Liver and kidney lesions	Gavage/IRIS	UF = 1,000 MF = 1
		Subchronic	1E-01	Low	Liver and kidney lesions	Inhalation/ECAO SHRTSC	UF = 1,000 MF = 1
	Inhalation	Chronic	2.9E-01	Low	Developmental toxicity	Inhalation/IRIS	UF = 300 MF = 1
		Subchronic	2.9E-01	NS	Developmental toxicity	Extrapolated from chronic	
Heptachlor	Oral	Chronic	5E-04	Low	Increased liver weight	Diet/IRIS	UF = 300 MF = 1
		Subchronic	5E-04	NS	Increased liver weight	Diet/HEAST	UF = 300
	Inhalation	Chronic	5E-04	NS	NA	Extrapolated from oral	
		Subchronic	5E-04	NS	NA	Extrapolated from oral	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Heptachlor epoxide	Oral	Chronic	1.3E-05	Low	Liver, increased weight, males only	Diet/IRIS	UF = 1,000 MF = 1
		Subchronic	1.3E-05	NS	Liver, increased weight	Diet/HEAST	UF = 1,000
	Inhalation	Chronic	ND	NA	NA	NA	
		Subchronic	ND	NA	NA	NA	
Indeno(1,2,3-cd)pyrene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
Lead	Oral	Chronic	ND	NS	Neurological effects	NA/IRIS and HEAST	
		Subchronic	ND	NS	Neurological effects	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	Neurological effects	NA/IRIS and HEAST	
		Subchronic	ND	NS	Neurological effects	NA/IRIS and HEAST	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Lindane	Oral	Chronic	3E-04	Medium	Liver and kidney toxicity	Diet/IRIS	UF = 1,000 MF = 1
		Subchronic	3E-03	NS	Liver and kidney toxicity	Diet/HEAST	UF = 100
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Manganese	Oral	Chronic	5E-03	Varied	CNS effects	Drinking water/IRIS	UF = 1 MF = 1
		Subchronic	5E-03	NS	CNS effects	Drinking water/HEAST	UF = 1
	Inhalation	Chronic	1E-04	Medium	Respiratory symptoms and psychomotor disturbances	Occupational/IRIS	UF = 300 MF = 3
		Subchronic	1E-04	NS	Respiratory symptoms, psychomotor disturbances	Intermittent inhalation/HEAST	UF = 900
Mercury (inorganic)	Oral	Chronic	3E-04	NS	Kidney effects	Oral/HEAST	UF = 1,000
		Subchronic	3E-04	NS	Kidney effects	Oral/HEAST	UF = 1,000

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
	Inhalation	Chronic	9E-05	NS	Neurotoxicity	Occupational/ HEAST	UF = 30
		Subchronic	9E-05	NS	Neurotoxicity	Occupational/ HEAST	UF = 30
2-Methylphenol	Oral	Chronic	5E-02	Medium	Decreased body weight, neurotoxicity	Oral/IRIS	UF = 1,000 MF = 1
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
4-Methylphenol (p-Cresol)	Oral	Chronic	5E-03	NS	Maternal death, respiratory distress	Gavage/IRIS	UF = 1,000 MF = 1
		Subchronic	5E-02	NS	Maternal death, respiratory distress	Gavage/HEAST	UF = 100
	Inhalation	Chronic	5E-03	NS	NA	Extrapolated from oral	
		Subchronic	5E-02	NS	NA	Extrapolated from oral	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Methylene chloride	Oral	Chronic	6E-02	Medium	Liver toxicity	Drinking water/IRIS	UF = 100 MF = 1
		Subchronic	6E-02	NS	Liver toxicity	Drinking water/HEAST	UF = 100
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Naphthalene	Oral	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Nickel	Oral	Chronic	2E-02	Medium	Decreased body and organ weight	Diet/IRIS	UF = 300 MF = 1
		Subchronic	2E-02	NS	Decreased body and organ weight	Diet/HEAST	UF = 300
	Inhalation	Chronic	2E-02	NS	NA	Extrapolated from oral	
		Subchronic	2E-02	NS	NA	Extrapolated from oral	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
N-Nitrosodiphenylamine	Oral	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Phenol	Oral	Chronic	6E-01	NS	Reduced fetal body weights	Oral/IRIS	UF = 100 MF = 1
		Subchronic	6E-01	NS	Reduced fetal body weights	Gavage/HEAST	UF = 100
	Inhalation	Chronic	ND	NA	NA	NA/IRIS and HEAST	
		Subchronic	ND	NA	NA	NA/IRIS and HEAST	
Polychlorinated biphenyls	Oral	Chronic	ND	NS	Immunological effects	NA/IRIS and HEAST	
		Subchronic	ND	NS	Immunological effects	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Toluene	Oral	Chronic	2E-01	Medium	Changes in liver and kidney weights	Gavage/IRIS	UF = 1,000 MF = 1
		Subchronic	2E+00	NS	Changes in liver and kidney weights	Gavage/HEAST	UF = 100
	Inhalation	Chronic	1.2E-01	NS	CNS effects, eyes and nose irritation	Inhalation/IRIS	UF = 300 MF = 1
		Subchronic	6E-01	Medium	CNS effects, nose and eyes irritation	Inhalation/HEAST	UF = 100
1,1,1-Trichloroethane	Oral	Chronic	9E-02	Medium	Hepatotoxicity	Oral/IRIS (withdrawn)	UF = 1,000 MF = 1
		Subchronic	9E-01	NS	Hepatotoxicity	Oral/HEAST (withdrawn)	UF = 100
	Inhalation	Chronic	3E-01	NS	Reduced body weight gain	Inhalation ECAO SHRTSC	UF = 1,000
		Subchronic	3E-01	NS	NA	Extrapolated from chronic	
Trichloroethene	Oral	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	

Key at end of table.

Table 4-3

TOXICITY VALUES FOR POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	Reference Dose (RfD)				Critical Effects	RfD Basis/ Source	Uncertainty (UF) and Modifying (MF) Factors
	Route	Type	Value mg/kg-day	Confidence Level			
Xylene(s)	Oral	Chronic	2E+00	Medium	Hyperactivity, decreased body weight, increased mortality	Gavage/IRIS	UF = 100 MF = 1
		Subchronic	2E+00	NS	NA	Extrapolated from chronic	
	Inhalation	Chronic	ND	NS	NA	NA/IRIS and HEAST	
		Subchronic	ND	NS	NA	NA/IRIS and HEAST	
Zinc	Oral	Chronic	3E-01	Medium	Decrease in ESOD concentration	Diet/IRIS	UF = 3 MF = 1
		Subchronic	3E-01	NS	Decrease in ESOD concentration	Extrapolated from chronic	
	Inhalation	Chronic	3E-01	NS	NA	Extrapolated from oral	
		Subchronic	3E-01	NS	NA	Extrapolated from oral	

Key at end of table.

Table 4-3 (Cont.)

Key:

DI = Data inadequate for quantitative risk assessment.
HEAST = Health Effects Assessment Summary Tables.
IRIS = Integrated Risk Information System.
NA = Not applicable.
ND = Not determined.
NS = Not specified.
ECAO SHRTSC = Environmental Criteria and Assessment Office Superfund Health Risk Technical Support Center.

Source: Ecology and Environment, Inc. 1994.

5. RISK CHARACTERIZATION

5.1 INTRODUCTION

This section combines the information developed in the exposure and toxicity assessment sections to estimate the potential risks to human health posed by the Dearcop Farm site contaminants. The risk estimation process is explained in this section.

Risks due to carcinogenic and noncarcinogenic contaminants are assessed differently, as discussed in Sections 4.3.2 and 4.3.3. Briefly, carcinogenic risks are assessed by multiplying the estimated lifetime average daily intake (LADI) of a carcinogen by its estimated SF to obtain the estimated risk, expressed as the probability of that exposure resulting in an excess incidence of cancer (i.e., more cancers than would normally be expected in that population). The potential for adverse effects resulting from exposure to noncarcinogens is assessed by comparing the chronic daily intake (CDI) or subchronic daily intake (SDI) of a substance to its chronic or subchronic RfD. This comparison is performed by calculating the ratio of the estimated CDI or SDI to the corresponding RfD, which is called a hazard quotient. If the hazard quotient is less than 1, no adverse effects would be expected; however, if it is greater than 1, adverse effects could be possible.

The excess cancer risk or the hazard quotient for exposure to each chemical by each route of exposure, exposure pathway, category of receptor (i.e., adult or child), and exposure case are initially estimated separately. The separate cancer risk estimates are then summed across chemicals and across all exposure routes and pathways applicable to the same population to obtain the total excess cancer risk for that population. Hazard quotients for noncarcinogens are summed across chemicals that produce the same type of adverse effects (such as liver damage) but are kept separate if their effects are different. Hazard quotients for

subchronic and chronic effects are separately summed across all chemicals, exposure routes, and pathways applicable to the same population to obtain hazard indices for that population.

Section 5.2 presents tables that contain the detailed risk estimates just described. Section 5.3 summarizes the risk estimation results and identifies the chemicals, pathways, and receptors that account for the most significant risks at the Dearcop Farm site. Uncertainties in the risk estimation process are discussed in Section 6.3.

5.2 RISK ESTIMATES

Because of the number of exposure pathways, receptors, exposure cases, and chemicals that were evaluated, several tables are necessary to present the results. Tables containing estimates of exposures and associated risks for the scenarios described earlier in Section 3.2 are contained in Appendix B.

A directory has been included in the appendix to assist the reader in locating the exposure and risk estimates for specific exposure pathways. The risk estimates are summarized and discussed in Section 5.3. The toxicity estimates (SFs and RfDs) used in calculating the risk estimates, along with key information qualifying the toxicity estimates, are presented in Tables 4-2 and 4-3.

5.3 SUMMARY DISCUSSION OF RISK CHARACTERIZATION

5.3.1 Characterization of Contamination Present at the Site

The remedial investigation was designed to characterize the nature and extent of contamination originating at the Dearcop Farm site. The possible source areas were identified based on a review of past activities at the site and previous sampling activities. Potential source areas and migrating pathways were then investigated using various field techniques and by collection and analysis of samples. In this way, the nature of the contamination was characterized and its extent defined.

Given the information available about the site, it seems unlikely that any significant source areas or migration pathways were overlooked. Because samples were collected from a variety of media encompassing the likely source areas and migration pathways, it is unlikely that any significant contaminants have been missed.

5.3.2 Magnitude and Source of Risks Posed by Site Contamination

The magnitude of the potential excess cancer risks posed by the site contaminants are presented in Appendix B and are summarized in Tables 5-1 and 5-2. The hazard indices for the potential noncarcinogenic effects provided in the Appendix B tables are summarized in Tables 5-3 and 5-4. These tables provide risk and hazard index estimates corresponding to the reasonable maximum and average exposure cases. Risk management decisions are usually based on risk estimates for the RME case to ensure that the decisions are adequately protective of public health.

As shown in the tables, trespasser exposure to contaminants at the Dearcop Farm site under the RME case appears to pose a potential increased risk of developing cancer. Nearby residents also may experience an increased potential of developing cancer.

Under existing site conditions, the estimated excess potential cancer risks under the RME case for adolescent trespassers is 3.4×10^{-6}. Total estimated current cancer risks for the average case are lower by approximately an order of magnitude (2.1×10^{-7}). Results of the site trespasser scenario do indicate that if the landfill was converted to residential use, residents potentially could experience significant risks from exposure to site-related contamination through soil ingestion, dermal contact with contaminated soil, and inhalation of soil particulates.

Site contaminants, under the RME case, do not appear to pose an increased risk of adverse noncarcinogenic health effects to site trespassers. The total hazard index for all potential pathways through which trespassers could be exposed to site-related contamination is 0.6, which is approximately half of the threshold hazard index. Under current conditions it does not appear that nearby residents would experience potential adverse noncarcinogenic health effects due to the presence of fill material in residential yards that reportedly came from the landfill.

As discussed in previous sections, site-derived waste material reportedly was placed in residential yards and used as fill. The excess cancer risk and the potential for significant adverse health effects associated with this fill material were assessed for nearby residents. Data from composite soil samples collected from high-use areas (under swing sets, picnic tables) of residential yards were used to estimate these potential risks and adverse health effects. PAH concentrations were generally higher in residential soils than in on-site soils. Because PAHs are a common byproduct of combustion, there may be an off-site source of

these PAHs. In addition, concentrations of PAHs in the residential soils were generally within typical urban soil concentrations (600-3000 μg/kg range) as given by Menzie *et al.*, 1992. The maximum estimated potential cancer risk to residents from soil ingestion using reasonable maximum exposure assumptions was 1.2×10^{-4}, which is greater than the EPA's allowable risk range. These excess estimated cancer risks are entirely due to PAHs.

Hazard indices for nearby residents were estimated for dermal exposure and soil ingestion. The maximum hazard indices due to dermal contact and soil ingestion under the RME case are 0.0072 and 0.10, respectively, which are below EPA's threshold hazard index of 1.0. Hazard indices also were calculated for exposure to metals in tomatoes and broccoli grown in yards containing fill reportedly from the landfill. Exposures were estimated using typical consumption rates for these vegetables derived from government dietary surveys (EPA 1989a). Because no formal screening criteria exist for vegetables, values reported in two documents (Kabata-Pendias and Pendias 1992; Adriano 1986) for typical concentrations of metals in these vegetables were used as benchmarks to determine whether metals were present in concentrations exceeding typical background levels. Based on a comparison between the metals concentrations detected in the vegetable samples and the typical concentrations reported in the above-referenced documents, lead, nickel, selenium, and zinc are elevated in the broccoli sample and chromium is elevated in the tomato sample. Due to the uncertainty surrounding these typical concentrations, all of the metals for which toxicity indices are available were carried through the quantitative risk assessment. These calculations yielded hazard indices greater than 1.0 under the RME case for consumption of broccoli containing cyanide (1.03) and manganese (2.68).

5.3.3 Nature of Potential Adverse Health Effects

Under current site conditions, for site residents, ingestion of PAH-contaminated soils is responsible for the majority of the increased potential cancer risk. PAHs can cause cancer at the point of exposure. Animals exposed to high levels of benzo(a)pyrene in air have developed lung tumors, and when exposed via ingestion, they develop stomach tumors. There also is evidence that dermal contact with benzo(a)pyrene causes skin tumors.

5.3.4 Level of Confidence/Uncertainty in the Risk Estimates

These matters are discussed fully in Section 6 of this report; briefly, the level of confidence in the exposure estimates is moderate. The level of confidence in the toxicity estimates varies from chemical to chemical as shown in Tables 4-2 and 4-3.

Overall, the level of confidence in the risk estimates is also moderate. However, as discussed in Section 6.3, the nature of the risk assessment process strongly favors overestimation of the true risks.

5.3.5 Characteristics of the Potentially Exposed Populations

Residents living near the landfill are the individuals most likely to be exposed to site-related contaminants both in residential yards and on site. The nearby residential area is not known to be enriched in sensitive subpopulations such as young children or sick or elderly individuals. It is likely that trespassers reside in the nearby residential area; therefore, certain individuals may be subject to exposure both in the residential area and the landfill area. Because it is difficult to determine which exposures would occur under both scenarios, the risks and hazard quotients for these receptors were not summed.

Table 5-1

SUMMARY OF ESTIMATED EXCESS CANCER RISKS - RME CASE

Exposure Scenario	Exposure Media	Receptors		Risk Contributions by Exposure Route	Risk Contribution by Chemical
		Adolescent	Resident		
Site Trespasser	Soil	2.3×10^{-6}	—	Dermal contact - 66% Incidental ingestion - 34%	PCBs - 92% Benzo(a)pyrene - 3% Dibenz(a,h)anthracene - 3%
	Air	1.1×10^{-6}	—	Inhalation of soil particulates - 100%	Nickel - 64% Cadmium - 25% PCBs - 9%
Nearby Resident	Soil	—	$1{:}2 \times 10^{-4}$	Incidental ingestion - 100%	Benzo(a)pyrene - 53% Dibenz(a,h)anthracene - 32% Benzo(a)anthracene - 7% Benzo(b)fluoranthene - 4% Indeno(1,2,3-cd)pyrene - 3%
	Vegetables	—	—	—	—

Table 5-2

SUMMARY OF ESTIMATED EXCESS CANCER RISKS - AVERAGE CASE

Exposure Scenario	Exposure Media	Receptors		Risk Contributions by Exposure Route	Risk Contribution by Chemical
		Adolescent	Resident		
Site Trespasser	Soil	4.8×10^{-8}	—	Incidental ingestion - 71% Dermal contact - 29%	PCBs - 61% Benzo(a)pyrene - 18%
	Air	1.6×10^{-7}	—	Inhalation of soil particulates - 100%	Nickel - 64% Cadmium - 29% PCBs - 4% Benzo(a)pyrene - 1% Dibenz(a,h)anthracene - 1%
Nearby Resident	Soil	—	1.9×10^{-5}	Incidental ingestion - 100%	Benzo(a)pyrene - 53% Dibenz(a,h)anthracene - 31% Benzo(a)anthracene - 7% Benzo(b)fluoranthene - 4% Indeno(1,2,3-cd)pyrene - 4%
	Vegetables	—	—	—	—

Table 5-3

SUMMARY OF ESTIMATED HAZARD INDICES - RME CASE

Exposure Scenario	Exposure Media	Receptors			Significant Hazard Index Contributions by Exposure Route	Significant Hazard Index Contributions by Chemical
		Adolescent	Adult			
Site Trespasser	Soil	0.02	—		Incidental ingestion - 99% Dermal contact - 1%	Copper - 48% Cadmium - 26% Barium - 25%
	Air	0.58	—		Inhalation of soil particulates - 100%	Barium - 99%
Nearby Resident	Soil	—	0.42		Incidental ingestion - 98% Dermal contact - 2%	Copper - 48% Zinc - 16% Mercury - 16% Cadmium - 13% Barium - 4%
			Adult	Child		
	Vegetables	—	1.3	5.6	Ingestion of homegrown vegetables - 100%	Manganese - 56% Cyanide - 18% Copper - 11% Zinc - 8% Nickel - 4%

Table 5-4

SUMMARY OF ESTIMATED HAZARD INDICES - AVERAGE CASE

Exposure Scenario	Exposure Media	Receptors			Significant Hazard Index Contributions by Exposure Route	Significant Hazard Index Contributions by Chemical
		Adolescent	Adult			
Site Trespasser	Soil	7.3×10^{-4}	—		Incidental ingestion - 95% Dermal contact - 5%	Cadmium - 43% Copper - 27% Nickel - 13% Barium - 10% Zinc - 3% Mercury - 1%
	Air	0.024	—		Inhalation of soil particulates - 100%	Barium - 99%
Nearby Resident	Soil	—	0.21		Incidental ingestion - 99% Dermal contact - 1%	Copper - 49% Mercury - 16% Zinc - 16% Cadmium - 12% Barium - 4%
	Vegetables	—	Adult	Child	Ingestion of homegrown vegetables - 100%	Manganese - 51% Cyanide - 18% Copper - 11% Zinc - 8% Nickel - 4%
			0.079	0.4		

6. UNCERTAINTIES IN THE RISK ASSESSMENT

6.1 UNCERTAINTIES RELATED TO THE EXPOSURE ASSESSMENT

A number of factors will cause the exposure levels estimated in the exposure assessment to differ from the exposures that potential receptor populations might actually experience. This section will identify these factors, discuss the potential effects of the factors on the exposure estimates, and, where possible and appropriate, estimate the degree of confidence that should be placed in the various assumptions and parameter estimates that have gone into the exposure estimates.

6.1.1 Environmental Sampling

Samples collected during the RI were intended to characterize the nature and extent of contamination at the site. Accordingly, most were collected from locations selected in a biased or directed manner to accomplish this goal. Samples collected in this manner provide considerable information about the site but are not statistically representative of the contamination that may be present in a specific area or on the site as a whole. To gather statistically representative data, the sampling locations need to be selected in a random or systematic fashion, usually using a grid system. This was done only for some Phase I soil samples collected from residential yards adjacent to the landfill. Other Phase I RI samples were taken in a directed fashion. Sampling locations selected in a directed fashion tend to be concentrated in areas having higher levels of contamination; therefore, data from sampling locations selected in this way tend to overestimate the average concentrations present in a representative exposure area.

Development of the source concentrations used to estimate exposures is discussed in Section 3.3.

6.1.2 Analytical Result Limitations

Two aspects of the analytical data marginally reduce the level of confidence in the estimates of contaminant concentrations in environmental media. One aspect is the inclusion of estimated results (J flags) that may not have the same precision and accuracy as data meeting all of the standard QA criteria. This is a minor concern.

The second aspect is the use of analytical detection limits that could allow potentially hazardous concentrations of some contaminants to go undetected. This source of uncertainty reduces the level of confidence that can be placed in the upper limit of the risk associated with environmental media in which these contaminants could be present at concentrations less than but close to the detection limit.

6.1.3 Contaminant Migration Modeling

Uncertainties about the reliability of modeling predictions arise in two areas. The first is the validity and accuracy of the model itself, and the second is the selection of appropriate parameter values for use in the calculations. Two types of models were used in developing exposure estimates.

One type of modeling concerned the emission of soil particles from ground surface to the ambient air. The method described by EPA (1985) and used to estimate the particulate emission rate employs the same general methods for estimating emission rates as some of the more sophisticated models and is believed to provide a reasonable degree of accuracy (GRI 1988). Site-specific soil properties and published average meteorological data for the Dearcop Farm site area were used as inputs to this model; therefore, the results should be moderately to highly reliable.

The second type of modeling is air dispersion modeling, which was used to estimate ambient air concentrations downwind from the source areas. A simple box model was used for this purpose. The box model is a basic dilutional model that estimates air concentrations by diluting the particle flux from the ground by the volume of air passing over the source area. The key parameters, the size of the source area, and the wind speed are selected on a site-specific basis. The results of this model should be moderately reliable.

Taken together, the results for both types of modeling should be moderately to highly reliable.

6.1.4 Exposure Estimation Calculations

The primary uncertainty regarding the exposure calculations is that associated with the selection of appropriate parameter values. The values used and a brief rationale for their selection are given in Section 3.3.3, which describes exposure calculations for the various pathways. Individual parameter values were selected so that the overall pathway exposure estimates would approximate average and reasonable maximum exposures. Recent EPA exposure assessment guidance indicates that the exposure factors used in estimating the RME should be selected so that the RME case represents a plausible high-end exposure; one that would be expected to fall above the 90th percentile exposure for the population in question, but not above the 99.9th percentile or the maximum exposure that could reasonably be expected.

6.1.5 Exposure Assessment Uncertainty Summary

Overall, the exposure estimates obtained are probably moderately reliable. Several of the factors adding uncertainty to the estimates tend to result in overestimation of the exposure. These include:

- The directed nature of most of the sampling program;
- The use of the upper 95th percent confidence limits or the maximum observed value for the source concentrations; and
- The use of many 90th percentile values in the exposure estimation calculations.

One factor could lead to underestimation of the exposures:

- The use of sample quantitation limits that could result in missing low concentrations of some compounds that might pose significant risks.

6.2 UNCERTAINTIES RELATED TO THE TOXICITY ASSESSMENT

6.2.1 Introduction

To evaluate the meaning of any risk assessment, one must consider the uncertainties in the assumptions made, the impact of changing the magnitude of those assumptions on the risk estimates, and the relevance of the findings to real world exposures and risks. Because

of the number of assumptions, data points, and calculations, a degree of uncertainty is necessarily associated with the numerical toxicity values in any risk assessment.

6.2.2 Evaluation of Carcinogenic Toxicity Assessment Assumptions

The COPCs have been evaluated by the EPA using its weight-of-evidence carcinogenicity evaluation criteria and have been placed in Group A, human carcinogens, or Group B, probable human carcinogens, based on sufficient data in humans or sufficient data in animals and insufficient data in humans, respectively (EPA 1986).

Rodent bioassay and epidemiological studies, such as those performed for the COPCs, would require tens of thousands of animals or humans to determine whether a chemical is carcinogenic at low doses. Because the relationship between tumor location, time to appearance, and the proportion of animals with cancer determines the estimated carcinogenic SF, animal bioassay or human epidemiological data are not routinely sufficient for directly estimating SF at low doses. Therefore, by necessity, agencies such as the EPA use carcinogenic extrapolation models for estimating low-dose SFs. Based on prudent public policy, these agencies assume that there is no threshold dose below which carcinogenic risks will not occur. This is equivalent to the assumption that every dose above zero, no matter how low, carries with it a small but finite risk of cancer. They also assume that the dose-response relationship is linear at low doses. This is contrary to approaches used for other toxic effects, for which thresholds are assumed to exist.

The current model favored by the EPA and certain other federal regulatory agencies is the linearized multistage model. The agency then uses the statistically derived upper 95% confidence bounds, rather than a maximum likelihood value for the SF. The EPA has concluded, based on theoretical grounds consistent with human epidemiological and animal data, that cancer follows a series of discrete stages (i.e., initiation, promotion, and progression) that ultimately can result in the uncontrolled cell proliferation known as cancer. Consistent with this conclusion, the use of the linearized multistage model permits an estimation of the SF that is not likely to be exceeded if the real slope could be measured. However, compelling scientific arguments can be made for several other extrapolative models, which, if used, could result in significantly reduced values for SFs, approximately tens of millions of times lower than those estimated using the linearized multistage model. The one-hit model, used to estimate risks due to exposures above the linear range of the multistage model, is one such

model. Thus, the current EPA SFs calculated in this fashion represent upper-bound values based on animal data that should not be interpreted as necessarily equivalent to actual human cancer potencies. It is this conservative value, nevertheless, that is used in this risk assessment on policy grounds for the protection of public health.

6.2.3 Evaluation of Noncarcinogenic Toxicity Assessment Assumptions

Key assumptions used in assessing the likelihood of noncarcinogenic effects are that threshold doses exist below which various noncarcinogenic effects do not occur and that the occurrence or absence of noncarcinogenic effects can be extrapolated between species and occasionally between routes of exposure and over varying exposure durations. The threshold assumption appears to be sound for most noncarcinogens based on reasonably good fits of experimental data to the usual dose response curves. One possible exception to this is lead, which may not have a threshold base for its noncarcinogenic effects (ATSDR 1991).

The other assumptions generally appear to be true to varying degrees. The effects observed in one species or by one route of exposure may not occur in another species or by another route, or they may occur at a higher or lower dose due to differences in the biokinetics of a compound in different species or when exposure occurs by different routes. The uncertainty in these assumptions is taken into account in the development of RfDs through the use of safety or uncertainty factors. These factors reflect uncertainty associated with species-to-species extrapolation and include safety factors to protect sensitive individuals. In addition to uncertainty factors, a modifying factor is applied to reflect a qualitative professional assessment of additional uncertainties in the critical study and in the entire data base for the chemical not explicitly addressed by the preceding uncertainty factors. The modifying factor ranges from greater than 0 to 10 with a default value of 1 (EPA 1989b).

The uncertainty factors and modifying factors used by the EPA are conservative (health protective) in nature in that they tend to overestimate the uncertainties so that the RfDs obtained are unlikely to be too high. Use of the resulting RfDs tends to overestimate the potential for noncarcinogenic effects occurring at a given exposure level. Section 4.3.3 discusses uncertainty factors used to derive the RfDs for COPCs at the Dearcop Farm site.

6.2.4 Route-to-Route Extrapolation of Reference Doses and Slope Factors

Route-to-route extrapolation of RfDs and SFs adds an additional source of uncertainty to the risk estimates obtained through their use. Such extrapolation may result in either under- or overestimation of the true risks for the extrapolated route. Although this practice adds uncertainty to the risk assessment process, it appears to be preferable to omitting consideration of exposure to a chemical by a route for which no RfD or SF is available from the quantitative risk assessment, which would lead to underestimation of the overall risks posed by the chemical.

6.2.5 Summary of Toxicity Assessment Uncertainties

The basic uncertainties underlying the assessment of the toxicity of a chemical include:

- Uncertainties arising from the design, execution, or relevance of the scientific studies that form the basis of the assessment; and
- Uncertainties involved in extrapolating from the underlying scientific studies to the exposure situation being evaluated, including variable responses to chemical exposures within human and animal populations, between species, and between routes of exposure.

These basic uncertainties could result in a toxicity estimate, based directly on the underlying studies, that either under- or overestimates the true toxicity of a chemical in the circumstances of interest. Additional uncertainty results from the absence of EPA-approved toxicological indices for some chemicals, which made it necessary to use surrogate values (which could overestimate or underestimate risk) or to evaluate contaminants qualitatively rather than quantitatively (which will result in a slightly lower estimate of the total risks posed by the site).

The toxicity assessment process compensates for these basic uncertainties through the use of safety factors (uncertainty factors) and modifying factors when assessing noncarcinogens, and the use of the upper 95th percentile confidence limit from the linearized multistage model for the SF when assessing carcinogens. The use of the safety factors and the upper 95th percentile confidence limit in deriving the RfDs and SFs ensures that the toxicity values used in the risk estimation process are very unlikely to underestimate the true toxicity of a chemical.

6.3 RISK CHARACTERIZATION UNCERTAINTIES

The risk characterization combines and integrates the information developed in the exposure and toxicity assessments; therefore, uncertainties associated with these assessments also affect the degree of confidence that can be placed in the risk characterization results. Sections 6.1 and 6.2 provide full discussions of the factors causing uncertainty in the exposure and toxicity assessments, respectively.

Several additional factors need to be considered when discussing uncertainties associated with the overall risk characterization. These include the cumulative effects of using conservative assumptions throughout the process and the likelihood of the exposures postulated and estimated in the exposure assessment actually occurring.

The cumulative effects of using conservative assumptions throughout the risk estimation process is that the resulting estimates could substantially overstate the true risks. *The Risk Assessment Guidance for Superfund* manual (EPA 1989b) recommends that individual parameter values be selected so that the overall estimate of exposure represents an RME. In many cases, the statistical distribution of a parameter is unknown and the risk assessor is left to use best professional judgment to select a value that is sufficiently conservative to avoid underestimating the true risk, yet not so conservative that the resulting risk estimate turns out to be unreasonably high. When in doubt, the risk assessor will usually elect to err in favor of protecting human health and select a value that results in overestimating the true risk. The nature of the risk estimation process itself virtually ensures that the true risks are much more likely to be overestimated than underestimated.

7. SUMMARY AND CONCLUSIONS

Results of this quantitative risk assessment performed using data from samples collected from the Dearcop Farm site and the nearby residential area indicate that exposure to site-related contamination both on site and in the nearby residential area poses an increased excess potential cancer risk for both adolescent trespassers and nearby residents. The major factors driving the site risks are the presence of PAHs in residential surface soils and the presence of PCBs in on-site soils. The source of the PAH contamination in the residential soils is unknown. Under existing conditions, it does not appear that nearby residents or site trespassers would experience an increased potential risk of developing adverse noncarcinogenic health effects due to their exposure to site-related contamination, except for residential ingestion of contaminated vegetables, as discussed earlier.

Landfill Area

Metals (barium, cadmium, cobalt, copper, lead, mercury, nickel, and zinc), PAHs, and PCBs were detected in on-site surface soils at concentrations exceeding applicable criteria (i.e., NYSDEC-recommended soil cleanup goals, benchmark health risk values, and/or EPA Region III RBCs). Results of this risk assessment indicate that, under the RME case, the greatest excess potential cancer risk associated with exposure to contaminated surface soils on site is due to dermal contact with PCB-contaminated soil (1.5×10^{-6}), whereas inhalation of contaminated soil particulates leads to calculated excess potential cancer risk of 1.1×10^{-6}. Ingestion of contaminated on-site surface soil results in an estimated potential excess cancer risk of 7.8×10^{-7}. These results indicate that site trespassers may experience an increased potential of developing cancer due to their activities on site. It is important to note, however, that these estimated carcinogenic risks do not exceed the upper boundary of the EPA's

allowable risk range (10^{-6} to 10^{-4}). In addition, these risks were estimated using reasonable, but conservative assumptions that may have led to overestimation of potential carcinogenic risks.

Nearby Residential Area

Elevated concentrations of PAHs and metals (barium, cadmium, cobalt, copper, lead, mercury, nickel, and zinc) were detected in composite residential surface soil samples. As discussed previously, soil and debris reportedly were taken from the Dearcop Farm property and used as fill material in nearby residential yards. Although detected concentrations of PAHs in residential soils were greater than concentrations detected in on-site soils, concentrations of PAHs in the residential soils were generally within the typical urban soil concentration range (600 - 3,000 μg/kg) reported by Menzie, *et. al.*, 1992.

The greatest estimated excess potential cancer risk for nearby residents is associated with incidental ingestion of these PAH-contaminated soils. Under the RME case, the maximum estimated excess potential cancer risk associated with residential ingestion of contaminated soils is 1.2×10^{-4}. As with the trespasser scenario, these risks were estimated using reasonable, but conservative, assumptions that may result in estimates that could substantially overstate the true risks.

Where the cumulative carcinogenic site risk to an individual, based on the RME case for current land uses, is less than 10^{-4}, action generally is not warranted. A specific risk estimate of approximately 1×10^{-4} may be considered acceptable if justified based on site-specific conditions (Clay 1991).

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APPENDIX A

AIR MODELING

This appendix summarizes the models, assumptions, and input data used to estimate chemical concentrations in the air to which site trespassers may be exposed. These models comprise the air pathway analysis methods.

This appendix is divided into two sections. Section A.1 summarizes the estimation of air emissions of particulates due to wind erosion; and Section A.2 describes near-field air dispersion using the "box model."

A.1 AIR EMISSIONS OF PARTICULATES DUE TO WIND EROSION

The inhalable particulate (PM_{10}) emissions from the Dearcop Farm site due to wind erosion were estimated using the model for an "unlimited reservoir" of erodible soil as described in *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites* (EPA 1985). According to this model, the emission factor for wind erosion is determined as follows:

$$E_{10} = 0.036\,(1\text{-}V)\,\frac{\bar{u}^{3}}{u_{t7}} \cdot F(x)$$

Where:

E_{10} = Annual average emission rate of inhalable particulates (PM_{10}) per unit area of contaminated soil surface ($g/m^2 \cdot hr$)

V = Fraction of contaminated surface covered by continuous vegetation (dimensionless)

$\bar{u}$ = Mean annual wind speed (m/s)

$x = 0.886\,\frac{u_{t7}}{\bar{u}}$ (dimensionless ratio)

u_{t7} = Threshold wind speed at 7m above ground surface (m/s)

$F(x)$ = Function value from EPA 1985, p. 36 (dimensionless).

For a specific chemical contaminant in the erodible surface soil, the contaminant emission rate on wind-blown PM_{10} particulates is then calculated as:

$$R_{10} = \alpha \cdot E_{10} \cdot A \cdot \frac{1\ hr}{3{,}600\ s} \times \frac{10^3\ mg}{1\ g}$$

Where:

R_{10} = Annual average emission rate of contaminant on PM_{10} particulates generated by wind erosion over a total area A (mg/s)

α = Contaminant mass fraction in PM_{10} emissions, assumed to be the same as in bulk surface soil (mg/kg) or (g/g)

A = Contaminated soil area subject to wind erosion (m^2).

The site-specific values of the input parameters assumed in applying this model are presented in Table A-1.

A.2 NEAR-FIELD AIR DISPERSION: "BOX MODEL"

For scenarios where the receptor is at the source or very close (within 100 meters downwind) to the source, the near-field "box model" described in *Management of Manufactured Gas Plant Sites, Volume III, Risk Assessment* (Gas Research Institute 1988) was applied. This model is as follows:

$$C_a = f \cdot Q/(H_b \cdot W_b \cdot U_m)$$

Where:

C_a = Contaminant air concentration (mg/m^3)

Q = Contaminant source strength or emission rate from surface (mg/s)

f = Fraction of time wind blows in the sector from the source toward the receptor (dimensionless)

H_b = Height of box at downwind edge (m) [depends on downwind distance (X) of receptor]

X = Downwind distance of receptor from upwind edge of source area (m)

W_b = Crosswind width of box = crosswind dimension of contaminated source area (m)

U_m = Average wind speed throughout box (m/s) = $0.22\ U_{10}\ \ln(2.5\ H_b)$

where U_{10} = Wind speed at 10m elevation (m/s).

In applying the above model, the height (H_b) is determined by the downwind distance (X) of the receptor by using a table of precalculated values. This table is reproduced here as Table A-2. The source strength (Q) is assumed to be the annual average contaminant emission rate of PM_{10} particulates, calculated using an appropriate model as shown in Section A.1.

Table A-3 summarizes the site-specific input parameter values assumed in applying this model to estimate on-site air exposure concentrations.

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Gas Research Institute (GRI), 1988, *Management of Manufactured Gas Plant Sites, Volume III, Risk Assessment*, prepared by Atlantic Environmental Services, Inc., May 1988, Appendix B, Exposure Models.

Table A-1

SUMMARY OF SITE-SPECIFIC INPUTS USED IN THE MODEL FOR AIR EMISSIONS OF PARTICULATES DUE TO WIND EROSION

Symbol	Parameter	Units	Value	Source
"Unlimited Reservoir" Model				
V	Fraction of contaminated surface covered by continuous vegetation	—	0	Bare soil assumed for conservative estimate
$\bar{u}$	Mean annual wind speed	m/s	4.29	Approximate mean for site area from local climatological data (9.6 mph)
u_{t7}	Threshold wind speed at 7m	m/s	5.64	Calculated from the surface threshold speed of 32 cm/s as shown in EPA (1985)
F(x)	Function value from graph	—	1.4	EPA (1985), Fig. 4-3, p. 36
E_{10}	Annual average PM_{10} emission rate due to wind erosion	$g/m^2 \cdot hr$	2.2×10^{-2}	Calculated using the model equation with the above input parameter values
A	Contaminated soil area subject to wind erosion	m^2	1,858	100 feet x 200 feet (open, non-vegetated area)
α	Contaminant mass fraction in PM_{10} emissions	g/g	—	Contaminant-specific value: maximum observed concentration in surface soil samples from site

Compiled by: Ecology and Environment, Inc. 1994.

Table A-2

PLUME HEIGHTS USED IN NEAR-FIELD BOX MODEL

Length of Side of Box, x (m)	Box Height, H_b (m)
10	1.4
20	2.1
30	2.7
40	3.3
50	3.8
60	4.3
70	4.8
80	5.3
90	5.8
100	6.2

Source: GRI 1988, Exhibit 1.2.2-2, p. B-147.

Table A-3

SUMMARY OF SITE-SPECIFIC INPUTS USED IN THE BOX MODEL FOR NEAR-FIELD AIR DISPERSION

Symbol	Parameter	Units	Value	Source
On-Site Receptors: Trespassers				
Q	Contaminant source strength	mg/s	—	Contaminant-specific value. For particulates, the annual average emission rate of contaminant on PM_{10} due to wind erosion, calculated during the model in Section A.1.
f	Fraction of time the wind blows from the source area toward the receptor	—	0.25	On-site receptors assumed to be downwind of each source area 25% of the time.
x	Downwind distance to receptor from upwind edge of source area	m	60.96	For hot-spot areas, the dimensions of which are assumed to be approximately 100 by 200 feet. On-site receptors are assumed to be at the downwind edge of each source area.
H_b	Height of box	m	4.4	For 100- by 200-foot area. H_b is determined from Table A-2 using length corresponding to the downwind receptor distance x.
W_b	Crosswind width of box	m	30.48	For hot-spot areas, width is assumed to be 100 feet.
U_{10}	Wind speed at 10 m height	m/s	4.29	Annual mean value from local climatological data (9.6 mph).

Source: Ecology and Environment, Inc. 1994.

APPENDIX B

RISK TABLES

Table B-1

SUMMARY OF THE POTENTIAL EXPOSURE SCENARIOS INCLUDED IN THE QUANTITATIVE RISK ASSESSMENT

Receptor	Exposure Media	Exposure Routes	Age Group	Exposure Case	Exposure and Risk Estimates Table
Site Visitor	Soil	1A Soil ingestion	Adolescent	RME Average	B-2 B-3
		1B Dermal contact	Adolescent	RME Average	B-4 B-5
	Air	1C Inhalation of soil particulates	Adolescent	RME Average	B-6 B-7
Nearby Residents	Soil	2A Soil ingestion	Adult/Child	RME Average	B-8 B-9
		2B Dermal contact	Adult/Child	RME Average	B-10 B-11
	Vegetables	2C Vegetable ingestion	Adult	RME Average	B-12, B-16 B-13, B-17
			Child	RME Average	B-14, B-18 B-15, B-19

Table B-2

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
TRESPASSER INGESTION OF SOIL
Location: Dearcop Farm Site
Receptor: Adolescent Trespasser
Case: RME

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects		Carcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Cancer Risk
Benzo(a)anthracene	0.226	5.31E-08	—	7.58E-09	5.53E-09
Benzo(a)pyrene	0.237	5.57E-08	—	7.95E-09	5.80E-08
Benzo(b)fluoranthene	0.232	5.45E-08	—	7.78E-09	5.68E-09
Chrysene	0.233	5.47E-08	—	7.82E-09	5.71E-11
Dibenzo(a,h)anthracene	0.24	5.64E-08	—	8.05E-09	5.88E-08
Indeno(1,2,3-cd)pyrene	0.24	5.64E-08	—	8.05E-09	5.88E-09
Aroclor-1254	1.55	3.64E-07	—	5.20E-08	4.00E-07
Aroclor-1260	0.945	2.22E-07	—	3.17E-08	2.44E-07
Barium	1,550	3.64E-04	5.20E-03	5.20E-05	—
Cadmium	8.8	2.07E-06	4.13E-03	2.95E-07	—
Cobalt	620	1.46E-04	—	2.08E-05	—
Copper	1,540	3.62E-04	9.77E-03	5.17E-05	—
Lead	421	9.89E-05	—	1.41E-05	—
Mercury	0.161	3.78E-08	1.26E-04	5.40E-09	—
Nickel	160	3.76E-05	1.88E-03	5.37E-06	—
Zinc	858	2.01E-04	6.72E-04	2.88E-05	—
Ingestion route subtotal:			2.2E-02		7.8E-07

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-3

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
TRESPASSER INGESTION OF SOIL
Location: Dearcop Farm Site
Receptor: Adolescent Trespasser
Case: Average

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects		Carcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Cancer Risk
Benzo(a)anthracene	0.21	8.22E-09	—	1.17E-09	8.57E-10
Benzo(a)pyrene	0.22	8.43E-09	—	1.20E-09	8.79E-09
Benzo(b)fluoranthene	0.15	5.75E-09	—	8.21E-10	5.99E-10
Chrysene	0.21	8.38E-09	—	1.20E-09	8.73E-12
Dibenzo(a,h)anthracene	0.20	7.92E-09	—	1.13E-09	8.26E-09
Indeno(1,2,3-cd)pyrene	0.18	7.05E-09	—	1.01E-09	7.35E-10
Aroclor-1254	0.19	7.50E-09	—	1.07E-09	8.25E-09
Aroclor-1260	0.16	6.09E-09	—	8.70E-10	6.70E-09
Barium	142.14	5.56E-06	7.95E-05	7.95E-07	—
Cadmium	3.57	1.40E-07	2.80E-04	2.00E-08	—
Cobalt	68.51	2.68E-06	—	3.83E-07	—
Copper	188.07	7.36E-06	1.99E-04	1.05E-06	—
Lead	79.77	3.12E-06	—	4.46E-07	—
Mercury	0.08	3.24E-09	1.08E-05	4.62E-10	—
Nickel	51.59	2.02E-06	1.01E-04	2.88E-07	—
Zinc	158.17	6.19E-06	2.06E-05	8.84E-07	—
Ingestion route subtotal:			6.9E-04		3.4E-08

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-4

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
TRESPASSER DERMAL CONTACT WITH ON-SITE SOILS
Location: Dearcop Farm Site
Receptor: Adolescent Trespasser
Case: RME

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects		Carcinogenic Effects	
		Absorbed Dose (mg/kg/day)	Hazard Index	Absorbed Dose (mg/kg/day)	Cancer Risk
Aroclor-1254	1.55	8.30E-07	—	1.19E-07	9.13E-07
Aroclor-1260	0.945	5.06E-07	—	7.23E-08	5.57E-07
Cadmium	8.8	7.85E-07	1.57E-03	1.12E-07	—
Dermal route subtotal:			1.6E-03		1.5E-06

Table B-5

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
TRESPASSER DERMAL CONTACT WITH ON-SITE SOILS
Location: Dearcop Farm Site
Receptor: Adolescent Trespasser
Case: Average

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects		Carcinogenic Effects	
		Absorbed Dose (mg/kg/day)	Hazard Index	Absorbed Dose (mg/kg/day)	Cancer Risk
Aroclor-1254	0.19	5.58E-09	—	7.97E-10	6.06E-09
Aroclor-1260	0.16	4.53E-09	—	1.04E-09	8.04E-09
Cadmium	3.57	1.73E-08	3.47E-05	2.48E-09	—
Dermal route subtotal:			3.5E-05		1.4E-08

Table B-6

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
TRESPASSER INHALATION OF SOIL PARTICULATES
Location: Dearcop Farm Site
Receptor: Adolescent Trespasser
Case: RME

Chemical	Exposure Point Concentration (mg/m^3)	Noncarcinogenic Effects		Carcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Cancer Risk
Benzo(a)anthracene	1.87E-06	7.47E-09	—	1.07E-09	6.51E-10
Benzo(a)pyrene	2.62E-06	1.05E-08	—	1.49E-09	9.12E-09
Benzo(b)fluoranthene	4.03E-06	1.61E-08	—	2.30E-09	1.40E-09
Chrysene	2.43E-06	9.72E-09	—	1.39E-09	8.47E-12
Dibenzo(a,h)anthracene	1.87E-06	7.47E-09	—	1.07E-09	6.51E-09
Indeno(1,2,3-cd)pyrene	2.25E-06	8.97E-09	—	1.28E-09	7.82E-10
Aroclor-1254	1.45E-05	5.79E-08	—	8.28E-09	6.37E-08
Aroclor-1260	8.85E-06	3.53E-08	—	5.05E-09	3.89E-08
Barium	1.45E-02	5.79E-05	5.79E-01	8.28E-06	—
Cadmium	7.77E-05	3.10E-07	—	4.43E-08	2.79E-07
Cobalt	5.80E-03	2.32E-05	—	3.31E-06	—
Copper	1.44E-02	5.76E-05	—	8.22E-06	—
Lead	3.94E-03	1.57E-05	—	2.25E-06	—
Mercury	1.22E-06	4.86E-09	5.40E-05	6.94E-10	—
Nickel[a]	1.50E-03	5.98E-06	2.99E-04	8.54E-07	7.18E-07
Zinc	8.03E-03	3.21E-05	1.07E-04	4.58E-06	—
Inhalation route subtotal:			5.8E-01		1.1E-06

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

[a] Inhalation of nickel-contaminated soil particulates generally is considered more of a concern in an industrial setting; including nickel in this scenario may overestimate risks.

Table B-7

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
TRESPASSER INHALATION OF SOIL PARTICULATES
Location: Dearcop Farm Site
Receptor: Adolescent Trespasser
Case: Average

Chemical	Exposure Point Concentration (mg/m^3)	Noncarcinogenic Effects		Carcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Cancer Risk
Benzo(a)anthracene	1.87E-06	2.05E-09	—	2.93E-10	1.79E-10
Benzo(a)pyrene	2.20E-06	2.41E-09	—	3.44E-10	2.10E-09
Benzo(b)fluoranthene	1.59E-06	1.74E-09	—	2.49E-10	1.52E-10
Chrysene	1.97E-06	2.15E-09	—	3.08E-10	1.88E-12
Dibenzo(a,h)anthracene	1.78E-06	1.95E-09	—	2.78E-10	1.70E-09
Indeno(1,2,3-cd)pyrene	1.59E-06	1.74E-09	—	2.49E-10	1.52E-10
Aroclor-1254	3.00E-06	3.28E-09	—	4.69E-10	3.61E-09
Aroclor-1260	2.34E-06	2.56E-09	—	3.66E-10	2.82E-09
Barium	2.19E-03	2.40E-06	2.40E-02	3.43E-07	—
Cadmium	4.77E-05	5.23E-08	—	7.47E-09	4.71E-08
Cobalt	1.17E-03	1.28E-06	—	1.83E-07	—
Copper	2.96E-03	3.24E-06	—	4.63E-07	—
Lead	8.04E-04	8.81E-07	—	1.26E-07	—
Mercury	8.43E-07	9.23E-10	1.03E-05	1.32E-10	—
Nickel[a]	7.92E-04	8.68E-07	4.34E-05	1.24E-07	1.04E-07
Zinc	1.87E-03	2.05E-06	6.84E-06	2.93E-07	—
Inhalation route subtotal:			2.4E-02		1.6E-07

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

[a] Inhalation of nickel-contaminated soil particulates generally is considered more of a concern in an industrial setting; including nickel in this scenario may overestimate risks.

Table B-8

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
RESIDENTIAL INGESTION OF SOIL
Location: Nearby Residential Area
Case: RME

Chemical	Noncarcinogenic Effects				Carcinogenic Effects			
	Median		Maximum		Median		Maximum	
	Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Cancer Risk	Intake (mg/kg/day)	Cancer Risk
Benzo(a)anthracene	6.34E-06	—	1.08E-04	—	7.01E-07	5.12E-07	1.20E-05	8.73E-06
Benzo(a)pyrene	7.42E-06	—	7.86E-05	—	8.21E-07	5.99E-06	8.70E-06	6.35E-05
Benzo(b)fluoranthene	7.08E-06	—	6.19E-05	—	7.83E-07	5.71E-07	6.85E-06	5.00E-06
Benzo(k)fluoranthene	4.37E-06	—	4.62E-05	—	4.84E-07	3.53E-08	5.11E-06	3.73E-07
Chrysene	6.04E-06	—	7.96E-05	—	6.68E-07	4.88E-09	8.80E-06	6.43E-08
Dibenzo(a,h)anthracene	4.82E-06	—	4.72E-05	—	5.33E-07	3.89E-06	5.22E-06	3.81E-05
Indeno(1,2,3-cd)pyrene	6.63E-06	—	5.90E-05	—	7.34E-07	5.36E-07	6.52E-06	4.76E-06
Naphthalene	6.12E-06	—	1.77E-04	—	—	—	—	—
Barium	5.75E-04	8.21E-03	1.20E-03	1.71E-02	—	—	—	—
Cadmium	1.08E-05	2.16E-02	2.46E-05	4.91E-02	—	—	—	—
Cobalt	4.96E-05	—	9.24E-05	—	—	—	—	—
Copper	3.64E-04	9.83E-03	7.64E-03	2.06E-01	—	—	—	—
Lead	8.07E-04	—	2.33E-03	—	—	—	—	—
Mercury	6.39E-07	2.13E-03	1.97E-05	6.55E-02	—	—	—	—
Nickel	9.29E-05	4.64E-03	1.70E-04	8.50E-03	—	—	—	—
Zinc	1.73E-03	5.77E-03	2.00E-02	6.65E-02	—	—	—	—
Ingestion route subtotal:		5.22E-02		4.13E-01		1.15E-05		1.21E-04

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-9

NONCANCER HAZARD INDEX AND CANCER RISK ESTIMATES
RESIDENTIAL INGESTION OF SOIL
Location: Nearby Residential Area
Case: Average

Chemical	Noncarcinogenic Effects				Carcinogenic Effects			
	Median		Maximum		Median		Maximum	
	Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Hazard Index	Intake (mg/kg/day)	Cancer Risk	Intake (mg/kg/day)	Cancer Risk
Benzo(a)anthracene	3.17E-06	—	5.41E-05	—	1.12E-07	8.21E-08	1.92E-06	1.40E-06
Benzo(a)pyrene	3.71E-06	—	3.93E-05	—	1.32E-07	9.61E-07	1.39E-06	1.02E-05
Benzo(b)fluoranthene	3.54E-06	—	3.10E-05	—	1.26E-07	9.16E-08	1.10E-06	8.02E-07
Benzo(k)fluoranthene	2.19E-06	—	2.31E-05	—	7.76E-08	5.66E-09	8.20E-07	5.98E-08
Chrysene	3.02E-06	—	3.98E-05	—	1.07E-07	7.83E-10	1.41E-06	1.03E-08
Dibenzo(a,h)anthracene	2.41E-06	—	2.36E-05	—	8.54E-08	6.24E-07	8.37E-07	6.11E-06
Indeno(1,2,3-cd)pyrene	3.32E-06	—	2.95E-05	—	1.18E-07	8.59E-08	1.05E-06	7.64E-07
Naphthalene	3.06E-06	—	8.85E-05	—	—	—	—	—
Barium	2.87E-04	4.11E-03	5.99E-04	8.56E-03	—	—	—	—
Cadmium	5.41E-06	1.08E-02	1.23E-05	2.46E-02	—	—	—	—
Cobalt	2.48E-05	—	4.62E-05	—	—	—	—	—
Copper	1.82E-04	4.91E-03	3.82E-03	1.03E-01	—	—	—	—
Lead	4.04E-04	—	1.16E-03	—	—	—	—	—
Mercury	3.19E-07	1.06E-03	9.83E-06	3.28E-02	—	—	—	—
Nickel	4.64E-05	2.32E-03	8.50E-05	4.25E-03	—	—	—	—
Zinc	8.65E-04	2.88E-03	9.98E-03	3.33E-02	—	—	—	—
Ingestion route subtotal:		2.61E-02		2.07E-01		1.85E-06		1.93E-05

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-10

NONCANCER HAZARD INDEX ESTIMATE
RESIDENTIAL DERMAL CONTACT WITH SOIL
Location: Nearby Residential Areas
Case: RME

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects			
		Absorbed Dose (mg/kg/day)		Hazard Index	
		Median	Maximum	Median	Maximum
Cadmium	2.5	1.58E-06	3.60E-06	3.2E-03	7.2E-03

Table B-11

NONCANCER HAZARD INDEX ESTIMATE
RESIDENTIAL DERMAL CONTACT WITH SOIL
Location: Nearby Residential Areas
Case: Average

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects			
		Absorbed Dose (mg/kg/day)		Hazard Index	
		Median	Maximum	Median	Maximum
Cadmium	1.15	2.75E-07	6.25E-07	5.5E-04	1.3E-03

Table B-12

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (BROCCOLI)
Location: Residential garden
Receptor: Adult
Case: RME

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	10.8	1.03E-02	—
Calcium	2,325	2.23E+00	—
Chromium	0.52	4.98E-04	4.98E-04
Copper	4.7	4.50E-03	1.22E-01
Iron	12.8	1.23E-02	—
Lead	0.5	4.79E-04	—
Magnesium	624	5.98E-01	—
Manganese	3	2.87E-03	5.75E-01
Nickel	0.55	5.27E-04	2.63E-02
Potassium	3,980	3.81E+00	—
Selenium	0.08	7.66E-05	1.53E-02
Sodium	1,190	1.14E+00	—
Zinc	26.9	2.58E-02	8.59E-02
Cyanide	4.6	4.41E-03	2.20E-01
Ingestion route subtotal:			1.1E+00

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-13

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (BROCCOLI)
Location: Residential garden
Receptor: Adult
Case: Average

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	10.8	7.10E-04	—
Calcium	2,325	1.53E-01	—
Chromium	0.52	3.42E-05	3.42E-05
Copper	4.7	3.09E-04	8.35E-03
Iron	12.8	8.42E-04	—
Lead	0.5	3.29E-05	—
Magnesium	624	4.10E-02	—
Manganese	3	1.97E-04	3.95E-02
Nickel	0.55	3.62E-05	1.81E-03
Potassium	3,980	2.62E-01	—
Selenium	0.08	5.26E-06	1.05E-03
Sodium	1,190	7.82E-02	—
Zinc	26.9	1.77E-03	5.90E-03
Cyanide	4.6	3.02E-04	1.51E-02
Ingestion route subtotal:			7.2E-02

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-14

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (BROCCOLI)
Location: Residential garden
Receptor: Child
Case: RME

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	10.8	4.83E-02	—
Calcium	2,325	1.04E+01	—
Chromium	0.52	2.33E-03	2.33E-03
Copper	4.7	2.10E-02	5.68E-01
Iron	12.8	5.72E-02	—
Lead	0.5	2.24E-03	—
Magnesium	624	2.79E+00	—
Manganese	3	1.34E-02	2.68E+00
Nickel	0.55	2.46E-03	1.23E-01
Potassium	3,980	1.78E+01	—
Selenium	0.08	3.58E-04	7.15E-02
Sodium	1,190	5.32E+00	—
Zinc	26.9	1.20E-01	4.01E-01
Cyanide	4.6	2.06E-02	1.03E+00
Ingestion route subtotal:			4.9E+00

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-15

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (BROCCOLI)
Location: Residential garden
Receptor: Child
Case: Average

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	10.8	3.31E-03	—
Calcium	2,325	7.13E-01	—
Chromium	0.52	1.60E-04	1.60E-04
Copper	4.7	1.44E-03	3.90E-02
Iron	12.8	3.93E-03	—
Lead	0.5	1.53E-04	—
Magnesium	624	1.91E-01	—
Manganese	3	9.21E-04	1.84E-01
Nickel	0.55	1.69E-04	8.44E-03
Potassium	3,980	1.22E+00	—
Selenium	0.08	2.45E-05	4.91E-03
Sodium	1,190	3.65E-01	—
Zinc	26.9	8.25E-03	2.75E-02
Cyanide	4.6	1.41E-03	7.06E-02
Ingestion route subtotal:			3.4E-01

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-16

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (TOMATO)
Location: Residential garden
Receptor: Adult
Case: RME

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	11.7	1.20E-02	—
Calcium	113	1.16E-01	—
Chromium	0.49	5.03E-04	5.03E-04
Copper	0.58	5.95E-04	1.61E-02
Lead	0.17	1.74E-04	—
Magnesium	117	1.20E-01	—
Manganese	0.49	5.03E-04	1.01E-01
Nickel	0.39	4.00E-04	2.00E-02
Potassium	494	5.07E-01	—
Sodium	755	7.74E-01	—
Zinc	3.2	3.28E-03	1.09E-0
Ingestion route subtotal:			1.5E-01

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-17

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (TOMATO)
Location: Residential garden
Receptor: Adult
Case: Average

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	11.7	5.56E-04	—
Calcium	113	5.37E-03	—
Chromium	0.49	2.33E-05	2.33E-05
Copper	0.58	2.76E-05	7.45E-04
Lead	0.17	8.08E-06	—
Magnesium	117	5.56E-03	—
Manganese	0.49	2.33E-05	4.66E-03
Nickel	0.39	1.85E-05	9.27E-04
Potassium	494	2.35E-02	—
Sodium	755	3.59E-02	—
Zinc	3.2	1.52E-04	5.07E-04
Ingestion route subtotal:			6.9E-03

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-18

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (TOMATO)
Location: Residential garden
Receptor: Child
Case: RME

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	11.7	5.60E-02	—
Calcium	113	5.41E-01	—
Chromium	0.49	2.35E-03	2.35E-03
Copper	0.58	2.78E-03	7.50E-02
Lead	0.17	8.14E-04	—
Magnesium	117	5.60E-01	—
Manganese	0.49	2.35E-03	4.69E-01
Nickel	0.39	1.87E-03	9.33E-02
Potassium	494	2.36E+00	—
Sodium	755	3.61E+00	—
Zinc	3.2	1.53E-02	5.11E-02
Ingestion route subtotal:			6.9E-01

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.

Table B-19

NONCANCER HAZARD INDEX ESTIMATES
RESIDENTIAL INGESTION OF VEGETABLES (TOMATO)
Location: Residential garden
Receptor: Child
Case: Average

Chemical	Exposure Point Concentration (mg/kg)	Noncarcinogenic Effects	
		Intake (mg/kg/day)	Hazard Index
Aluminum	11.7	2.60E-03	—
Calcium	113	2.51E-02	—
Chromium	0.49	1.09E-04	1.09E-04
Copper	0.58	1.29E-04	3.48E-03
Lead	0.17	3.77E-05	—
Magnesium	117	2.60E-02	—
Manganese	0.49	1.09E-04	2.17E-02
Nickel	0.39	8.65E-05	4.33E-03
Potassium	494	1.10E-01	—
Sodium	755	1.68E-01	—
Zinc	3.2	7.10E-04	2.37E-03
Ingestion route subtotal:			3.2E-02

Note: Hazard quotients were added for informational purposes only. It may be inappropriate to add the hazard quotients for the individual chemicals since they may not all contribute to the same adverse health effects.